E. Medical Complications

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110 Pages

E1. Dysphagia

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E1.1 Introduction to Dysphagia Post Stroke

Q1. Define dysphagia.

Answer
1. Dysphagia is defined as difficulty with swallowing

Q2. Why is dysphagia important following a stroke?

Answer
1. Dysphagia is common following a stroke.
2. Incident rates are high following an acute stroke
3. Associated with increased mortality and morbidity such as malnutrition, dehydration and pneumonia.
4. Diagnosing and treating dysphagia in stroke patient improves outcomes such as reduced risk of pneumonia, length of hospital stay and overall health expenditures.
5. Aspiration, the most important clinical consequence of dysphagia, has long been associated with an increased risk of pneumonia, death and sepsis.

Discussion

Dysphagia is defined as difficulty with swallowing and is a common complication of stroke. The incidence rates are reported to be between 29-65% in acute stroke patients. Some of the variability is related to differences in the timing and method of swallowing assessment. The presence of dysphagia can be identified on the basis of clinical or radiographic examinations, or both.

The Heart and Stroke Foundation (2002) noted *"the presence of dysphagia in stroke survivors has been associated with increased mortality and morbidities such as malnutrition, dehydration and pulmonary compromise"* (Smithard et al. 1996, Barer 1989, Kidd et al. 1995, Finestone et al. 1995, Teasell et al. 1994, Gordon et al. 1987, Schmidt et al. 1994, Sharma et al. 2001). However, emerging evidence indicates that detecting and managing dysphagia in acute stroke survivors improves outcomes such as reduced risk of pneumonia, length of hospital stay and overall healthcare expenditures (Smithard et al. 1996).

Aspiration following stroke, the most clinically significant symptom of dysphagia, has long been associated with pneumonia, sepsis and death. Silver et al. (1984) and Bounds et al. (1981) reported that pneumonia was the second most common cause of death during the acute phase of a stroke, with up to 20% of individuals with stroke-related dysphagia dying during the first year post stroke from aspiration pneumonia (Schmidt et al. 1988). Steele (2002) found that the number of swallowing difficulties seen in stroke survivors was associated with the length of hospitalization. Detection of aspiration, both silent and audible, and subsequent adaptive

management strategies are regarded as important in the prevention of pneumonia (Horner and Massey 1988a, Horner et al. 1988b, Logemann 1983, Teasell et al. 1996, Tobin 1986, Veis and Logemann 1985). Management of dysphagia largely focuses on strategies to avoid aspiration following stroke.

E1.2 The Normal Swallowing Process

Q3. Describe the normal swallowing process.

Answers
Normal swallowing consists of 4 phases:
1. Oral preparatory phase
2. Oral propulsive phase
3. Pharyngeal phase
4. Esophageal phase

Discussion

Normal Swallowing
Normal swallowing has four sequential coordinated phases: the oral preparatory phase, the oral propulsive phase, the pharyngeal phase and the esophageal phase. Each of the phases of a normal swallow is described below.

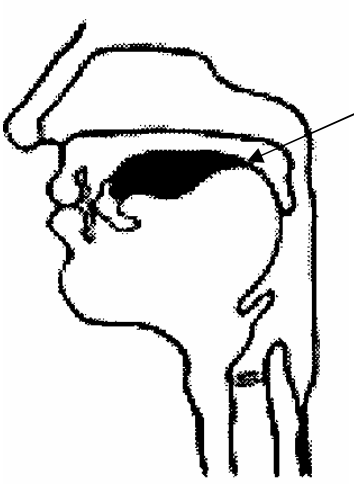
1. **Oral Preparatory Phase.** During this phase, food in the oral cavity is manipulated and masticated in preparation for swallowing. The back of the tongue controls the position of the food, preventing it from falling into the pharynx (Platt 2001).

2. **Oral Propulsive Phase**. During the oral propulsive, the tongue transfers the bolus of food to the pharynx, triggering the pharyngeal swallow (Platt 2001).

3. **Pharyngeal Phase.** During the pharyngeal phase, complex and coordinated movements of the tongue and pharyngeal structures propel the bolus into the esophagus, while protecting the airway (Platt 2001).

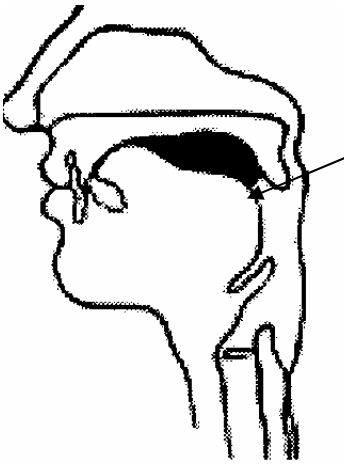
4. **Esophageal Phase.** During the esophageal phase of swallowing coordinated contractions of the esophageal muscle move the bolus through the esophagus towards the stomach (Platt 2001).

Oral Preparatory Phase



Food in the oral cavity is manipulated and masticated in preparation for swallowing. The back of the tongue controls the position of the food, preventing it from falling back into the pharynx.

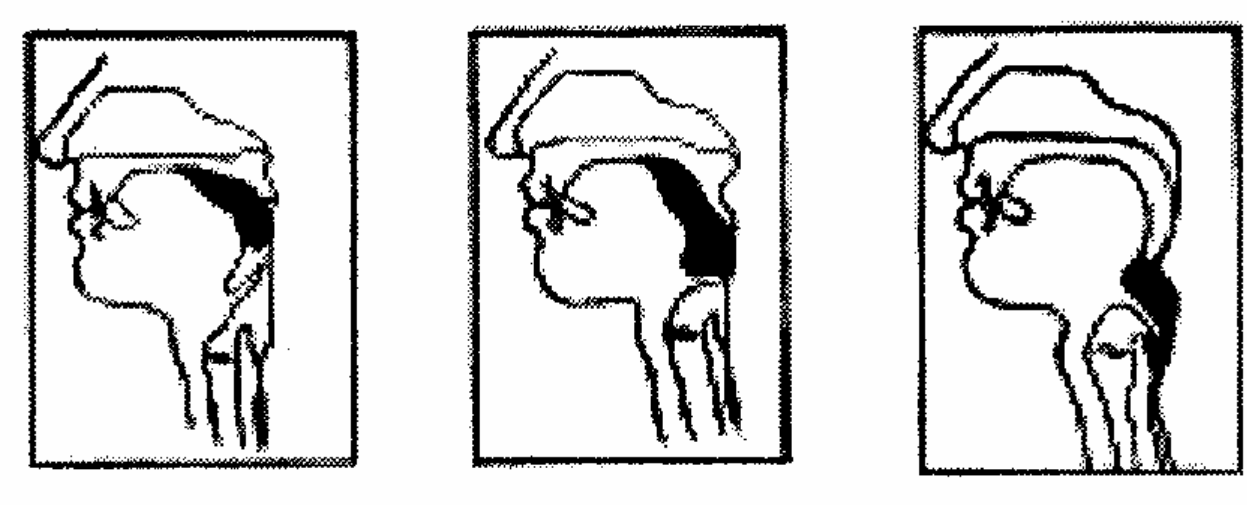
1. Oral Propulsive Phase



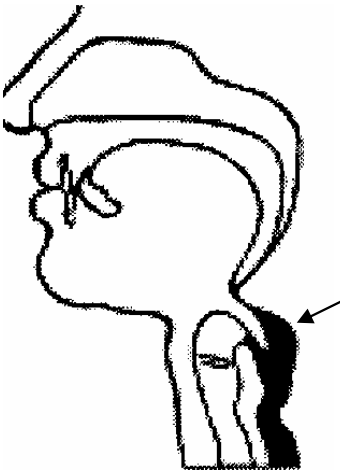
The tongue transfers the bolus of food to the pharynx which triggers the pharyngeal swallow.

2. Pharyngeal Phase

Complex and coordinated movements of the tongue and pharyngeal structures propel the food bolus into the esophagus while protecting the airway.



3. Esophageal Phase



Coordinated contractions of the esophagus muscle move the bolus through the esophagus toward the stomach.

E1.3 Defining Dysphagia and Aspiration

Q4. What is the difference between Dysphagia and Aspiration?

Answers

1. Dysphagia is defined as difficulty with swallowing.
2. Aspiration is defined as entry of oral or gastric material into the airway below the level of the true vocal cords.

Discussion

Dysphagia
Dysphagia post stroke is characterized by a delay and reduced function in the pharyngeal phase of swallowing. Although the incidence of dysphagia is more common following brainstem or bilateral hemispheric stroke, it frequently occurs following unilateral hemispheric strokes. Signs and Symptoms of dysphagia include: Choking on food, coughing during meals, drooling or loss of food from mouth, pocketing on food in cheeks, slow, effortful eating, difficulty swallowing pills, avoiding food or fluids, complaining of food sticking in throat, problems swallowing, reflux or heartburn (Schmidt et al. 1994).

Aspiration
Aspiration is defined as "entry of material into the airway below the level of the true vocal cords". Since many stroke patients with dysphagia do not aspirate, the two terms are not synonymous, although they are closely associated. The diagnosis of aspiration should be suspected when the stroke patient has any of the following: a subjective complaint of trouble swallowing, an abnormal chest x-ray, congested voice quality, or a delay in voluntary initiation of the swallow reflex and coughing during or after swallowing (Horner et al 1988b). Diagnosis is initially established through clinical assessment involving an oral motor examination followed by the introduction of one or several teaspoons of water. If patients are able to successfully swallow this minimal amount of fluid, a small cup of water is carefully introduced. The full assessment is described elsewhere (Smithard et al. 1996). While all stroke patients are potential aspirators, there are certain identifiable risk factors that have been recognized as greatly increasing the likelihood of aspiration. These clinical risk factors are below.

E1.4 Risk Factors for Aspiration Post Stroke

Q5. What are the risk factors or clinical red flags for aspiration post stroke?

Answer
1. Brainstem Stroke
2. Difficulty swallowing oral secretions
3. Coughing/throat clearing or wet, gurgly voice quality after swallowing water
4. Choking more than once while drinking 50 cc of water
5. Weak voice and cough
6. Wet-hoarse voice quality
7. Recurrent lower respiratory infections
8. Low-grade fever or leukocytosis
9. Auscultatory evidence of lower lobe congestion
10. Immunocompromised state

E1.5 Dysphagia Post Stroke

Q6. How common is dysphagia following acute stroke?

Answer

1. The incidence of dysphagia appears to be quite high following acute stroke with between one third and two-thirds of patients affected, depending on the population studied and the criteria used to diagnose dysphagia.

Discussion

The majority of studies reviewed above assessed swallowing status in the acute phase of stroke. Among these studies, the incidence of dysphagia ranged from 19% to 45%. Only two trials (DePippo et al. 1994, Gottlieb et al. 1996) assessed swallowing upon rehabilitation to a rehabilitation unit. The incidences of dysphagia were 61% and 28%, respectively.

E1.6 Silent Aspiration

Q7. What is silent aspiration and why is it important?

Answers

1. Silent aspiration is defined as “penetration of food below the level of the true vocal cords without cough or any outward signs of difficulty”.
2. Important because aspiration risk may go undetected placing the patient at higher risk of developing aspiration related complications.
3. 9-27% of acute stroke patients are silent aspirators, a condition which is only reliably detectable through VMBS studies.
4. Between one-third to one-half of aspirators are “silent” aspirators.

Discussion

In addition to overt signs of aspiration, such as chocking or coughing, a substantial number of patients experience silent aspiration, highlighting the utility of using VMBS studies. "Silent aspiration" is defined as "penetration of food below the level of the true vocal cords, without cough or any outward sign of difficulty" (Linden and Siebens 1983). Detailed clinical swallowing assessments were shown to under-diagnose or miss these cases of aspiration (Horner and Massey 1988a, Horner et al. 1988b, Splaingard et al. 1988, Terre & Mearin 2006). In particular, the presence or absence of a gag reflex failed to distinguish aspirating from non-aspirating stroke patients (Horner and Massey 1988a, Horner et al. 1988b, Splaingard et al. 1988). Silent aspirators were considered to be at increased risk of developing complications. Since the condition was not diagnosed, precautions to decrease aspiration risk would often not be employed. 9 to 27% of acute stroke patients are silent aspirators, a condition which is only reliably detectable through VMBS studies. Between one-third and one-half of aspirators are “silent” aspirators.

Q8. When should silent aspiration in a stroke patient be suspected?

Answer

1. Silent aspiration should be suspected in any stroke patient who presents with recurrent lower respiratory infections, chronic congestion, a low-grade fever, leukocytosis, weak voice or cough or a wet-hoarse voice quality after swallowing.

Discussion

Silent aspiration should be suspected in the stroke patient with recurrent lower respiratory infections, chronic congestion, low-grade fever or leukocytosis (Muller-Lissner et al. 1982). Clinical markers of silent aspiration may include a weak voice or cough or a wet-hoarse voice quality after swallowing.

Canadian Stroke Strategy Guideline (2008): Recommendation 6.1 – Dysphagia Assessment

Patients with stroke should have their swallowing ability screened using a simple, valid, reliable bedside testing protocol as part of their initial assessment, and before initiating oral intake of medications, fluids or food [Evidence Level B] (CSQCS, NZ, SCORE, SIGN 78).

i. Patients who are not alert within the first 24 hours should be monitored closely and dysphagia screening performed when clinically appropriate [Evidence Level C].

ii. Patients with stroke presenting with features indicating dysphagia or pulmonary aspiration should receive a full clinical assessment of their swallowing ability by a speech–language pathologist or appropriately trained specialist who should advise on safety of swallowing ability and consistency of diet and fluids [Evidence Level A] (CSQCS, NZ, RCP, SCORE).

iii. Patients who are at risk of malnutrition, including those with dysphagia, should be referred to a dietitian for assessment and ongoing management. Assessment of nutritional status should include the use of validated nutrition assessment tools or measures [Evidence Level C] (AU).

E1.7 Pneumonia and Aspiration Post Stroke

Q9. What is the relationship between aspiration and pneumonia?

Answers

1. Aspiration alone is not sufficient to cause pneumonia. Aspiration of small amounts of saliva occurs during sleep in almost half of normal subjects.
2. Aspiration pneumonia is thought to occur when the lung's natural defences are overwhelmed when excessive and/or toxic gastric contents are aspirated, leading to a localized infection or a chemical pneumonitis.

3. Those patients who aspirate over 10% of the test bolus or who have severe oral and/or pharyngeal motility problems on VMBS studies are considered at high risk for pneumonia.
4. The severity of aspiration correlates with the risk of developing pneumonia.

Discussion

Aspiration alone is not sufficient to cause pneumonia. Aspiration of small amounts of saliva occurs during sleep in almost half of normal subjects (Finegold 1991, Huxley et al. 1978). Aspiration pneumonia is thought to occur when the lung's natural defences are overwhelmed when excessive and/or toxic gastric contents are aspirated, leading to a localized infection or a chemical pneumonitis. Those patients who aspirate over 10% of the test bolus or who have severe oral and/or pharyngeal motility problems on VMBS studies are considered at high risk for pneumonia (Logemann 1983, Milazzo et al. 1989). In many cases, it is difficult to practically assess whether 10% or more of the test bolus has been aspirated. Nevertheless, the degree of aspiration seen on VMBS study is a critical determinant of aspiration risk and subsequent patient management.

The importance of the diagnosis and management of aspiration post stroke has been driven by the purportedly causal relationship between aspiration and pneumonia (Brown and Glassenberg 1973, Hanning et al. 1989, Holas et al. 1994, Johnson et al. 1993). In turn, mortality following a stroke as a consequence of pneumonia (presumably due to aspiration) has been reported as high as 3% within the first 3 months (Kidd et al. 1995) and 6% within the first year (Hanning et al. 1989). Aspiration pneumonia has therefore been regarded as important because of its significant contribution to morbidity and mortality (Arms et al. 1974, Gordon et al. 1987, Hanning et al. 1989, Johnson et al. 1993, Logemann 1983, Silver et al. 1984, Veis and Logemann 1985).

E1.8 Risk Factors for Aspiration Pneumonia

Q10. What are some of the risk factors for aspiration pneumonia?

Answers

1. Brainstem stroke
2. Aspiration of VMBS (risk greater if greater than 10% aspirated)
3. Aspiration of thick fluids or solids
4. Slower pharyngeal transit time on VMBS
5. Reduced levels of consciousness
6. Elderly
7. Dysarthria
8. Cognitive difficulties
9. Failure of water swallow test

Discussion

Factors associated with an increased risk of aspiration pneumonia include: dysphagia related factors due to stroke (see Table E1), as well as reduced levels of consciousness, a tracheostomy, gastric reflux or emesis, nasogastric tubes (due to mechanical interference with

the cardiac sphincter), and a compromised immune system (Finegold 1991). Predicting whether a patient will develop pneumonia post aspiration is, to some extent, dependent on other factors such as the immune state or general health of the stroke patient. Sellars et al. (2007) prospectively evaluated 412 stroke patients for up to 3 months following stroke. Over this period, there were 160 cases of either confirmed or suspected pneumonias. Independent predictors of pneumonia were age >65 years, dysarthria or no speech due to aphasia, a modified Rankin Scale score ≥4, an Abbreviated Mental Test score <8, and failure on the water swallow test. The presence of 2 or more of these risk factors carried 90.9% sensitivity and 75.6% specificity for the development of pneumonia.

Factors More Likely to be Associated with Aspiration Pneumonia Following Stroke

- Brainstem stroke
- Aspiration on VMBS (risk greater if aspirates over 10% of barium laced test material)
- Aspiration of thick fluids or solids
- Slower pharyngeal transit time on VMBS

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E2. Dysphagia Case Study

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E2.1 Lateral Medullary Infarction (Wallenburg's Syndrome)

Case Study

A 68-year old man presents with a stroke involving the territory of the left lateral medulla. This is due to an infarct of the posterior inferior cerebellar artery. The patient presents to the Emergency Room with significant ataxia, dizziness and dysarthria.

Q1. Describe the affected vasculature and the typical presentation of a left lateral medullary infarction (Wallenburg's syndrome)?

Answers

Ipsilateral Side (right-side):

- Horner's syndrome (ptosis, anhydrosis and miosis)
- Decrease in pain and temperature over ipsilateral face
- Cerebellar signs such as ataxia

Contralateral Side (left-side):

- Decreased pain and temperature over contralateral body
- Dysphagia, dysarthria, hoarseness and paralysis of vocal cord
- Vertigo, nausea and vomiting
- Hiccups
- Nystagmus, diplopia

Should not mention facial or extremity weakness.

E2.2 Assessment of Dysphagia Post Stroke

Case Study (continued)

The nurse in the emergency room provides the patient with a drink of water. The patient responds with choking. He develops a persistent cough while in the emergency room.

Q2. What would be the next step?

Answers

1. Having had a brainstem stroke, this patient would be considered at relatively high risk of aspirating.
2. Acute stroke patients should be maintained NPO until their swallowing ability is determined.
3. A clinical bedside screening should be conducted by a trained team member and if there are any concerns or the patient is regarded at risk of dysphagia than an oral motor assessment should be performed, generally by a speech-language pathologist.
4. This trial involves 1-2 teaspoons of water and if that is well tolerated is followed by a small cup of water.

Discussion

The Water Swallowing Test

The water-swallowing test has also been studied extensively. It has been used as both a stand alone screening method and also as part of a clinical swallowing screening or assessment. While the original test required a patient to swallow 3 oz (90 mL) of water, lesser amounts have also been used. To be clinically useful, screening tests need to be valid, reliable, easy to use, non-invasive, quick to administer (15-20 min) and pose little risk to the patient. Although many screening tools have been developed it is unclear how many of them are used in institutions beyond those where they were developed. Many institutions use informal processes, or simply restrict all food and drink until complete assessment by an SLP. A wide range of sensitivities were reported among the tools we reviewed (0% to 100%). Usually, as sensitivity increased, specificity decreased, such that the number of patients who were incorrectly identified as dysphagic increased. Generally screening tools with sensitivity > 80%, with a specificity that approaches this figure are considered to be both valid and clinically useful.

The results of a systematic review by Martino et al. (2000), evaluating the screening accuracy of 49 individual clinical screening tests for orophayngeal dysphagia suggest that there is only sufficient evidence to support the value of two tests: abnormal pharyngeal sensation and the 50 mL water-swallowing test. Both of these tests assessed only for the presence or absence of aspiration. Their associated likelihood ratios were 5.7 (95% CI 2.5-12.9) and 2.5 (95% CI 1.7-3.7), respectively. Limited evidence for screening benefit suggested a reduction in pneumonia, length of hospital stay, personnel costs and patients.

Case Study (continued)

The nurse looking after the patient on the acute floor has concerns that when given some water the patient again chokes. The patient manages the 2 teaspoons of water without difficulty but chokes once on a small cup of water (50ml) and his voice now has a wet, gurgly sound to it.

Q3. Discuss management options now.

Answers

1. Once a patient fails a screening test and it has been determined that a problem exits, a more comprehensive assessment should follow, from which treatment options are determined.
2. The assumption needs to be made that the patient is an aspirator and a videoscopic modified barium swallow (VMBS) needs to be performed.
3. While the patient is waiting for the VMBS, the patient is kept NPO and an nasogastric (NG) tube is inserted to ensure that the patient receives adequate nutrition and hydration.

Discussion

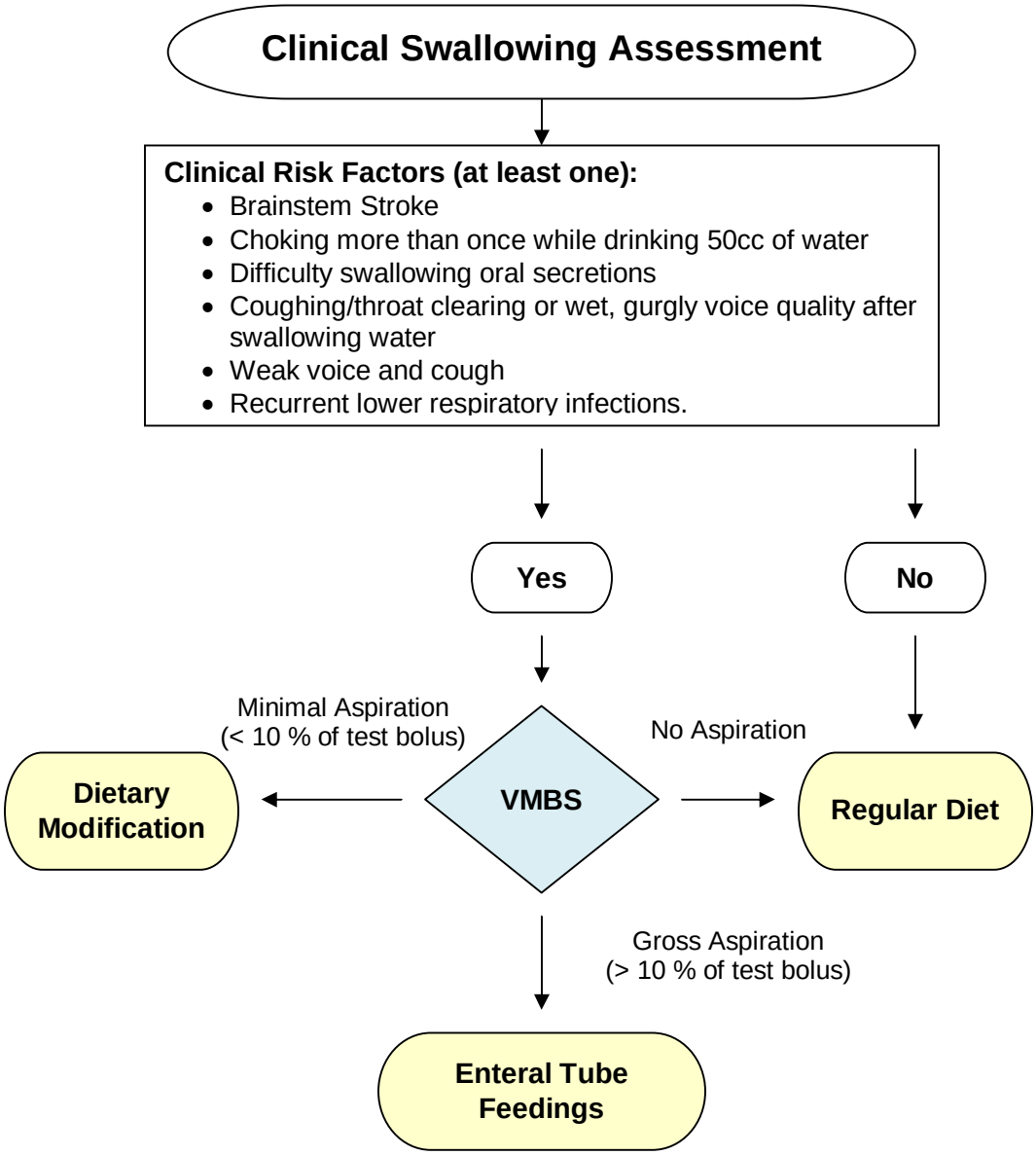
VMBS Examination

When aspiration is suspected, the videofluoroscopic modified barium swallow (VMBS) study is often considered the "gold standard" in confirming the diagnosis (Splaingard et al. 1988). A VMBS study examines the oral and pharyngeal phases of swallowing. The patient must have sufficient cognitive and physical skills to undergo testing (Bach et al. 1989). The subject is placed in the sitting position in a chair designed to simulate the typical mealtime posture. Radio-opaque materials of various consistencies are tested: barium impregnated thin and thick liquids, pudding, bread, and cookies are routinely used. Various aspects of oral, laryngeal, and pharyngeal involvement are noted during the radiographic examination (Table 15.13). The VMBS study is then followed by a chest x-ray to document any barium, which may have been aspirated into the tracheobronchial tree.

The VMBS assessment not only establishes the presence and extent of aspiration but may also reveal the mechanism of the swallowing disorder. Aspiration most often results from a functional disturbance in the pharyngeal phase of swallowing related to reduced laryngeal closure or pharyngeal paresis. A VMBS study is recommended in those cases where the patient is experiencing obvious problems maintaining adequate hydration/nutrition, where concern is expressed regarding frequent choking while eating, or in the case of recurrent respiratory infections.

While VMBS studies can be useful in analyzing the anatomic structures during swallowing and detecting silent aspiration, there are some disadvantages: i) The procedure is relatively complex, time consuming and resource intensive; ii) there is some exposure to small amounts of radiation; iii) the test is not appropriate for some patients who may have difficulty in sitting upright in a chair. The results of the test can also be difficult to interpret and there can be significant variation among individual raters (Ramsay et al. 2003).

Assessment of Swallowing Post Stroke at Time of Admission



E2.3 Management of Dysphagia Post Stroke

Case Study (continued)

The patient aspirates all consistencies of barium laced food on VMBS but only coughs on the thin liquids. The video modified barium swallow shows significant aspiration due to poor coordination of the pharyngeal phase of swallowing (>10% of the test bolus).

Q4. Discuss management strategies.

Answers

1. The patient should not be fed by mouth and an NG tube is required for an extended period of time.
2. At this point many centers would install a GJ tube to facilitate maintenance of nutrition and hydration rather then wait.

Discussion

The VMBS study is still considered the "gold standard" in the diagnosis of aspiration. Those patients who have difficulty with high volumes of thin liquids are considered to be at mild to moderate risk of aspiration. In these cases oral feedings are regarded as appropriate. Before deciding if a patient is a candidate for oral feeding, factors such as the patient's respiratory status, the effectiveness of airway clearance along with the type and amount of aspirate must first be considered (Bach et al. 1989). Aspirating more than 10% of the test bolus is generally considered an indication for non-oral (ie. nasogastric, gastrostomy, jejunostomy tube) feedings; however, the actual risks present with oral feedings for this group of patients have not been fully established. Determining whether the patient actually aspirates more or less than 10% of the test bolus is, as mentioned previously, an inexact science.

Management Strategies for Dysphagia

The Heart and Stroke Foundation Dysphagia Guidelines noted that, “a well coordinated care plan can minimize the development of dysphagic complications, reduce length of hospital stay in acute-care facilities and expedite access to specialized rehabilitation centers.

Dysphagia management has the following goals:

- Meeting the nutrition and hydration requirements of the stroke survivor.
- Preventing aspiration-related complications.
- Maintaining and promoting swallowing function as much as possible.

Dysphagia management strategies include the following:

- Modifying food and fluid textures to increase safety of oral intake.
- Using low-risk feeding practices and compensatory strategies to prevent complications such as aspiration and choking.
- Monitoring oral intake to prevent dehydration.
- Supplementing the diet to maintain adequate nutrition.
- Using enteral feeding for individuals who are unable to swallow.
- Implementing swallow therapy to rehabilitate specific physiological swallowing impairments.

A speech-language pathologist should regularly monitor the status of individuals with dysphagia to ensure that the management strategies employed remain appropriate,” (Heart and Stroke Foundation of Ontario 2002).

Case Study (continued)

The patient has a GJ tube installed. A second modified barium swallow is done and this shows an improvement in the aspiration (<10% of test bolus). The patient is able to handle pureed consistencies well but still shows aspiration on thin liquids.

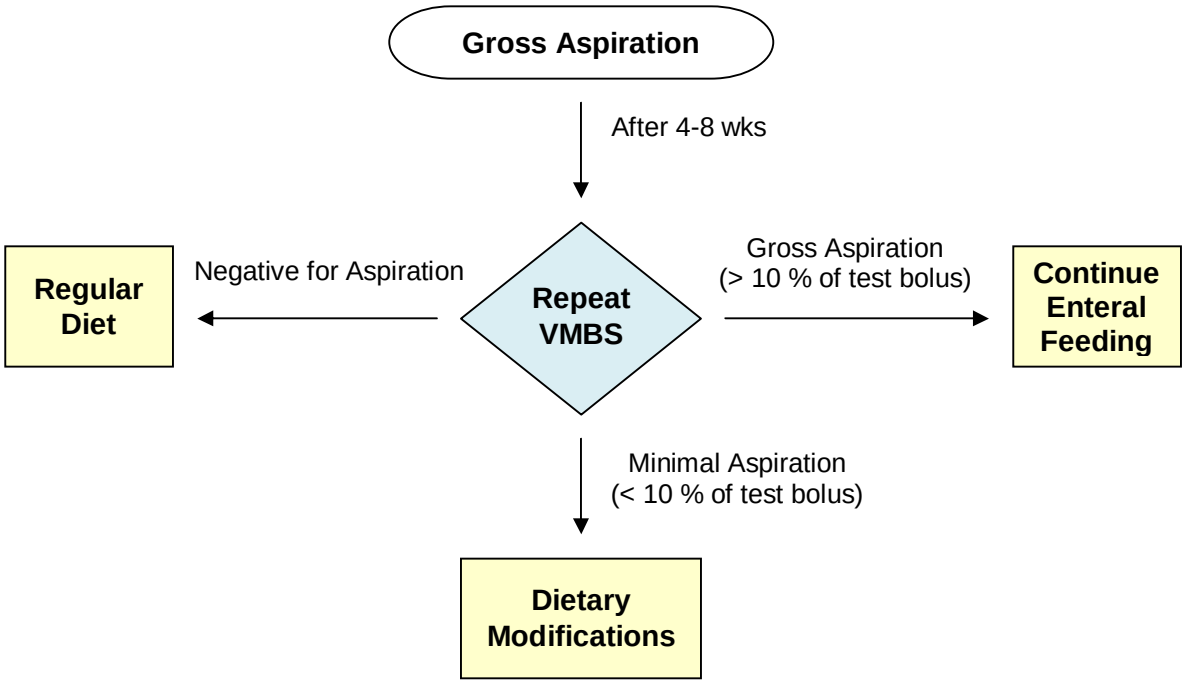
Q5. Discuss management.

Answers

1. The patient can be fed orally a pureed diet with a restriction on thin liquids.
2. Fluids can be supplemented through the G-J tube.
3. Another VMBS would need to be ordered for 2-3 months and the patient carefully monitored by a dietician and speech-language pathologist.

Discussion

Management of Gross Aspiration



Case Study (continued)

3 months later the patient has a repeat VMBS and continues to show improvement, although she is still having trace aspiration with thin liquids.

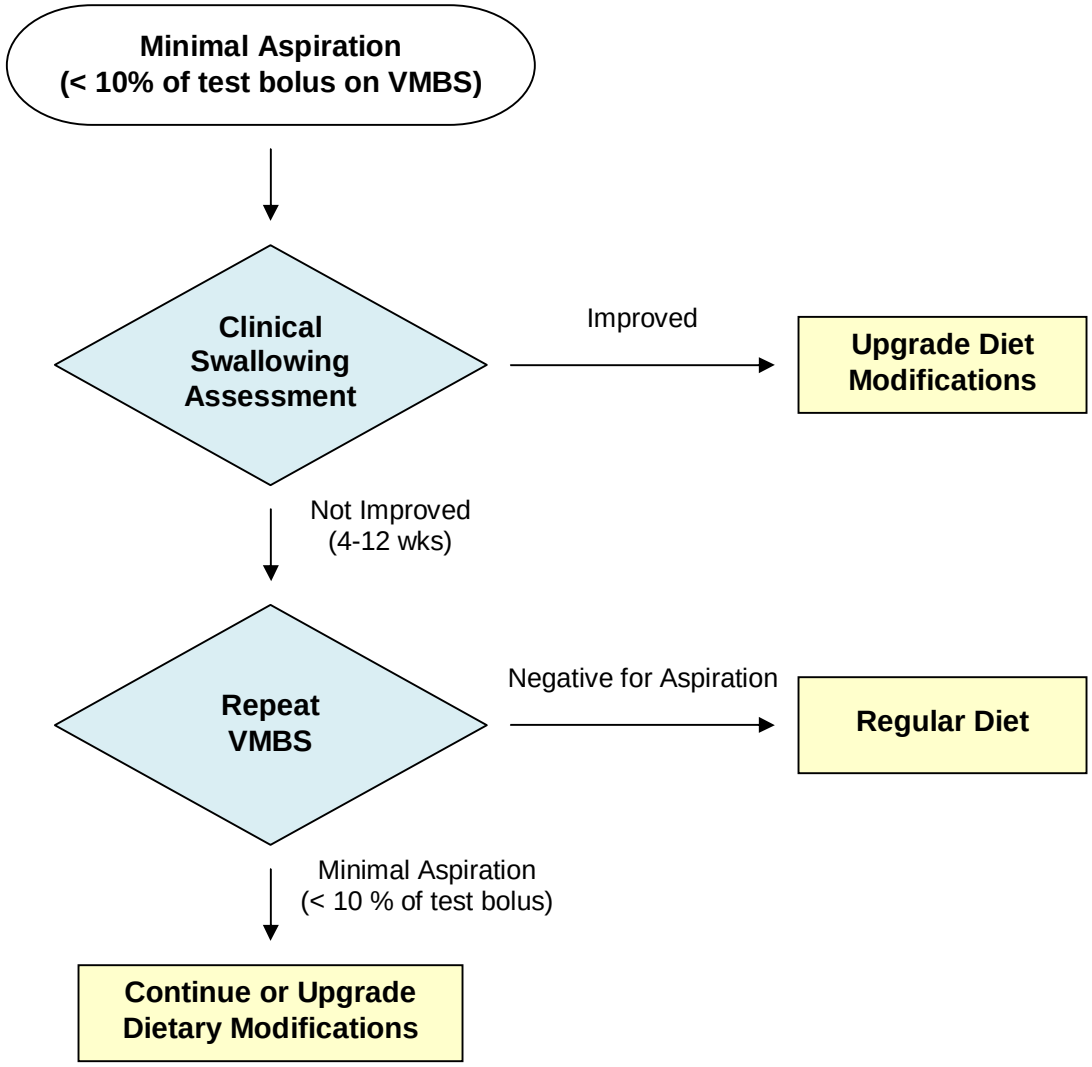
Q6. Discuss management.

Answers

1. The patient can continue to be fed orally an unrestricted diet.
2. However, they are advised to sip thin liquids and double cough afterwards.
3. The patient needs to continue to be monitored for a lung infection.

Discussion

Continuing Management of Minimal Aspiration



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E3. Dysphagia Case Study

E3. Dysphagia Case Study

Case Study

A 56-year-old right-handed hypertensive, type II diabetic was admitted to a local hospital with complaints of occipital headache, right hand tingling, right limb ataxia, hoarse voice, dysphagia, nausea, vomiting and vertigo. Blood pressure was elevated but weakness was not noted on examination. He was transferred to a tertiary care center where an occlusion of the posterior inferior cerebellar artery was diagnosed.

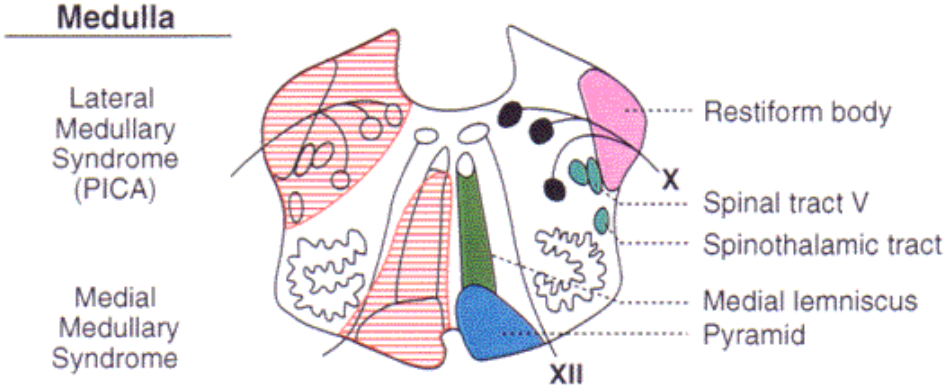
E3.1 Lateral Medullary Infarction (Wallenburg's syndrome)

Q1. Describe the affected vasculature and the typical presentation of a right lateral medullary infarction (Wallenburg's syndrome)?

Ipsilateral Side (right-side):
- Horner's syndrome (ptosis, anhydrosis and miosis)
- Decrease in pain and temperature over ipsilateral face
- Cerebellar signs such as ataxia

Contralateral Side (left-side):
- Decreased pain and temperature over contralateral body
- Dysphagia, dysarthria, hoarseness and paralysis of vocal cord
- Vertigo, nausea and vomiting
- Hiccups
- Nystagmus, diplopia

Should not mention facial or extremity weakness.



E3.2 Management of Dysphagia Post Stroke

Case Study (continued)

The following day he was noted to have severe dysphagia which was his primary complaint and bilateral aspiration pneumonia which was confirmed on x-ray. Antibiotics were initiated for the pneumonia. The speech language pathologist did a bedside swallowing assessment on the day of admission. His vocal quality was described as "wet" and he could not elevate the hyoid bone, indicating a probable swallow reflex problem. Oral motor elevation revealed tongue elevation was significantly reduced, with a low resting soft palate on the left and reduced movement on that side during phonation. No gag reflex could be elicited. Mild hypernasality during conversation was noted. Maximum phonation duration at 6 seconds indicated reduced breath support, likely resulting from vocal cord paralysis. During phonation and conversation vocal quality was moderately breathy. He was noted to have a weak cough and diminished throat-clearing ability. A swallow reflex could not be elicited.

Q2. What step would you do next?

Answers

1. The next step would be to decide to do a VMBS or if not readily available, assume the patient was a gross aspirator.

Case Study (continued)

The bedside swallow assessment therefore deemed he was unsafe for oral feeds and at risk for the development of further aspiration. A VMBS was not performed at that time because it was not perceived that the test would change clinical management, in view of the severity of the results of the bedside assessment.

Initially the patient was kept NPO and a NG tube was inserted to provide nonoral feeds. The patient was carefully followed and one week after the stroke the SLP noted evidence of initiation of a swallow reflex. At that point, a clinical trial of both thin and thick fluids was attempted. He continued to demonstrated clinical signs of possible aspiration, such as coughing and a wet voice with both fluid consistencies. There was a delay in the initiation of the swallow reflex and the patient reported marked difficulty in clearing food through

the pharynx. Vocal quality was wet and gurgly and there was post-swallowing coughing and throat clearing. A head turn compensatory strategy toward the left side was attempted to direct the bolus down the stronger side of the pharynx. The patient reported that this seemed to only minimally help the pharyngeal clearing. Given the severity of his swallowing problems, the apparent high risk of aspiration, and the anatomical location of his stroke, a GJ tube was recommend and this was inserted percutaneously.

During the first month following his stroke, the patient demonstrated improvement in his pharyngeal swallow including better clearing of oral secretions and mild improvement in clearing of thickened fluids. He continued to use lateral head rotation to the left side during the swallow assessment; however, there were continuing clinical signs of laryngeal penetration of oral contents.

The patient was subsequent admitted to a stroke rehabilitation unit one month following the onset of his stroke. Further bedside assessments indicated that he was unable to tolerate more than half a teaspoon of thin liquids at any time. The SLP noted that the patient likely had experienced a delay in the initiation of his pharyngeal swallow, and he was experiencing weakness of pharyngeal peristalsis. This likely resulted in residue in the both valleculae and pyriform sinuses. The patient was given oral and pharyngeal exercises to be used when clearing his oral secretions to improve the strength of pharyngeal constriction, i.e., hard glottal swallows with the head turned to the left.

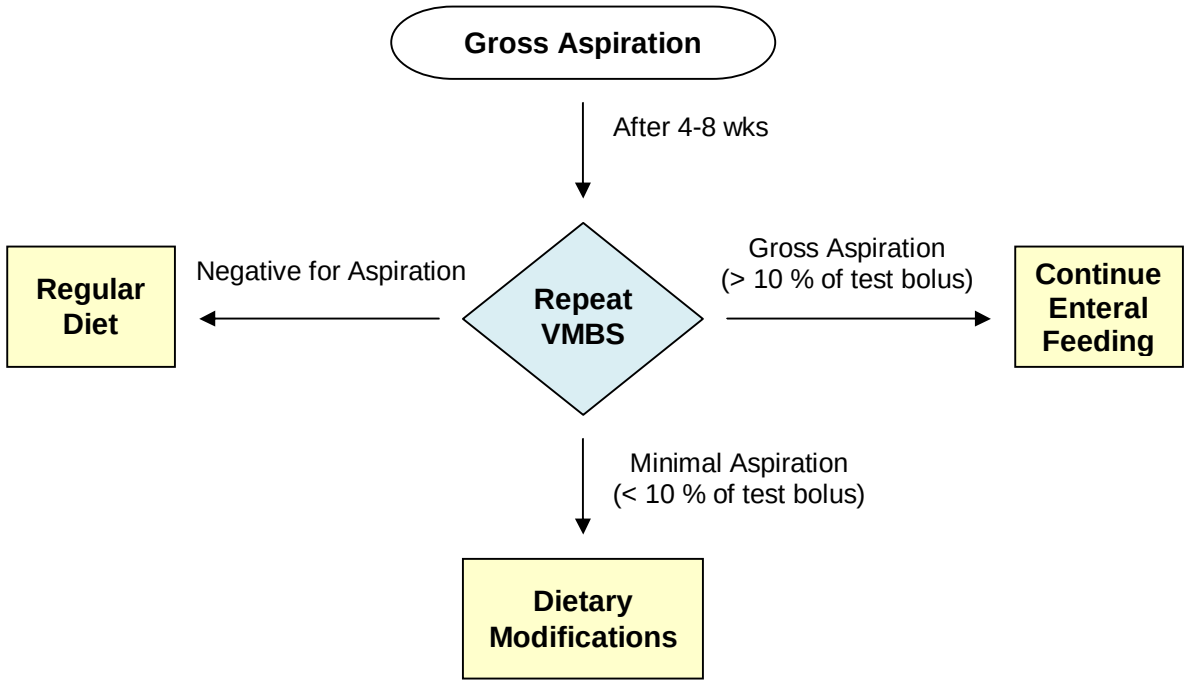
Q3. What would be your next step?

Answer

1. Next step would be to conduct a repeat VMBS study (see the algorithm below).

Discussion

Management of Gross Aspiration



Case Study (continued)

A VMBS study was subsequently performed and indicated good oral transport of all consistencies presented. A half teaspoon of pudding revealed moderate delay in the swallow reflex with minimal residue in the valleculae but a large residue present in the pyriform sinus that was then grossly aspirated. Presentation of a half teaspoon of thick liquids with the chin tucked again revealed gross aspiration occurring from a large residue in the pyriform sinus. A second half teaspoon of thick liquids with head turned to the left continued to show large residual in the pyriform sinus along with gross aspiration. It was recommended that the patient continue with the GJ tube feedings exclusively. He was receiving tube feedings from 1900 to 0800 hours and at 1200 to 1330 hours. This schedule was designed to minimize disruption to rehabilitation therapies.

At the time of discharge from the stroke rehabilitation program almost 2 months after his initial presentation with the stroke, the patient was close to be an independent with a walker, requiring only minimal assistance with tub transfers and being discharged home with the G-J tube feedings.

Q4. What would be the next step?

Answers

1. The next step would be to carefully follow the patient and later repeat the VMBS hoping to get this gentleman off of the G-J tube feedings.

Case Study (continued)

A second VMBS was performed 4 months post-stroke onset. This showed that the pudding and thick fluids still resulted in large residue being present in the pyriform sinus with moderate to large aspiration from both. No cough was heard when he aspirated. With thin liquids large residue was present in the pyriform sinus resulting in laryngeal penetration and aspiration. This VMBS study indicated that he was still unsafe with swallowing and required tube feedings. Upon laryngeal penetration of thick liquids he was asked to cough and this cleared the penetration. A third VMBS was conducted 2 months later (6 months post-stroke onset). Poor epiglottic motion and weak pharyngeal peristalsis were present. It was noted that there was marked pyriform sinus residue and mild vallecular residue with thin and thick liquid barium and pudding consistencies. Aspiration was demonstrated with these consistencies but they did not elicit a cough. It was recommended that he continue to be fed through the GJ tube.

Six months later (1 year subsequent to his lateral medullary stroke) this man had his fourth VMBS study. The SLP noted a significant improvement in his swallowing. He continued to show large residue in the valleculae and pyriform sinuses, more so in the latter. Double swallowing with consistencies of thick liquids and pudding allowed him to clear the residue. He was able to swallow small amounts of thin liquids, with double swallowing, with no aspiration. He had trouble with bread and cookie consistencies.

Q5. How would you manage his swallowing problem at this stage?

Answers

1. The patient should be placed on a pureed diet with thick fluids but not allowed bread or cookie consistencies
2. Care needs to be taken with thin liquids and it would be best to not allow him to drink thin liquids but continue to provide additional fluid through the G-J tube.

Case Study (continued)

The patient was put on a pureed diet with thick fluids but was not allowed bread or cookie consistencies. He was still maintained on GJ tube feedings primarily at need, but this was reduced to allow for his oral feedings.

A fifth and final VMBS was performed 6 months later, more than 18 months following the onset of his stroke. Most of the thick liquid barium was transferred into the esophagus; however, there was impaired pharyngeal peristalsis so that after swallowing a minimal residue remained in the valleculae and a minimum to moderate residue remained in the pyriform sinus. No laryngeal penetration was noted. With pudding, bread, and cookie consistencies the results were the same. With thin liquids there was occasional episodes of trace laryngeal penetration and trace aspiration. After the swallows minimal residue was in the valleculae and minimum to moderate residue was in the pyriform sinus. Results with thin liquids from a cup were similar.

Q6. What would you do at this stage?

Answers

1. The patient can now be fed orally for all consistencies.
2. Care still needs to be taken with thin liquids with the patient double swallowing and throat clearing after the fact.
3. He would need to be carefully monitored for any future episodes of pneumonia.

Case Study (continued)

It was noted that despite abnormalities in the pharyngeal phase of swallowing, there was no laryngeal penetration or aspiration with thick fluids, pudding, bread or cookie consistencies. It was suggested that the patient continue to use supraglottic swallowing as well as throat clearing to remove any material that may have penetrated. It was recommended that he be placed on a diet of thin liquids and regular solids and no further interventions were felt to be necessary. The GJ tube was subsequently removed. At one time during his post-stroke period, cloxacillin was ordered for a subcutaneous skin infection around the GJ tube.

E4. Dysphagia Management Post Stroke in Nursing Home Patient

E4. Dysphagia Management Post Stroke in Nursing Home Patient

E4.1 Low-Risk Feeding Strategies

Case Study

You are asked to see an 82 year old female who had a large right hemispheric stroke 2 years previously and who is now in a nursing home. Initially, while in hospital she had trouble with dysphagia, initially had an NG tube in place but eventually graduated to a regular diet. She has a left spastic hemiparesis, can ambulate with a cane and the assist of one person, does not have use of her left arm and hand, and still tends to neglect items on the left side. She has recently suffered two episodes of pneumonia, both of which necessitated admission to an acute care hospital.

You are able to observe her eating lunch on the day you arrive at the nursing home. Lunch is served in a large room with a number of the nursing home residents which is regarded as important to ensuring residents are able to regularly socialize. The patient herself is a very slow eater and so the nursing home has kindly assigned young students to help feed her. Initially, the patient was able to feed herself but increasingly she has come to depend largely on help with feeding. She is provided with a regular diet. She sits in her chair, frequently leaning back and watching patients and staff, mostly on her right side. The students are attentive and knowing that she tires easily, they try to get her to eat as much as possible before she asks to go back to her room. Hence, they feed her utilizing a tablespoon until she complains of being tired at which point she is taken to her room and allowed to lie down and rest. Her family was pleased at the attention given her to ensure she receives adequate nutrition and a couple of times per week they will assist.

Q1. What recommendations would you make with regard to feeding strategies?

Answers

1. The patient should be in a quiet environment.
2. The patient should be sitting at a 90 degree angle with the neck in a slightly flexed forward position.
3. The patient should be fed by seated individual at eye level with the patient.
4. The patient should be fed slowly with a teaspoon – no tablespoon.
5. The feeding assistant should assure that the patient swallows oral contents before offering more food.
6. Once done the patient should be kept upright for a half-hour.

Discussion

Low-Risk Feeding Strategies for Dysphagia

The Heart and Stroke Foundation Dysphagia Guidelines noted that, *"Stroke survivors should be encouraged and assisted to feed themselves. Individuals with dysphagia who are fed are approximately 20 times more likely to develop pneumonia than those who feed themselves (Langmore et al. 1998). Therefore, if dysphagic individuals cannot feed themselves independently, hand- over-hand support should be provided from an eye level position. If full feeding assistance is necessary, it should be provided using low risk feeding strategies.*

Routine use of low-risk feeding strategies can prevent serious health problems and improve the quality of the experience for the person being fed. All health care professionals involved in feeding dysphagic individuals should also be able to deal with emergencies, such as choking, which may occur during feeding." Guidelines for low-risk feeding practices are summarized in the following Table.

Heart and Stroke Foundation of Ontario Guidelines for low-risk feeding practices (2002)

- Check the food tray to ensure the correct diet type has been provided.
- Ensure the environment is calm during meals and minimize distractions.
- Position the stroke survivor with the torso at 90degree angle to the seating plane, aligned in mid-position with the neck slightly flexed.
- Support the stroke survivors with pillows if necessary.
- Perform mouth care before each meal to remove bacteria that have accumulated on the oral mucosa.
- Feed from a seated position, so that you are at eye level with the stroke survivor.
- Do not use tablespoons. Use metal teaspoons, never plastic for feeding individuals with bite reflexes.
- Use a slow rate of feeding and offer a level teaspoon each time.
- Encourage safe swallowing of liquids by providing them with wide-mouth cup or glass or in a cut-down nosey cup, which helps prevent the head from flexing backward and reduces the risk of aspiration. Some individuals may benefit from drinking through a straw.
- Ensure that swallowing has taken place before offering any additional food or liquid.
- Observe the stroke survivor for any signs or symptoms of swallowing problems during and for 30 minutes after the meal.
- Perform mouth care after each meal to ensure that all food debris is cleared from the mouth.
- Position the patient comfortably upright for at least 30 minutes after each meal to promote esophageal clearance and gastric emptying and to reduce reflux.
- Monitor the oral intake of the stroke survivor with dysphagia: note any food items that are not consumed and ensure that intake is adequate, especially important in individuals receiving a thickened-liquid diet.
- Document the patient's intake, any changes in swallowing status and any self-feeding problems.

References

Heart and Stroke Foundation of Ontario. Improving Recognition and Management of Dysphagia in Acute Stroke. 2002.

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E5. Nutritional Issues Following Stroke

E5. Nutritional Issues Following Stroke

Case Study

A 75- year old female suffered a small subcortical stroke resulting in dysarthria and clumsy hand syndrome. On admission to rehabilitation 10 days following the onset of symptoms, the patient was observed to appear thin and had only been consuming about half of her meal trays while on the acute service. She was safe with a regular diet. A dietitian was consulted and an assessment was completed within several days.

History: *She has been living on her own for several years in a seniors apartment since her husband died. She rarely cooks anymore but receives 4 hours a week of home-care services and has meals provided by Wheels (an external agency) 3x/week. She does not have a scale and is unsure if she has lost any weight over the past 6 months. She ambulates independently but uses a category II walker for safety. She reports her appetite is "fair". She has no problems with nausea or vomiting.*

Physical: *Ht: 160 cm wt: 47 kg. (6'3", 103.4 lbs.) Her shoulders have a squared-off appearance. There is loss of fat in the interosseous and palmar areas of the hand.*

Biochemistry: *Normal Reference range included in paraentheses*
Total protein - 70 g/L (60-80 g/L)
Serum albumin - 37 g/L (>35 g/L)
Serum prealbumin - 0.20 g/L (>0.18 g/L)
Serum creatinine - 4 g/L (8 to 14 g/L)
Random glucose - 6 mmol/L (<11 mmol/L)

E5.1 Body Mass Index (BMI)

Q1. What is this patient's Body Mass Index (BMI)?

Answers
BMI or body mass index is often used to estimate whether a person is underweight, normal weight or overweight. While there are limitations associated with the use of BMI to detect overweight or obese individuals, it is useful to help quickly identify those who are underweight and may be malnourished.

BMI = **Weight** [in kilograms] / (**Height** [in meters])^2

$$BMI = 47/1.60^2$$

BMI = 18.4

There are many interpretations of BMI although values between 19.5 and 23.5 are considered optimum by most professionals. A BMI of 25 to 29.9 is considered overweight and one 30 or above is considered obese. A value less than 19.5 is considered to be underweight.

E5.2 Assessment of Nutritional State

Q2. What information can be used from her history and physical to help with the assessment of her nutritional state?

Answers

1. Based on the limited information given, we can assume that both her remote and recent intake have been less than optimum. She has been living on her own and doesn't cook for herself regularly. Her oral intake while in the hospital appears to be poor. Her low BMI also supports this finding.
2. Her physical appearance suggests evidence of both muscle wasting and fat loss.
3. The *biochemical data* we are given reveals few abnormalities; other than a low serum creatinine value.

Q3. What is this woman's nutritional status?

1. This woman presents with classic protein-energy malnutrition (PEM), or marasmus.
2. Key indicators are a low BMI, history of poor intake and a wasted appearance. It is important to note that in this condition, there are few abnormal biochemical values. Visceral proteins, such as albumin, frequently used to assess nutritional status, are often spared at the expense of skeletal muscle, which is used to help meet energy requirements. The low serum creatinine (4 g/L) suggests a loss of muscle mass, consistent with a low BMI.

Q4. Name some other nutrition assessment tools that can be used?

1. Subjective Global Assessment.
2. Mini-Nutritional Assessment.

Discussion

As we demonstrated above, nutritional status was established using all available data without the use of a formal or structured process. This method is common in usual clinical practice. This is largely due to the fact that there is no gold standard that has been established. Furthermore,

few methods of nutritional assessments have been previously validated and none have been validated for use on patients recovering from stroke. There are two methods which have been validated previously and have been used in published trials examining stroke. They are both suitable for clinical use.

A traditional approach to nutritional assessment, which includes a combination of anthropometric and biochemical measurements can also be used to determine nutritional status, although none of these methods have been validated. Careful attention should be paid to the limitations of the individual items that are used, and the reference ranges and cut-off limits that are chosen.

Other, more sophisticated techniques such as bioelectrical impedance analysis, whole body counts (of Potassium, Nitrogen, Phosphorous, Oxygen etc) can also be used to estimate body cell mass and fat free mass, but they are usually reserved for research purposes. Functional measures including grip strength, respiratory muscle strength and muscle fatigue tests are also rarely used in a clinical setting to assess nutritional state.

Q4-1. Describe the SGA.

Answer
The SGA is a method of nutritional assessment that was designed for use in the prediction of risk for complications following general surgery, based on pre-operative nutritional state (Detsky et al. 1987).

Discussion

Questions	***Answer***
What does it measure?	Nutritional status
What is the scale?	Two part clinical assessment History: focus on weight loss, edema, anorexia, vomiting, diarrhea, decreased food intake, chronic illness Physical exam: focus on jaundice, glossitis, cheilosis, loss of subcutaneous fat, muscle wasting, edema
What are the key scores?	Patients were classified as normally nourished (A), mildly malnourished or suspected of being malnourished (B) or severely malnourished (C) based subjective assessment
What are the strengths?	Quick, non-invasive, inexpensive
What are the limitations?	Specialized training required. Poor reliability due to subjective nature of the items assessed.

Q4-2. Describe the MNA

Answer
The MNA was developed as a screening and assessment tool to identify geriatric patients at risk for malnutrition (Guigoz et al. 1994).

Discussion

Questions	***Answer***
What does it measure?	Nutritional status-may be used for both screening and assessment
What is the scale?	18 items including Anthropometric measurements Global assessment (6 questions related to lifestyle, medication & mobility) Dietary questionnaire Subjective assessment (self-perception of health and nutrition)
What are the key scores?	Screening: scores < 12 possible malnutrition Sub total maximum score 14 Assessment: Scores < 17 indicates malnutrition Scores of 17 to 23.5 indicates at risk of malnutrition Sub total maximum score 16 Maximum score: 30
What are the strengths?	Provides prognostic information
What are the limitations?	Time-consuming to perform (about 20 minutes) Dependent on subject's ability to answer questions related to their diet history

E5.3 Protein and Energy Requirements

Q5. What are this patient's protein and energy requirements?

Answers
There are a number of different methods to estimate energy requirements. One of the most commonly used is the Harris Benedict equation, which estimates resting energy requirements using a patient's height, weight and age

M: 66.5 +13.8(W) + 5 (H) - 6.8 (age) = Kcal/day
F: 655 + 9.6 (W)+1.8 (H) - 4.7 (age) = Kcals/day

This patient would require 1,047 Kcals/day
655 + 9.6 (103) + 1.8 (63) – 4.7 (75)

A stress factor of 1.2 combined with an activity factor of 1.2 are used to estimate the total amount of energy required for repletion

1,047 x 1.2 x 1.2 = 1,508 Kcals/day

A simpler method of estimating energy intake is to use a standard requirement of 35 Kcals/kg

35 Kcals/day x 47 kg = 1,645 Kcals/day to achieve weight gain

Protein requirements are estimated to be 1-1.2 g/kg/day (47-56 g/day).

Q6. What diet would you recommend for this patient? What are the goals of treatment?

Answers

1. Given that this woman appears to be suffering from uncomplicated malnutrition and does not require dietary restrictions due to dysphagia or co-morbidities, *a high calorie, high protein diet* is indicated.
2. Weight gain is the goal of treatment.

Discussion

Given that this woman appears to be suffering from uncomplicated malnutrition and does not require dietary restrictions due to dysphagia or co-morbidities, *a high calorie, high protein diet* is indicated. Oral supplements and/or high energy/protein snacks should be included. Oral supplements come in many flavours and forms including liquids, bars and puddings. Supplements may be given at mealtime along with reduced portions, or between meals. Most oral supplements contain approximately 250 to 300 Kcal and 8 to 13 g of protein per 250-mL serving.

Calorie counts should be initiated to ensure that she consumes sufficient energy and protein.

Weight gain is the goal of treatment. If she consumes her estimated requirements daily, she should achieve a weight gain of 1 to 2 lbs per week.

Gariballa et al (1998) demonstrated that malnourished stroke subjects receiving oral supplements that provided an additional 600 Kcals/day and 20 g protein had significantly higher energy and protein intakes compared to subjects who consumed a regular hospital diet. At the end of follow-up (12 weeks), subjects in the supplement group had gained an average of 0.2 kg compared to subjects in the control group who had lost an average of 0.7 kg.

Q7. How who you monitor and assess the progress?

Answers

1. *Weekly weights* should be obtained for the duration of the inpatient stay.
2. *Calorie counts* should be continued intermittently to ensure that adequacy of intake is maintained.

Reference

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E6. Deep Venous Thromboembolism

E6. Deep Venous Thromboembolism

Canadian Stroke Strategy Guidelines 2008: Recommendation 4.2a – Venous Thromboembolism Prophylaxis

All stroke patients should be assessed for their risk of developing venous thromboembolism (including deep vein thrombosis and pulmonary embolism).

Patients considered as high risk include patients with inability to move one or both lower limbs and those patients unable to mobilize independently.

i. Patients who are identified as high risk for venous thromboembolism should be considered for prophylaxis provided there are no contraindications [Evidence Level B] (ESO).

ii. Early mobilization and adequate hydration should be encouraged with all acute stroke patients to help prevent venous thromboembolism [Evidence Level C] (AU, ESO, SCORE).

iii. The use of secondary stroke prevention measures, such as antiplatelet therapy, should be optimized in all stroke patients [Evidence Level A] (ASA, AU, NZ, RCP, SIGN 13).

iv. The following interventions may be used for patients with acute ischemic stroke at high risk of venous thromboembolism in the absence of contraindications:
a. low molecular weight heparin (with appropriate prophylactic doses per agent) or heparin in prophylactic doses (5000 units twice a day) [Evidence Level A] (ASA, AU, ESO);
b. external compression stockings [Evidence Level B] (AU, ESO).

v. For patients with hemorrhagic stroke, nonpharmacologic means of prophylaxis (as described above) should be considered to reduce the risk of venous thromboembolism [Evidence Level C].

Case Study

A 75 year-old man presents to the Emergency-Room with left hemiplegia, due to a right MCA stroke. 48 hours later the nurse alerts you to the fact that his left leg shows signs of swelling and erythema.

Q1. What do this patient's symptoms suggest?

Answer
1. This patient is showing signs of deep venous thrombosis (DVT).
2. The clinical features of DVT are present in less than half of patients with this condition.

E6.1 Incidence of Venous Thromboembolism in Stroke

Q2. Describe the incidence and impact of DVT in the stroke population in the absence of prophylaxis.

Answers

1. In the absence of prophylaxis, over 60% of dense hemiplegics develop DVTs, 9-15% suffer a pulmonary embolus, with a 1-2% mortality rate (Sioson et al. 1988).
2. In the acute phase, in the absence of prophylaxis the incidence is 50%.
3. Most are below the knee, asymptomatic and unlikely to lead to a pulmonary embolus.
4. Peak onset is 2 to 7 days post stroke.
5. The incidence of DVT on rehabilitation is less than 10% in the rehabilitation phase in the absence of prophylaxis.

Discussion

In the absence of prophylaxis, over 60% of dense hemiplegics develop DVTs, 9-15% have pulmonary emboli, with a 1-2% mortality rate (Sioson et al. 1988). Indeed, pulmonary embolism has been reported to be the fourth most cause of death in the 30 days following stroke, while the risk of thromboembolism still persists thereafter.

Studies of patients with acute hemiplegic stroke have shown a DVT incidence of approximately 50% within 2 weeks in the absence of heparin prophylaxis. The majority of DVTs affect the paralyzed leg and are asymptomatic. Further, approximately two thirds of DVTs are below the knee, in contrast to symptomatic DVTs, in whom the majority are proximal. 20% of distal DVTs extend into the proximal veins. Furthermore, over 80% of symptomatic DVTs involve the popliteal or more proximal veins. Peak onset for the development of DVT is between the second and seventh day after the onset of stroke. DVT is also present in a significant proportion of patients during the rehabilitation phase of stroke but with lower prevalence (12-40%) and incidence (less than 10%).

E6.2 Risk Factors for Venous Thromboembolism Post Stroke

Q3. The nurse asks you how one can tell which patients are at risk for developing a DVT?

Answers

1. Risk factors include degree of lower limb paralysis, older age, reduced consciousness, obesity, previous DVT and atrial fibrillation.

Discussion

High risk patients have been identified as having lower limb plegia, reduced consciousness, obesity and having a previous DVT (Imberti and Prisco 2005). The risk of DVT correlates with the degree of paralysis and is greater in older patients as well as those who have atrial fibrillation. Predilection for the paralyzed leg is probably explained by a combination of loss of the calf muscle pump and repeated minor trauma.

E6.3 Diagnosis of Deep Venous Thrombosis

There are few ways to know the pretest probability of having a DVT, one of which is the Wells scale. Using this scale (see table below), you can determine if a patient has a high (3 or more points), moderate (1 or 2 points), or low (0) pretest probability for developing a DVT.

Clinical Model for Predicting Pretest Probability for Deep-vein Thrombosis

Clinical Feature	Score
Active cancer (treatment ongoing or within previous 6 months or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden for more than 3 days or major surgery, within 4 weeks	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling by more than 3 cm when compared with the asymptomatic leg (measured 10cm below tibial tuberosity)	1
Pitting oedema (greater in the symptomatic leg)	1
Collateral superficial veins (non-vericose)	1
Alternative diagnosis as likely or greater than that of deep-vein thrombosis	-2

Note: In patients with symptoms in both legs, the more symptomatic leg is used.

Q4. What diagnostic tests would you initially use to confirm a clinical suspicion of a DVT? Describe briefly the role of each.

Answers

1. D-Dimer Assay: D-dimers are fibrin degredation products. Very sensitive but lacks specificity.
2. Venous Ultrasound: Test is nonivasive and can do serial testing; sensitivity 95% for proximal DVTs and 73% for distal DVTs.

Discussion

Venous Ultrasound

Ultrasound has a sensitivity of 95% in patients with symptomatic proximal DVT and 73% for distal DVT. Although the majority of DVTs that extend do so within the first week, serial testing may be used if the test is negative but the patient remains symptomatic.

D-Dimer Assay

This is a rapid, noninvasive and relatively inexpensive test. Fibrin is the main component of thrombus formation and fibrin degredation products include d-dimers. Consequently, D-dimers are frequently in the blood when venous thromboembolism is present. The positive d-dimer test is very sensitive but lacks specificity since d-dimers are also found in other disease states, including cancer, congestive heart failure and inflammatory conditions. In other words, one can relax when results are negative because D-dimer assays have a high negative predictive value (high sensitivity) but, when results are positive, the predictive value is poor because the presence of D-dimers could be caused by something other than deep venous thrombosis (poor specificity).

Q5. How can you make a definitive diagnosis of a DVT?

Answer
1. Venography
2. MRI

Discussion

Venography
Although venography is considered the definitive test for DVT, it is an invasive test in which contrast dye is injected into the legs veins. Diagnosis is made if an intraluminal-filling defect is noted.

A positive diagnosis of a DVT can only be made if the venogram is positive or there is a positive venous ultrasound at two or more sites of the proximal veins. A negative diagnosis for DVT can be made if there is a negative venogram, a negative d-dimer test, or a normal venous ultrasound assuming the venous ultrasound is accompanied by one of the following findings: 1) normal d-dimer test, 2) low clinical suspicion for DVT, 3) normal serial testing with testing conducted one week later.

E6.4 Prevention of Deep Venous Thrombosis Post Stroke

Q6. What is the evidence for preventive pharmacological treatments for DVT recommended in ischemic strokes?

Answers
1. There is strong evidence that prophylactic anticoagulation significantly reduces the incidence of deep venous thromboembolism, as compared to placebo.
2. Although there is a slightly higher risk of intracerebral hemorrhage, the benefit of prophylaxis far outweighs the risk.

Q7. The nurse wants to know which pharmacological agents are recommended for DVT prophylaxis?

Answer

1. There is strong evidence that low molecular weight heparin is more effective with less risk of hemorrhagic complications than unfractionated heparin.
2. Warfarin is an effective anticoagulant but is less reliable, more cumbersome to use and has more bleeding complications than LMW heparin for prophylaxis.

Discussion

Unfractionated Heparin

Unfractionated heparin has immediate onset of action, must be given parenterally, and its effects can be reversed rapidly. Bleeding is the most common adverse effect while osteoporosis and thrombocytopenia are uncommon side effects. Heparin inactivates thrombin and anti-thrombin and hence is non-specific in its action. It means it is also very individual specific. In 2 RCTs of acute stroke patients, low-dose unfractionated heparin (5,000 units s/c q8h) reduced the rate of DVT from 73-75% in the placebo group to 13-22% in the treatment group. There are no studies of prophylaxis in subacute or rehabilitation stroke patients. However, it's accepted use is to maintain while at high risk (i.e. bedridden, in a wheelchair, paralysis). There is no accepted stop date although the trend is for longer and more frequent use.

Low Molecular Weight Heparin (LMWH)

The clinical advantages of LMWH include predictability, dose-dependent plasma levels, a long half-life, and less bleeding for a given anthitrombotic effect. It has become more popular in the treatment of DVT because it reduces the hemorrhagic complications. LMWH is administered once or twice daily, both during the high-risk period when prophylaxis for DVT is recommended and also while waiting for oral anticoagulation to take effect in the treatment of DVT. LMW heparin has a quantifiable and predictable anticoagulant effect. It does not inactivate thrombin but does inactivate anti-thrombin. LMW heparin drugs include:

- Daltaperin (Fragmin) 5,000 units daily
- Tinzaparain (Innohep) weight adjusted or 4,500 units daily
- Enoxaparin (Lovenox) 30 mg BID or 40 mg daily

LMW heparin is easy to administer, there is no need to monitor and patient appear to prefer it when monitoring is required.

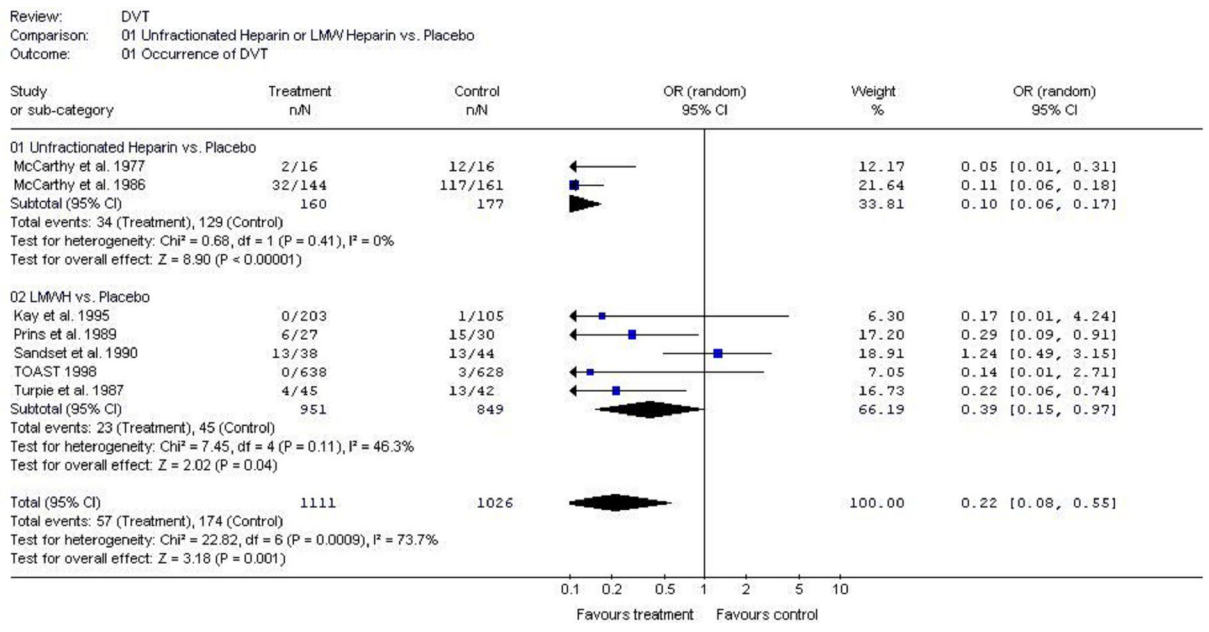
There are 5 RCTs comparing LMW heparin to placebo. 3 of the RCTs were positive and 2 were of no difference. A meta-analysis of 1900 stroke patients found significant difference (23 DVTs in Rx-group and 45 in placebo, p=0.04). There is an associated dose-dependent risk of intra- and extra-cranial hemorrhage with UFH. Evidence not definitive but LMW heparin appears to have more specific action with lower rates of hemorrhage than UFH.

Studies Evaluating Low Molecular Weight Heparin vs. Unfractionated Heparin

Author, Year, PEDro Score	n at randomization	Treatments	Results
Turpie et al. 1992 7	87	Low molecular-weight heparin vs. UFH heparin	+ (LMWH)
Dumas et al. 1994 8	179	Low molecular-weight heparin vs. standard heparin	-
Hillborn et al. 2002 8	212	Low molecular-weight heparin vs. UFH heparin	+ (LMWH)
PROTECT 2006 ns	272	Low molecular-weight heparin vs. UFH heparin	-
PREVAIL 2007 ns	1762	Low molecular-weight heparin vs. UFH heparin	+ (LMWH)

+ Indicates a reduction in the incidence of DVT compared to placebo/alternative treatment
- Indicates no difference in the incidence of DVT compared to placebo/alternative treatment.

Forest Plot of the Effectiveness of Heparin or LMW Heparin vs. Placebo in Preventing DVT Following Stroke



Advantages and Disadvantages of Heparin Use

Advantages	Disadvantages
• Acts immediately • Proven Efficacy in high risk patients • Can be neutralized • Reference drug	• Poor sc bioavailability when given in low doses • Short half-life • Repeated injections • Risk of thrombocytopenia (minimal with prophylaxis) • Risk of bleeding (minimal) • Not sufficiently effective in very high risk groups

Warfarin
Warfarin s the most widely prescribed oral anticoagulant. An international Normalized Ratio (INR) of 2.0-3.0 is sufficient for prophylaxis and treatment of venous tromboembolism while minimizing the risk of haemorrhage associated with higher INRs. The anthitrombotic effect of warfarin or the inability to expand and form clots is not present until approximately the fifth day of therapy; therefore, concomitant use of heparin is usually required during the transition in therapy.

Advantages and Disadvantages of Warfarin Use

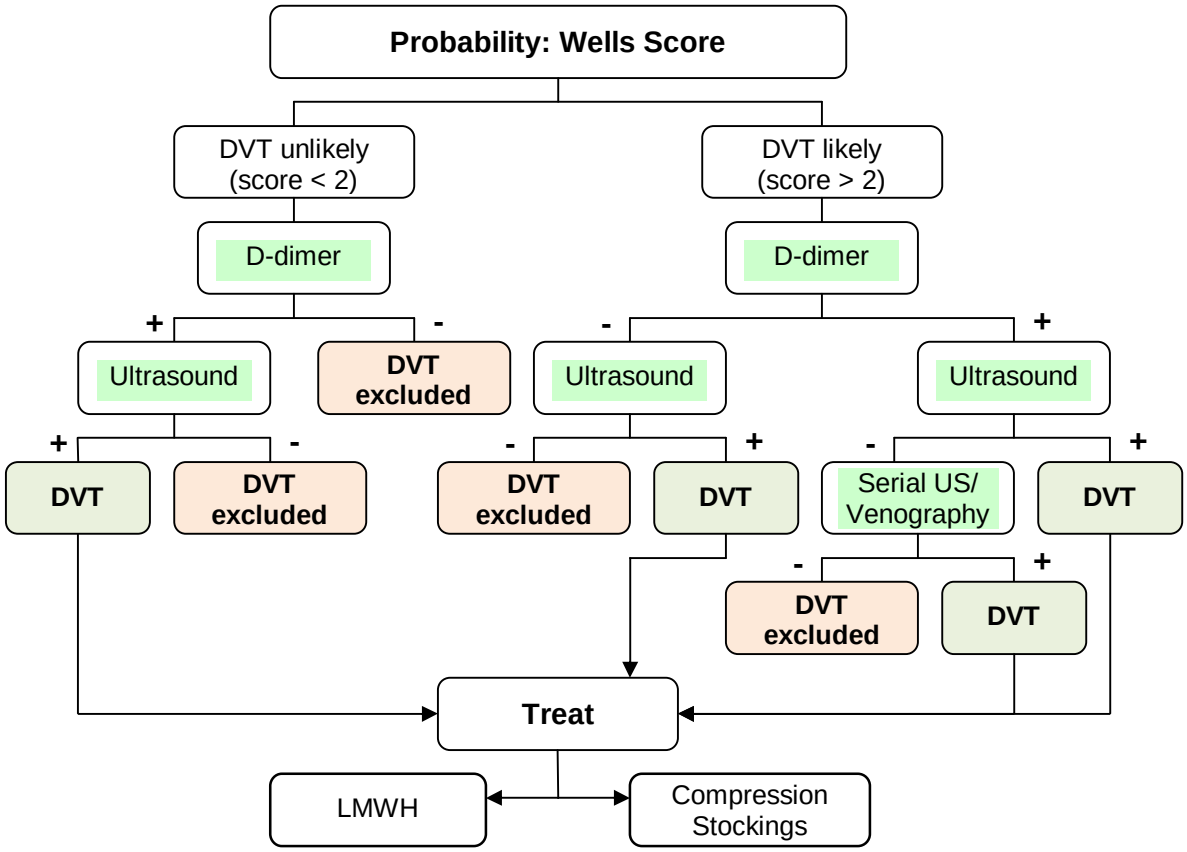
Advantages	Disadvantages
• Oral administration • Proven Efficacy	• Risk of bleeding • Delayed onset of action • Delayed neutralizing • Frequent monitoring necessary • Many drug interactions

Conclusions re Anticoagulation and Prevention of DVT Post Stroke
1. There is strong evidence that anticoagulation significantly reduces the incidence of DVT when compared to placebo.
2. There is strong evidence that LMW heparin is better than UF heparin for reducing hemorrhagic complications associated with anticoagulant therapy and decreasing the frequency of venous thromboembolism.

Q8. A resident asks you about the use of mechanical methods for preventing DVT, what can you tell them?

Answers
1. There are two physical forms of prophylaxis for DVT: graded compression stockings and intermittent pneumatic calf compression devices.
2. For both types, there is a moderate level of evidence that they are ineffective at reducing the risk of developing DVT.

DVT Assessment Algorithm



E6.5 Pulmonary Embolism

Case Study (continued)

You receive another call from the nurse who tells you that the patient is now experiencing chest pain, shortness of breath, and is not interested in participating in rehabilitation therapies.

Q9. What is the most likely diagnosis and how common is it in the stroke population?

Answer

1. Most likely a pulmonary thromboembolism
2. Relatively common following a stroke with leg paresis
3. Most severe and fatal PEs occur at 2-4 weeks post stroke.

Discussion
The patient is showing signs of pulmonary thromboembolism (PE), which may be a medical emergency: Pulmonary emboli account for 13% to 25% of early deaths after stroke and it has been reported that it is the fourth most common cause of death within 30 days after stroke.

Although they may occur as early as the third day post-stroke, fatal emboli are unusual in the first week, occurring most frequently between the second and fourth weeks, which is when they are most likely to be fatal. Patients who are more severely disabled are the most likely to be affected but PE may also occur in ambulatory patients.

The signs and symptoms of PE are varied and non-specific, which often leads to difficulties with diagnosis. The signs of PE can include sudden chest pain, shortness of breath, difficulty breathing or rapid breathing, coughing up blood, loss of consciousness, Tachycardia, pleural friction rub, and fever. Many cases are clinically silent with only 30% having the clinical features of DVT and only 70% demonstrating a DVT on venography. In most cases there are only a few clinical findings and a non-specific presentation with the major clinical complaints being malaise and fever.

Q10. The nurse asks you how she can tell which patients are at risk for developing a PE?

Answer
1. Factors which predispose to development of pulmonary embolism are the presence of a deep venous thrombosis, a paralyzed and immobile leg, failure to prophylactically treat with LMW heparin, previous DVT or PE and other risk factors such as cancer.

Discussion
There are a few ways to assess the pretest probability of having a PE, one of which is the Wells scale.

Criteria for the Calculation of the Wells Score

Criterion	Points Value
Clinical signs of DVT	+3
Alternative diagnosis less probable than PE	+3
Heart rate > 100 bpm	+1.5
Immobilization or surgery < 4 weeks ago	+1.5
Previous DVT or PE	+1.5
Haemoptysis	+1
Cancer	+1

According to this scale, a total score of < 2 indicates a low probability or PE; 2-6 indicates a moderate probability of PE; > 6 indicates a high probability of PE.

Q11. How would you make the diagnosis of Pulmonary Thromboembolism?

Answers

1. Diagnosis is most often made with ventilation-perfusion scanning and/or spiral CT scanning.

Discussion

Ventilation/Perfusion Scaning

A normal perfusion scan excludes PE but perfusion defects are non-specific with only about one third of those with defects actually having a PE. The probability that a perfusion defect is a PE increases with the size, shape, and number of defects as well as the presence of a normal ventilation scan.

Probability of pulmonary embolism based on ventilation-perfusion scan results and clinical suspicion in PIOPED study

Ventilation-perfusion scan results	Clinical suspicion pf pulmonary embolism*		
	Low	Intermediate	High
High probability	56%	88%	96%
Intermediate probability	16%	28%	66%
Low probability	4%	16%	40%
Normal/near-normal probability	2%	6%	0%

Adapted from the PIOPED investigators (Gill and Nahum 2000, PIOPED Investigators 1990).
** Percentage of patients with pulmonary embolism*

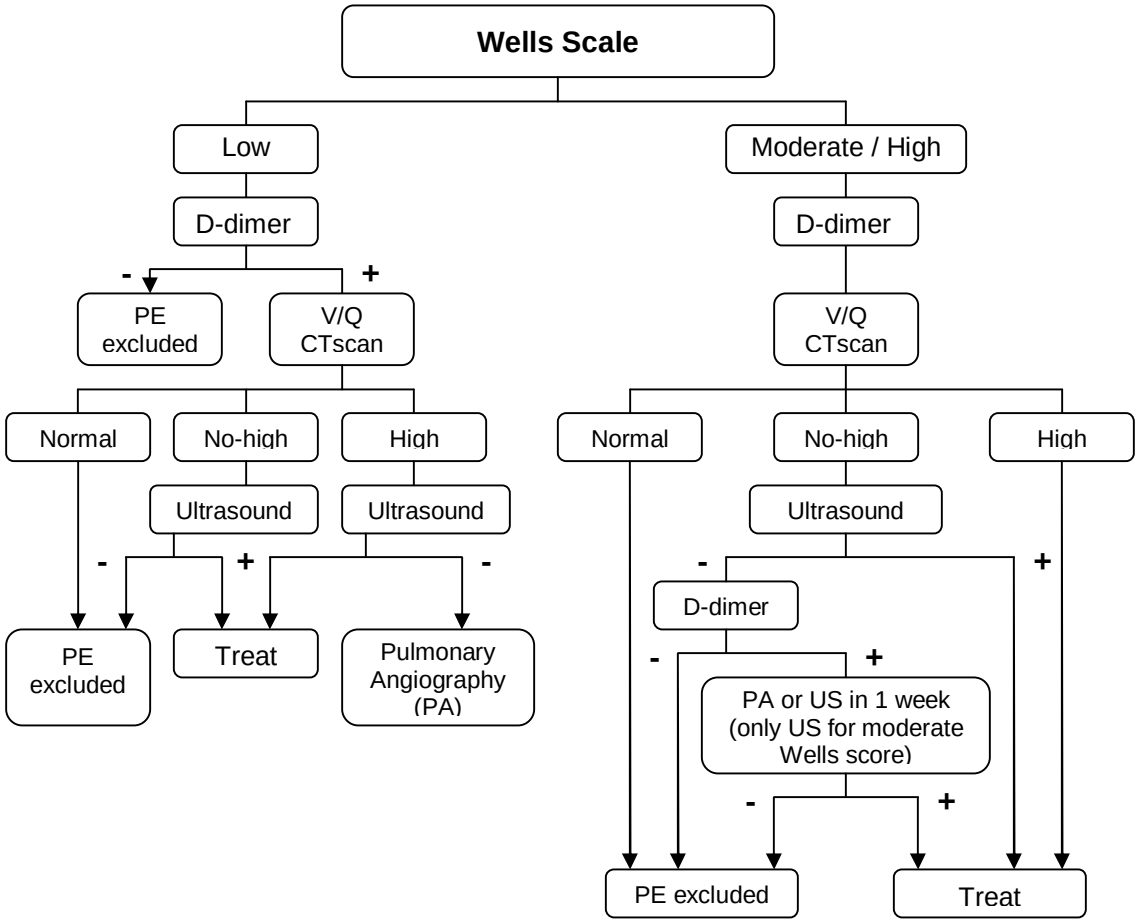
Pulmonary Angiography

A PA is the definitive diagnosis for PE. It is used when the V/Q scan is non-diagnostic but the clinical suspicion remains high. It is an expensive test that is associated with significant risk complications.

Spiral CT Scan

A quick spiral CT scan can scan the entire thorax in a single breath-hold. The sensitivity of this scan ranges from 64-93% and its specificity ranges from 89-100%.

PE algorithm



Case Study (continued)

The patient has been treated with warfarin and during the visit you notice that he is both less alert and slower to respond than he has been during previous visits. The physiotherapist also mentions that he was very difficult to work with that day.

Q12. What would you be concerned with considering the patient is being anticoagulated?

Answer

1. Sudden change in a patient on anticoagulation is always a concern. Bleeding is the main complication and with decreased alertness an intracranial hemorrhage is a possibility.

2. A CT scan should be ordered to rule out intracranial bleeding.

Discussion

It is important to consider secondary effects when you are treating a patient with anticoagulants. For example, Hemorrhage risk is well described in patients taking warfarin. In addition to intracranial haemorrhage, extracranial sites such as the gastrointestinal tract, genitourinary tract, and soft tissue are common sites for hemorrhage. Patients who have had previous hemorrhagic events while receiving warfarin are also at increased bleeding risk. Other risk factors include liver and renal disease, hypertension, cancer, and stroke. Regarding this patient, you must order a CT scan in order to search for intracranial bleeding.

Case Study (continued)

The CT shows a new small intracerebral haemorrhage. The warfarin is discontinued.

Q13. The resident asks you how long you should wait before restarting anticoagulation therapy after a warfarin-associated intracerebral haemorrhage.

Answer

1. Once the patient has stabilized consideration should be given to restarting the Warfarin, particularly if there is an ongoing risk of PE recurrence.
2. Decisions need to be made on a patient-by-patient basis. Factors which need to be taken into consideration are the general health of the patient, age, risk of falls and ongoing risk of PE recurrence.

Discussion

Recurrent primary ICH after reinstitution of warfarin therapy occurs less frequently than does recurrent thromboembolic events in patients who do not restart warfarin therapy. However, reinitiating anticoagulation therapy has been found to be associated with an increased risk of intracerebral haemorrhage and extracranial hemorrhagic complications (Classen et al. 2008). After an episode of WAICH, the clinician deciding whether to restart anticoagulation therapy should weigh several factors, including the patient's risk of falls, general medical condition, and other risk factors for systemic hemorrhage. Patients without these risks may benefit from reinstitution of warfarin therapy as failure to do so might unnecessarily subject them to thromboembolic complications.

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E7. Venous Thromboembolism Case Study

E7. Venous Thromboembolism Case Study

E7.1 Prophylaxis of Venous Thromboembolism Post Intracerebral Hemorrhage

Case Study

36 year old male admitted to stroke rehabilitation program and discharged 6 weeks later. 5 weeks previously he had suffered an intracerebral hemorrhage with right sided weakness. CT scan revealed a bleed in the left putamen and both caudate heads with compression of the ventricles. Etiology of the bleed was felt to be related to hypertension.

Q1. You know that the patient has a high risk for developing DVT, the nurse and the resident want to know if patients should be prophylactically anticoagulated after intracerebral hemorrhage?

Answer
The patient can be initiated on a typical LMW heparin prophylactic regimen without concern about increasing the size of the intracerebral hemorrhage.

Discussion
Eckman et al. (2003) try to answer this question. They used a Markov state transition decision model stratified by location of hemorrhage (lobar or deep hemispheric) and the effectiveness was measured in quality-adjusted life years (QALYs). Mark et al. (2003) found that for patients with prior lobar ICH, withholding anticoagulation therapy was strongly preferred, improving quality-adjusted life expectancy by 1.9 QALYs. For patients with prior deep hemispheric ICH, withholding anticoagulation resulted in a smaller gain of 0.3 QALYs. In sensitivity analyses for patients with deep ICH, anticoagulation could be preferred if the risk of thromboembolic stroke is particularly high.

Wasay et al. (2008 concluded that subcutaneous heparin in doses of 2500-5000 units twice daily during the acute phase in patients with ICH may be safe for DVT prophylaxis. However, it was not superior to elastic stockings in a non-randomized comparison to prevent DVT.

Subcutaneous heparin use in acute ICH was tested in a randomised controlled trial in which 5000U TID was started at 2, 4 and 10 days post-ictus (?REFERENCE). Treatment initiation on day 2 significantly reduced the frequency of deep venous thrombosis, with no concomitant increase in haematoma expansion.

Q2. Do you know of any other methods to prevent DVT in hemorrhagic strokes instead of pharmacological management?

Answer
There is moderate evidence that a combination of graded compression stockings and intermittent pneumatic devices reduce the risk of development of asymptomatic DVTs in patients with hemorrhagic stroke.

Discussion

Prevention of DVT Through Mechanical Methods
The use of physical forms of prophylaxis, including graded compression stockings (GCS) or TEDS has been questioned and debate continues over the risks and benefits of this treatment for stroke patients. The mechanism by which TED stockings reduce the risk of DVT is not entirely well-understood (Amaragiri and Lee 2000). Graduated compression stockings compresses the surface veins, keeping their diameter small, and forcing blood into the deep vein system. GCS can accelerate the velocity at which the blood flows through the deep veins, which helps to relieve the symptoms associated with venous insufficiency. Although seen as a relatively benign intervention, their use has been associated with side effects as serious as skin ulceration and necrosis. A Cochrane review by Amaragiri and Lee (2000) suggests that there is a significant decrease in DVT risk among post-surgical patients who wore the stockings, although an RCTs involving stroke patients was not included in this review (Muir et al. 2000).

Mazzone et al. (2004) authored a Cochrane meta-analysis and reported on the effectiveness of compression stockings and intermittent pneumatic compression devices among RCTs investigating stroke patients specifically. The review included only two small RCTs, both of which had follow-up periods of 10 days or less. The authors concluded that there was insufficient evidence to support the use of physical methods in routine DVT prophylaxis. A recent review by Andre et al. (2007) also suggests that there is insufficient evidence to suggest that either elastic stockings or IPC devices are effective means to reduce DVT. The results from the CLOTS (Clots in Legs Or TEDS after Stroke) Trial is pending. Neither Muir et al. (2000) or Prasad et al. (1982) reported differences in the development of DVT between groups in their small studies evaluating two different treatment approaches.

The prevention of DVT among patients suffering from ICH can be problematic given that traditional anticoagulants are hazardous to patients already at increased risk for bleeding complications. The VICTORIAh investigators found that a combination of TED stocking and IPC was more effective than TED stockings alone for preventing asymptomatic DVT. There were no cases of symptomatic DVT in either group during the study period. However, a significant percentage (19%) did not tolerate treatment with ICP well and stopped using it within 5 days.

E7.2 Prevention of Recurrent Pulmonary Emboli in Intracerebral Hemorrhage

Case Study (continued)

Six days after presenting with an intracerebral hemorrhage, the patient was diagnosed with a symptomatic DVT and a symptomatic pulmonary embolus.

Q3. The patient has developed a PE in association with an intracerebral bleed. What do you do now?

Answer
This patient should not be anticoagulated but the risk of PE recurrence; it is necessary to insert a vena cava filter.

Discussion
Another method to prevent PE is the vena cava filter. These filters are inserted in the vena cava to prevent the passage of emboli into the lungs. Some reports have demonstrated success rates as high as 96% in the prevention of PE (Greenfield and Michna 1988); however, vena cava filters are also associated with some risk. For example, they can become blocked or dislodged, both of which can increase the risk of an embolism.

Case Study (continued)
An IVC filter was subsequently inserted. Anticoagulation was initiated 7 days later, almost 2 weeks post SAH. An attempt was subsequently made to remove the IVC filter; however, attempted retrieval of the IVC filter was unsuccessful because of the presence of a large embolism which was trapped in the filter. The filter was therefore left in place. On admission to rehabilitation he had been initiated onto anticoagulation, initially with heparin and quickly switched over to Coumadin after the filter was put into place.

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E8. Post-Stroke Seizure Disorders

E8. Post-Stroke Seizure Disorders

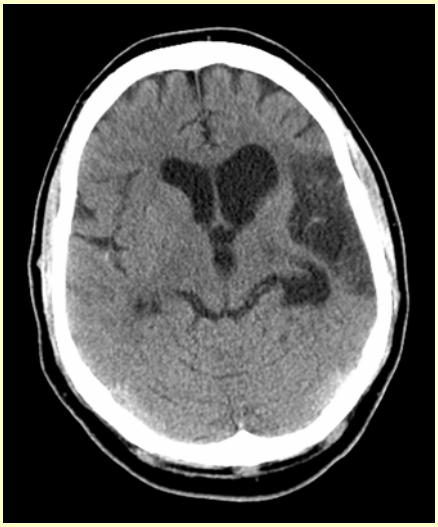
E8.1 Introduction and Case Study

Post stroke seizures may occur soon after stroke or be delayed; each appears to be associated with differing pathogeneses. Most seizures are single, either partial or generalized (Ferro and Pinto 2004. Wiebe and Butler (1998) noted that, "*Seizures are the clinical expression of excessive, hypersynchronous discharge of neurons in the cerebral cortex."*

Whether seizures worsen outcomes remains unclear. Vernino et al. (2003) reported new-onset seizure among patients with ischemic stroke to be an independent risk factor for mortality on multivariate analysis (Relative risk 1.81; 95%CI 1.16-2.83). Bladin et al. (2000) also reported higher mortality among patients with seizures at 30 days and 1 year, compared to patients who were seizure free (25% vs. 7% and 38% vs. 16%). However, the authors did not control for the confounding effects of stroke severity or Comorbidity. The results of other studies have not supported an increased risk of mortality (Labovitz et al. 2001, Reith et al. 1997).

Case Study

A 55-year old man is admitted into the inpatient rehabilitation unit 12 days post-stroke. He had a cardioembolic stroke involving the territory of the left MCA, which is affecting the cortex. He is consequently being treated with warfarin. During the second night in the unit, the patient's partner advised the nurse that he was making strong repetitive movements with his right arm and thought he was having a seizure. When the nurse arrived, the patient had a marked tendency to fall sleep and he felt very tired.



Q1. What do you think caused this patient's episode and what risk factors may have contributed?

Answers

1. He likely suffered an epileptic seizure (the clinical expression of excessive, hypersyncronous discharge of neurons in the cerebral cortex) (Wiebe and Butler 1998).
2. Younger patients and men are at increased risk for seizure activity post stroke (Arboix et al. 1997, Giroud et al. 1994). A lesion involving the cerebral cortex is a prerequisite for the development of epilepsy (Olsen et al. 1987).

Q2. Would you describe this as epilepsy?

Answers

1. Epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychological, and social consequences of this condition. The definition of epilepsy requires the occurrence of at least one seizure, although usually there needs to be more than one seizure.

2. One seizure in association with an enduring disturbance of the brain capable of giving rise to other seizures has been defined as epilepsy (Fisher et al. 2005).

E8.2 Incidence of Post-Stroke Seizures

Q3. Discuss the incidence of seizures post-stroke.

Answers

1. The incidence of seizures following ischemic or hemorrhagic stroke in earlier series is noted to be highly variable ranging from a low of 7.7% to a high of 42.8%. The average risk of seizure post-stroke is 10% within 9-10 years after stroke.
2. Hemorrhagic stroke patients have been found to have an almost 2-fold risk of developing a seizure following stroke compared to patients with an ischemic lesion (Bladin et al. 2000).

Discussion

Incidence of Post Stroke Seizures

Wiebe and Butler (1998) observed that the incidence of seizures following ischemic or hemorrhagic stroke in earlier series is noted to be highly variable ranging from a low of 7.7% to a high of 42.8%. This variability tends to be influenced by factors such study design, patient population, diagnostic methods, and follow-up (Black 1983, DeRueck et al. 1980, Holmes 1980, Louis and McDowell 1967, Meyer et al. 1971). Weibe and Butler suggest that high-resolution imaging such as with computed tomography (CT) and magnetic resonance imaging (MRI) has

improved the ability to identify and classify strokes, resulting in better estimates of their clinical course and consequences.

In comparison to earlier studies, recent reports reveal less variability in the risk of post-stroke seizures (PSS). The average risk of seizures is 10% within 9-10 years after stroke (Table 17.10), and well-conducted prospective studies report a 5-year cumulative incidence of 11.5% (Burn et al. 1997). At least two studies suggest a higher incidence of PSS (15-17%) in patients in rehabilitation units (Kotila and Waltimo 1992, Paolucci et al. 1997). It is not certain whether this reflects seizure ascertainment bias (e.g., seizures are less likely to be missed in closely observed patients), or a true increased seizure risk in this population (e.g., more extensive cerebral injury) or both. Also, Cordonnier et al. (2005) reported that pre-existing dementia increase the risk of late seizures defined as greater than one week post-stroke.

Black et al. (1983) reported 10% of all stroke patients developed seizures. Thirty-nine percent of seizures occurred within the first 24 hours of stroke onset, 57% within the first week and 88% within the first year. Sung and Chu (1986) demonstrated that for patients with intracerebral hemorrhages, seizure onset time was very similar to that reported for other stroke entities; 30% in the first 24 hours, 60% in the first two weeks and 90% in the first year. Sundaram and Chow (1986) reported that in subarachnoid haemorrhage (SAH) patients, 84% of PSS took place within the first 2 weeks of stroke onset.

The occurrence of post stroke seizures varies between 5% to 43%; or 10% on average. Seizures usually occur during the first 1 to 2 weeks following stroke. Consequently, a majority of seizure events will have already occurred before the patient is admitted to the stroke rehabilitation unit.

Seizures Following Hemorrhagic Stroke

There is no consensus as to whether patients suffering from hemorrhagic strokes have a higher incidence of seizures when compared to patients with infarctions. Some studies show evidence to support this concept (Kilpatrick et al. 1990, Vespa et al. 2003), while others dispute the notion that hemorrhagic strokes are associated with more PSS (Black et al. 1983, Olsen et al. 1987, Shinton et al. 1988). Reith et al. (1997) found a higher frequency of early seizures (within 14 days of onset) in patients with intracerebral hemorrhages when compared to those with cerebral infarction. However, in multivariate analysis, initial stroke severity was the sole predictor of early PSS and the apparent increased frequency of PSS with intracerebral hemorrhage reflected a higher initial stroke severity in this group of patients. Seizure incidence for subarachnoid hemorrhagic patients has been found to be as high as 24% (Sundaram and Chow 1986).

Seizures in Cortical vs. Subcortical Strokes

The results of some studies showed that PSS only occurred in patients with cortical involvement Lancman et al. 1993, Kilpatrick et al. 1990) while others did not (Gupta et al. 1988, Shinton et al. 1988). Olsen et al. (1987) found that a lesion involving the cerebral cortex, irrespective of size, was a prerequisite for the development of epilepsy. Kilpatrick et al. (1990) supported this concept by reporting an absence of seizure activity among 1,000 patients with subcortical vascular strokes. For patients with hemorrhagic strokes, cortical involvement appeared to be associated with the development of seizures, since deep-seated hemorrhages rarely cause seizures (Kirkpatrick et al. 1990, Olsen et al. 1987, Sung and Chu 1986).

E8.3 Types and Timing of Post-Stroke Seizures

Q4. What are the most common types of seizures after stroke and what is the relation between seizures and time since the onset of stroke?

Answers

1. Black et al (1983) reported that 39% of seizures occurred within the first 24 hours, 57% within the first week and 88% within the first year.
2. The overall percentage after stroke of focal seizures was 50%, generalized seizures 32%, focal seizures with secondary generalization 15%, and complex partial seizures 2,5%. (Wiebe-Velazquez and Blume 1993).

Discussion

Seizure Type Post Stroke

Wiebe-Velazquez and Blume (1993) totaled the frequency of various seizure types following stroke from seven studies. From the combined 231 patients, the overall percentage of focal seizures was 50%, generalized seizures 32%, focal seizures with secondary generalization 15% and complex partial seizures 2.5%.

E8.4 Treatment of Post-Stroke Seizures

Q5. Describe traditional treatment approach to post-stroke seizures.

Answer

1. There is consensus opinion that post-stroke seizures should be treated with anticonvulsant medication to prevent seizure recurrence.
2. Standard first-line therapy usually includes carbamazepine, valproic acid and phenytoin.
3. Phenytoin is known to interact with warfarin.
4. There is some concern that anti-epileptic drugs may impair recovery post stroke.
5. Benzodiazepines as an ongoing treatment should be avoided due to its sedating effects unless seizure activity is uncontrolled.

Discussion

Treatment of Post- Stroke Seizures

There have been few studies of the treatment of seizures post stroke and no definitive evidence recommending one treatment over another. There is consensus opinion that patients who have experienced seizures post stroke should be given preventative anticonvulsant medication to prevent seizure reoccurrence. Standard first-line therapy usually includes carbamazepine, valproic acid and phenytoin sodium. However, phenytoin is known to interact with warfarin, a commonly prescribed drug for patients who have suffered from a cardioembolic stroke. There is some concern that the use of antiepilectic agents may impair recovery post stroke (Camilo & Goldstein 2004), which should be a consideration in the decision of whether to treat an isolated

seizure. The newer anti-epileptic drugs such as lamotrigine may be better tolerated and have a better side-effect profile than some of the older drugs. Benzodiazepines as an ongoing treatment should be avoided unless seizure activity is uncontrolled due to its sedating effects.

Current stroke guidelines from the Stroke Council of the American Heart Association and the European Stroke Initiative also recognize the lack of evidence for treating stroke-related seizures and defer to consensus level evidence: "*Standard IV and oral antiepileptic drugs are in general use…there is no evidence that prophylactic anticonvulsive treatment is beneficial…few data concerning the efficacy of anticonvulsants in the treatment of stroke patients who have experienced seizures…*"(Adams et al. 2003, Broderick et al. 1999).

Only a single RCT has been conducted evaluating the efficacy of anticonvulsant therapy in which one treatment was compared with another. Although the results were not statistically significant the small sample size may have masked a real treatment effect of lamotrigine. Lamotrigine may be a better treatment option compared with carbamazepine since lamotrigine has relatively few side-effects, fewer potential drug interactions and does not require blood monitoring in monotherapy. There is moderate evidence that lamotrigine is superior to carbamazepine in the treatment of recurrent seizure post stroke.

E8.5 Treatment of Status Epilepticus Post Stroke

Q6. The nurse asks you how you would treat status epilepticus (just in case).

Answers

1. Benzodiazepines are considered the best first-line drugs for managing status acutely with a seizure control rate of approximately 79%.
2. Both Lorazepam and Diazepan given intravenously are acute treatment strategies, although Lorazepam may be more effective in terminating status epilepticus (59-89% VS 43-76%) and has longer lasting anticonvulsivant properties (12 hours VS 20 Minutes for Diazepam).
3. Midazolam 10 mg given by the buccal and intranasal route is an alternative where intravenous medications are difficult to give.

Discussion

Treatment of Status Epilepticus

On our stroke rehabilitation unit, benzodiazepines are considered the best first-line drugs for managing status epilepticus acutely with a seizure control rate of approximately 79%. Both lorazepam and diazepam given intravenously are equally acceptable acute treatment strategies, although lorazepam may be more effective in terminating status epilepticus (59-89% vs. 43-76%). Intravenous lorazepam is the preferred first line agent for seizure control due to its long lasting anticonvulsant properties. Lorazepam lasts 12 hours versus 20 minutes for diazepam, which places patients at risk of seizure recurrence unless a longer acting drug is given (A. Bluvol, personal communication). The intravenous approach is invasive and is not always preferred on the stroke rehabilitation unit. The newest benzodiazepine for seizure control is midazolam, administered by several routes; intramuscular, intranasal and buccal. There is a rapid onset of action (within five to 10 minutes), without accumulation or subsequent 'hangover

effect'. Metabolism is through a hepatic enzyme system with an elimination half-life of two hours. Studies of midazolam in pediatric seizure patients have shown midazolam given by the buccal or intranasal route is more, or as, effective as rectal diazepam and is the most reliably absorbed i.m benzodiazepine for seizure treatment. On our own unit, 10 mg midazolam given as 1 mL (5mg/mL) on each side of the mouth, between the teeth and inner aspect of the cheek, is part of a medical directive and can be given by nursing staff. However, further research is required to validate this treatment approach. A single RCT has examined the treatment of seizures, specifically post stroke.

E8.6 Impact of Anti-Epileptic Medications on Stroke Recovery

Q7. The patient's wife asks you if you need to treat the post-stroke seizures, that it seems to slow him down, and she is worried about side effects. What would you advise her?

Answers

1. There is some concern that the use of antiepileptic agents may impair recovery post stroke (Camilo and Golgstein 2004).
2. Benzodiazepines as ongoing treatment should be avoided unless seizure activity in uncontrolled due to its sedating effects.
3. There is consensus opinion that patients who have experienced seizures post stroke should be given preventive anticonvulsant medication to prevent reoccurrence.

E8.7 Phenytoin as a Treatment of Post-Stroke Seizures

Q8. The medical student asks you about treating post-stroke seizures with anticonvulsants. Is Phenytoin an appropriate treatment choice? What evidence is there for anticonvulsants?

Answers

1. Phenytoin is known to interact with warfarin; indeed, phenytoin interacts with many other drugs such us antibiotics.
2. There is no definitive evidence supporting one drug over another for the treatment of epilepsy in post-stroke patients. Standard first-line therapy usually includes carbamazepine, valproic acid, Lamotrigine and Phenytoin.
3. Gillad et al. (2007) showed that Lamotrigine may be better than Carbamazepine since Lamotrigine has relatively few side-effects, fewer potential drug interactions, and does not require blood monitoring in monotherapy.

E8.8 Driving and Post-Stroke Seizures

Q9. The patient's wife asks you about the possibility that her husband will be able to drive again. What can you tell her about epilepsy and driving?

Answer

1. The patient should be assessed by a neurologist and an EEG performed.
2. There needs to be an assumption he has suffered a single seizure event.
3. He will need to be seizure-free for at least 6 months before he can drive again, presuming the neurologist conducting the EEG concurs.
4. Individual circumstances may warrant prolonging or reducing the time period suggested.

Determining Medical Fitness to Operate Motor Vehicles: CMA Driver's Guide 7th edition, 2006

10.4 Seizures

As for all conditions, in all instances where a temporal recommendation is made, the time period should be considered a general guideline. Individual circumstances may warrant prolonging or reducing the time period suggested.

10.4.1 Single, unprovoked seizure before a diagnosis

Private drivers: These patients should not drive for *at least 3 months* and not before a complete neurologic evaluation — including electroencephalography (EEG) with waking and sleep recording and appropriate neurologic imaging, prefereably magnetic resonance imaging (MRI) — has been carried out to determine the cause.

Commercial drivers: Commercial drivers should be told to stop driving all classes of vehicles at once. For these drivers, there is a need for even greater certainty that another seizure will not occur while they are driving. As a minimum, commercial drivers should follow the private driver guideline and not drive private vehicles for at least 3 months after a single, unprovoked seizure. If a complete neurologic evaluation, including waking and sleep EEG and appropriate neurologic imaging, preferably MRI, does not suggest a diagnosis of epilepsy or some other condition that precludes driving, it is safe to recommend a return to commercial driving after the patient has been seizure free for 12 months.

10.4.2 After a diagnosis of epilepsy

Patients may drive any class of vehicle if they have been seizure free for 5 years with or without anticonvulsive medication.

Private drivers: Patients with epilepsy who are taking anti-seizure medication should not be recommended for Class 5 or 6 licensing until the following conditions are met:

• *Seizure-free period:* The patient should be seizure free on medication for not less than 6 months, unless seizures with altered awareness have occurred more than once a year in the previous 2 years, in which case the seizure-free interval should be 12 months. With certain types of epilepsy, this period may be reduced to not less than 3 months on the

recommendation of a neurologist, stating the reasons for this recommendation. The seizure-free period is necessary to establish a drug level that prevents further seizures without side effects that could affect the patient's ability to drive safely. The anti-seizure medication should have no evident effect on alertness or muscular coordination.
• *Patient compliance with medication and instructions:* The attending physician should feel confident that the patient is conscientious and reliable and will continue to take the prescribed anti-seizure medication as directed, carefully follow the physician's instructions and promptly report any further seizures. Medication compliance and dose appropriateness should be documented with drug levels whenever reasonably possible.

Physicians should advise epileptic patients that they should not drive for long hours without rest or when fatigued. Patients who require anti-seizure medication and who are known to drink alcohol to excess should not drive until they have been alcohol and seizure free for at least 6 months. These patients often neglect to take their medication while drinking. As well, alcohol withdrawal is known to precipitate seizures and the use of even moderate amounts of alcohol may lead to greater impairment in the presence of anti-seizure medication. Patients taking these drugs should be advised not to consume more than 1 unit of alcohol per 24 hours.

A patient who stops taking anti-seizure medication against medical advice should not be recommended for driving. This prohibition on driving may change if the physician feels confident that the formerly noncompliant patient, who is again taking anti-seizure medication as prescribed, will conscientiously do so in the future and if compliance is corroborated by therapeutic drug levels, when available.

Commercial drivers: It can be unsafe for commercial drivers who must take anti-seizure medication to operate passenger-carrying or commercial transport vehicles (Classes 1–4). For these drivers, there is a need for even greater certainty that another seizure will not occur while they are driving. Commercial drivers are often unable to avoid driving for long periods of time, frequently under extremely adverse conditions or in highly stressful and fatiguing situations that could precipitate another seizure. Unfortunately, seizures do sometimes recur even after many years of successful treatment.

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E9. Central Pain State

E9. Central Pain State

E9.1 The Incidence of Central Pain Post Stroke

Q1. What is the incidence of central pain post stroke?

Answer

1. Central post-stroke pain occurs in less than 2% of stroke patients.

Discussion

Central Post Stroke Pain (CPSP) is generally regarded as rare, occurring in less than 2% of strokes (Pagni 1984, Mucke and Miaciewicz 1987, Tasker 1990), although a recent study reported an 8% incidence among unselected stroke patients with 5% reporting moderate to severe pain (Andersen et al. 1995). Determining the actual incidence of CPSP is difficult since most studies have been based on selected cases (Hansson 2004). A prolonged gap between the stroke event and the onset of pain may also hinder a diagnosis.

E9.2 Pathophysiology of Central Pain Post Stroke

Q2. What is the pathophysiology of central pain post stroke?

Answers

1. The pathophysiology of central pain post stroke remains largely unknown.
2. Must be damage to the spino-thalamo-cortical pathway with disturbance in temperature and pain sensation.
3. Damage to the spinothalamiocortical tract resulting in denervation hyperexcitability of cortical or thalamic neurons are currently the most popular hypothesis for the pain.
4. Spontaneous or evoked dysesthesia, allodynia/hyperalgesia are manifestations of central post-stroke pain; paradoxical allodynia with associated numbness in the same territory.

Discussion

Central pain resulting from a stroke is often referred to as "thalamic pain" despite the fact that in many patients with CPSP, the cerebrovascular lesions do not involve the thalamus (Leijon et al. 1989a, Fields and Adams 1974, Loh et al. 1981, Agnew et al. 1983, Bowsher and Laheuerta 1985, Garcin and Lapresle 1969). Leijon et al. (1989a) noted that central pain states occurred following lower brainstem, thalamic and suprathalamic cerebrovascular events. CPSP is invariably associated with a lesion involving the spino-thalamo-cortical pathway with a disturbance in temperature and pain sensation (Andersen et al. 1995).

At present, the pathophysiology of CPSP states remains unknown. It is becoming increasingly clear that damage to the spinothalamicocortical pathway is associated with CPSP (Bovie et al. 1989, Jensen and Lenz 1995, Andersen et al. 1995, Vestergaard 1995, Dejerine and Roussy

1906) although not all patients with damage to this pathway experience pain (Andersen et al. 1995). CPSP is always associated with impaired sensory perceptions to cold and warm stimuli and to pinprick; these somatosensory functions are mediated by the spinothalamic tract (Bovie et al. 1989, Boivie 1992, Vestergaard et al. 1995). However, touch, 2-point discrimination and vibration sense, generally regarded to be mediated by lemniscal pathways in the CNS and often involved in CPSP states, may be intact (Boivie et al. 1989, Vestergaard et al. 1995). Vestergaard et al. (1995) reported that lemniscal system lesions are not necessary for the development of CPSP.

Most, but not all cases of CPSP, are associated with hyperalgesia and/or allodynia. This paradoxical presence of a sensory deficit in combination with hyperalgesia in that part of the body de-afferented by the stroke lesion suggests a central sensitization of third and fourth order CNS neurons as a result of loss of spino-thalamic (or thalamo-cortical) input (Vestergaard et al. 1995). Hyperexcitability of thalamic or cortical neurons could evoke the perception of pain. Vestergaard et al. (1995) noted that this hypothesis shares many features thought to be characteristic of other neuropathic pain syndromes associated with peripheral nerve lesions where spinal cord neurons that have lost their afferent input develop a central hyperexcitability (Bennett and Laird 1992, Dubner 1991, Wall 1991).

Conclusions Regarding the Pathophysiology of Post-Stroke Central Pain
Damage to the spinothalamiocortical tract appears to be necessary with denervation hyperexcitability of cortical or thalamic neurons being the most popular hypothesis for the pain. Spontaneous or evoked dysesthesia, allodynia/hyperalgesia are manifestations of central post-stroke pain.

Case Study

A 46 year old male sustained a right subcortical hemorrhage. He subsequently experienced gradual improvement in his motor function so that he was again ambulatory without support. However, since about one month after his stroke he had been troubled by a painful "pins and needles" sensation involving his entire left side, except for his scalp. He also had a burning pain ("like a sunburn") without superimposed shooting or lancinating pains. He was more comfortable at rest with a reported pain intensity of about 4/10. He had extreme sensitivity to light touch and other stimulation involving the left side of his body and in particular, just rubbing of his clothes while walking exacerbated his pain significantly. On neurological examination his cranial nerves were intact, motor tone was normal although there was a left sided pronator drift and mild weakness in left hip flexion. Fine finger coordination was normal bilaterally. On sensory examination there was blunting to pinprick involving his entire left side, except for his face. He had marked touch evoked pain or allodynia on the left side. The cold tuning fork was perceived as being even colder on the left side. Vibration sense was impaired in the left fingers and toes and he had difficulty detecting fine excursion of the left toes. Cortical sensation was intact and in particular, there was no sensory neglect. Gait testing revealed less arm swing on the left which was directly related to the fact that stimulation from his clothes increased his pain.

E9.3 Defining Post-Stroke Central Pain States

Q3. Define and Describe the Post-Stroke Central Pain State.

Answer

1. Central pain is pain due as a direct consequence of the stroke.
2. Described as burning or unpleasant sensations (parasthesiae) made worse with physical movements, emotional stress, cold, and light touch.
3. Associated sensory abnormality on affected side.

Discussion

Central pain is often described as a "burning" sensation in association with an unpleasant association of tingling, pins and needles, or numbness (Tasker 1990). It is often described in terms such as ripping, tearing, pressing, twisting, aching, pricking, and lacerating (Boivie et al. 1989, Leijon et al. 1989a, Anderson et al. 1995, Tasker 1990). The pain is generally constant and often associated with spontaneous paroxysms of pain (Boivie et al. 1989, Leijon et al. 1989a, Tasker 1990, Frese et al. 2006). It can also be exacerbated by physical movements, emotional stress, loud noises or voices, changes in the weather, cold and light touch (Boivie et al. 1989, Leijon et al. 1989a, Tasker 1990). All patients with CPSP generally have some kind of sensory abnormality on the affected side (Boivie et al. 1989). Decreased pinprick and threshold abnormalities to temperature detection are invariably present with touch, vibration, and two-point discrimination being more variably involved. Virtually all patient report spontaneous or evoked parasthesias and/or dysaesthesia (Leijon et al. 1989a, Andersen et al. 1995). Spontaneous dysesthesias occur in the majority of CPSP patients while almost all demonstrate some hypersensitivity to an external somatic stimuli (Leijon et al. 1989a). Hence the spontaneous pain seen in central pain states may be accompanied by further unpleasant effects induced by somatosensory stimuli known as hyperalgesia, allodynia and dysesthesia.

Q4. Define the terms "dysesthesia", "allodynia", and "hyperalgesia".

Answers

1. *Dysesthesia:* Unpleasant sensations, either spontaneous or evoked (Andersen et al. 1995).
2. *Allodynia:* Abnormally unpleasant somatosensory experience, often poorly localized, elucidated by normally non-nociceptive stimuli (Andersen et al. 1995).
3. *Hyperalgesia:* Increased pain response to a normally painful stimulus (Andersen et al. 1995).

E9.4 Treatment of Central Post-Stroke Pain

Q5. Describe an algorithm treatment approach to Central Post Stroke Pain.

Answer

1. Majority of CPSP are intractable to therapeutic interventions.
2. First line treatments include tricyclic antidepressants and antiepileptics.
3. Second line treatment is opioids.

Discussion

The majority of central post stroke pain states are intractable to therapeutic interventions. A number of interventions are commonly seen, the interventions which are the most likely to have a benefit are pharmacological interventions. Moreover, Henry et al. (2008) have noted, *"Our incomplete understanding of the mechanism underlying CPSP challenges the development of targeted treatment strategies (Hansson 2004). Moreover, the dearth of published data from large, well-designed clinical trials involving patients with CPSP has created a situation where guidelines for treatment are based upon "uncontrolled studies, clinical experience and expert opinion" (Gordon 2007)."* (Henry et al. 2008). Unfortunately, the medical literature does not provide us with anything more than consensus and anecdotal experience as to the best approach to treat CPSP.

Algorithm for Treatment of CPSP (Gordon 2007)

First-line treatment: Tricyclic antidepressants such as amitriptyline and nortriptyline as well as antiepileptics such as lamotrigine, gabapentin, pregabalin and carbamazepine.

Second-line treatment: Opioids such as morphine or levorphanol may be prescribed.

Not Recommended: Nonsteroidal anti-inflammatory drugs are not recommended. Local anaesthetics such as lidocaine, N-methyl-D-aspartate receptor antagonists including ketamine, cannabinoids, and botulinum toxin A are not recommended.

First-Line Treatments

First-line treatment should be tricyclic antidepressants such as amitriptyline and nortriptyline as well as antiepileptics such as lamotrigine, gabapentin, pregabalin and carbamazepine.

Amitriptyline: It has been noted that Amitriptyline, a tricyclic antidepressant, is usually the drug of first choice (Bowsher 1999, Leijon and Boivie 1989, Lampl et al. 2002). Based on the results of two RCTs, the SREBR (11th edition 2008) notes that there is conflicting evidence that treatment of amitriptyline results in a reduction of pain post stroke (Teasell et al. 2008). Henry et al. (2008) have noted that, *"its utility is limited by common adverse effect ts such as dry mouth, drowsiness and constipation, as well as more rare instances of urinary retention, orthostatic hypotension and cardiac arrhythmia (Hansson 2004). … the dose and blood levels of amitriptyline associated with pain relief are in the lower range of doses required for resolution of depression (McQuay et al. 1993)."*

Anti-Epileptic Treatments: There is moderate evidence that Lamotrigine may be an alternative to tricyclic antidepressants in the treatment of central post stroke pain.

SSRI Antidepressants: There is limited evidence that selective serotonin reuptake inhibitor fluxoxamine treatment is useful for the treatment of CPSP relatively early after a stroke.

Second-Line Treatments
For patients refractory to first-line treatment, opioids such as morphine or levorphanol may be prescribed.

Opioids: There is moderate evidence, based on one RCT, that intravenous morphine induced analgesic effects on some components of central neuropathic pain syndromes, but only a minority of patients may benefit from long-term opioid treatment. There is moderate evidence that high-strength u-opioid agonist levorphanol is effective in reducing pain in post-stroke patients.

Not Recommended Treatments
Nonsteroidal anti-inflammatory drugs are not recommended. Local anaesthetics such as lidocaine, N-methyl-D-aspartate receptor antagonists including ketamine, cannabinoids, and botulinum toxin A are also not recommended

Intravenous Lidocaine
Based on the results from a single RCT, there is moderate evidence that intravenous lidocaine results in short-term pain relief; however, the results are not maintained at 6 hours following treatment.

Non- Pharmacological Treatments
Nonpharmacological treatments of central post stroke pain are considered more adjuncts to treatment.

Motor Cortex Stimulation
There is limited evidence that motor cortex stimulation can provide long-term pain relief.

Case Study (continued)
When seen almost 18 months later, he had been tried on tricyclic antidepressants without benefit. He continued on Tegretol 100 mg BID which was the maximum dose he was able to tolerate, He also took Tylenol #2 x2 tablets up to 4 times daily with minimal relief. He was Lorazepam one tablet at night.

Q6. What options would be available now?

Answer
1. Second-line treatment would include stronger narcotic analgesics such as Oxycodone (short-acting or long-acting) or Morphine (long-acting).
2. Alternative anti-epileptics such as Dilantin, Gabapentin and Pregablin.

Case Study (continued)

The patient was initiated on a stronger analgesic Percocet 2 tablets qid and Neurontin (Gabapentin), gradually increasing dose to 300 mgs qid as well as Venlafaxine 75 mg daily. Duragesic was initiated and increased to 50 mcg per hour but was only receiving pain relief for the first 2 of 3 days. He experienced moderate but still inadequate pain relief. His Neurontin was increased to 1,600 mg tid without pain relief and Dilantin 350 mg daily was added to deal with seizures. Duragesic was increased to 150 mcg per hour every 2 days. Methadone was used to replace the Duragesic at a rate of 20 mg tid whch was increased to 50 mg q6h. Neurontin was decreased and Lyrica initiated at 300 mg bid; Venlafaxine was increased to 150 mg daily while Nabilone was given at 1mg daily.

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E10. Urinary Incontinence

E10. Urinary Incontinence

Canadian Stroke Strategy Guidelines: Recommendation 4.2d – Continence

i. All stroke patients should be screened for urinary incontinence and retention (with or without overflow), fecal incontinence and constipation [Evidence Level C] (RNAO).

ii. Stroke patients with urinary incontinence should be assessed by trained personnel using a structured functional assessment [Evidence Level B] (AU).

iii. The use of indwelling catheters should be avoided. If used, indwelling catheters should be assessed daily and removed as soon as possible [Evidence Level C] (AU, CSQCS, RCP, VA/DoD).

iv. A bladder training program should be implemented in patients who are incontinent of urine [Evidence Level C] (AU, VA/DoD).

v. The use of portable ultrasound is recommended as the preferred noninvasive painless method for assessing post-void residual and eliminates the risk of introducing urinary infection or causing urethral trauma by catheterization [Evidence Level C] (CCF).

E10.1 Normal Bladder Functioning

Q1. Describe the various areas of the central nervous system and peripheral nervous systems involved in the storage and emptying of urine.

Answer

Normal Bladder (detrusor) and urethral functioning involves the following areas of the central and peripheral nervous systems (Borrie1998):

1. The sympathetic nervous system relaxes the detrusor muscle while internal urethral sphincter control is maintained by sympathetic alphaadrenoreceptors.
2. Parasympathetic acetylcholine receptors mediate detrusor contracture.
3. Somatic (voluntary) nervous system innervates the pelvic floor muscles, including the external urethral sphincter.
4. A micturition centre in the brainstem (pons) informs when the bladder is filling and controls the sacral reflex when bladder filling reaches a certain level.
5. The micturition centre in the frontal lobes provides conscious input to the pontine micturition centre allowing the inhibition of urination until the time of voluntary control.

Discussion

Normal Bladder Function

Bladder (detrusor) and urethral functions are coordinated for the storage and emptying of urine (Borrie 1998). This involves areas of the central nervous system and multiple peripheral nervous systems as listed below:

1. Sympathetic nervous system relaxes the detrusor muscle while internal urethral sphincter control is maintained by sympathetic alpha-adrenoreceptors
2. Parasympathetic acetylcholine receptors mediate detrusor contracture.
3. Somatic (voluntary) nervous system innervates the pelvic floor muscles, including the external urethral sphincter.
4. A micturition centre in the brainstem (pons) inform when the bladder is filling and controls the sacral reflex when bladder filling reaches a certain level.
5. The micturition centre in the frontal lobes provides conscious input to the pontine micturition centre allowing the inhibition of urination until the time of voluntary control.

Normally, we are unaware of bladder fullness until a capacity of about 300 cc is reached. The need to void is then inhibited or controlled by the frontal lobes.

E10.2 Urinary Dysfunction Post Stroke

Q2. Describe the different types of urinary dysfunction occur post-stroke.

Answer

1. The most frequently occurring voiding abnormalities associated with a stroke have been identified as urinary frequency, urge incontinence and urinary retention (Marinkovic and Badlani 2001).
2. Urinary tract infection is not uncommon in stroke patients.

Discussion

Urinary Incontinence (UI) is defined as any involuntary leakage of urine (Abrams et al. 2002). Commonly seen post-stoke, UI is a strong predictor of poor functional recovery, discharge to long-term care, and limited resumption of social participation (Dumoulin et al. 2007).

Urinary Retention is commonly reported post acute stroke but less commonly seen in the rehabilitation phase. Stroke itself can cause retention but other contributing factors may include an inability to communicate the need to void, decreased level of consciousness or restricted mobility.

Q3. What three mechanisms are thought to be responsible for urinary incontinence post stroke?

Answers

1. Detrusor hyperreflexia. Urge incontinence and bladder hyperreflexia from damage to neuromicturition pathways.

2. Bladder retention. Overflow incontinence related to diabetic neuropathy or medications.
3. Stroke related barriers: paresis, cognition and language deficits.

Discussion

Gelber et al. (1993) suggested that three mechanisms were responsible for incontinence post stroke (van Kuijk et al. 2001). These included:

- Most common problems is detrusor hyperreflexia with urge incontinence and bladder hyperreflexia from disrupted neuromicturition pathways (frontal lobe or pontine micturition centers) (Nazarko 2003, Fader & Craggs 2003).
- Incomplete bladder emptying with overflow incontinence and bladder hyporeflexia from concurrent neuropathy or from the concurrent medications, such as diuretics (Fader & Craggs 2003).
- Incontinence from stroke-related motor, cognitive and language deficits, despite normal bladder function.

Subtypes of Urinary Incontinence

Type	Signs & Symptoms	Urodynamic Findings	Pathophysiology
Urge	Strong, sudden need to void.	Cystometry shows evidence of leakage with detrusor contractions only.	Detrusor instability.
Stress	Loss associated with effort or exertion (such as sneezing, jumping, or laughing).	Leakage during cystometry filling, without detrusor contractions.	Sphincter incompetence.
Mixed	Urge to void combined with loss on effort or exertion.	Leakage during cystometry filling, with and without detrusor contractions.	Sphinter incompetence combined with detrusor instability.
Overflow	Frequent small voids / loss with movement.	Postvoid residual (PVR) urine >150 ml.	Outlet obstruction and/or poorly contractile detusor.
Functional or Iatrogenic	Cognitive, language, and/or mobility deficits or the use of anticholinergic medications.		

E10.3 Detrusor Hyperreflexia Post Stroke

Q4. Discuss detrusor hyperreflexia.

Answer

Hyperactive bladder empties suddenly at a usually lower than normal volume.

Discussion
Cortical and subcortical strokes generally result in an unstable or hyperreactive detrusor (Borrie 1998). Borrie (1998) has noted that unstable detrusor contractions occur with little warning and result in symptoms of urinary urge incontinence. The bladder volume at which this occurs can be variable but it is usually lower than the volume at which a person with a normally functioning bladder would normally have a strong urge to void (Borrie 1998). Borrie (1998) notes that detruson hyperreflexia is not inevitable following a stroke. It has been noted that urinary incontinence is more common with larger strokes with a greater number of accompanying deficits and is closely associated with bowel incontinence, dysphagia and overall functional level, all markers of more severe strokes.

E10.4 Urinary Retention Post Stroke

Q5. Discuss urinary retention and potential contributing factors.

Answer
1. Urinary retention is common following an acute stroke (21-47%).
2. Potential contributing factors include difficulty with communication, mobility and decreased consciousness.
3. Other contributing factors may be a hypreflexic bladder, as seen in diabetic neuropathy, or an obstruction such as prostatic hypertrophy.
4. Occasionally urinary retention will persist in very severe stroke patients, often in patients with embolic strokes with no previous warnings.

Discussion
Urinary retention is commonly reported acutely following stroke; percentages of 21-47% post stroke have been reported (Doshi et al. 2003, Burney et al. 1996). Although stroke itself can cause retention, consequences of stroke including an inability to communicate the need to void, decreased level of consciousness, or restricted mobility may also be contributory (Marinkovic and Badlani 2001). Anecdotally, urinary retention is sometimes seen in the rehabilitation phase of stroke care, often in more severe stroke patients, although surprisingly little has been written on the subject. Retention may also be associated with prostatic hypertrophy in males, although usually there is a history of some difficulty prior to the stroke. Urinary retention can also be seen in patient with severe neuropathies, diabetics are the most common, with a hyporeflexic bladder. Incomplete bladder emptying with significant residual urine is a significant risk factor for the development of urinary tract infections.

E10.5 Other Factors Contribuing to Post-Stroke Incontinence

Q6. Discuss Other Factors Contributing to Post-Stroke Incontinence.

Answers

Stroke results in a number of factors which can affect continence, but which are often overlooked:

1. Communication difficulties, particularly an inability to communicate voiding needs, either due to aphasia, dysarthria or confusion/cognitive impairments.
2. Mobility problems, such as hemiplegia make some patients dependent on caregivers to void in a socially appropriate manner. Lack of caregiver support may also make it difficult to toilet the stroke patients quickly enough.
3. Post stroke depression and confusion may result in a failure to communicate the need for assistance.
4. Medications, such as diuretics can increase the frequency of the need to void; others can increase confusion, while still others such as antihypertensives may affect the autonomic nervous system leading to retention.

Discussion

Nazarko (2003) notes that it has been suggested that continence problems post stroke are a common consequence of immobility and dependency as opposed to neurological loss of control (Fader and Craggs 2003).

E10.6 Course of Urinary Incontinence Post Stroke

Q7. How common is urinary incontinence following stroke? What is the natural history?

Answer

1. Urinary incontinence has been reported in 37-79% of stroke patients in the acute phase and 17% of stroke survivors in the community.
2. Most incontinence resolves without treatment over 8 weeks although a significant percentage (14-19%) still have incontinence at 6 months.

Discussion

Brooks (2004) has noted that a stroke may exacerbate existing stress incontinence, a problem more common in women. Urinary incontinence is a common problem following a stroke with reported incidences ranging from 37% to 79% (van Kuijk et al. 2001, Forster and Young 2002, Brittain et al. 1985). The discrepancies in the reported rates likely arise from differing definitions of incontinence used and the populations under study. Urinary incontinence was reported among 17% (n=213) of non-institutionalized post stroke survivors, an average of nine years post stroke, compared to 7% of control subjects without stroke (Jorgensen et al. 2005) (OR=2.8, 95% CI; 1.5-5.2). Incontinence post stroke often resolves without treatment in eight weeks (Borrie et al. 1986, Brocklehurst et al. 1985). Borrie (1998) has noted that *"44-60% of stroke victims have urinary incontinence (Borrie et al. 1986, Brocklehurst et al. 1985, Nakayama et al. 1997) and of these, 17-22% will have premorbid urinary incontinence (Benbow et al. 1991, Borrie et al. 1986). 14-19% have persisting incontinence (Barer 1989, Nakayama et al. 1997) at six months."*

E10. 7 Risk Factors for Urinary Incontinence Post Stroke

Q8. What are the risk factors for urinary incontinence post stroke?

Answers

1. Severe strokes have a higher incidence of urinary incontinence.
2. Premorbid history of urinary incontinence, i.e comorbidities.
3. Motor weakness and mobility difficulties, including ataxia.
4. Altered level of consciousness.
5. Cognitive impairment
6. Depression
7. Elderly
8. Diabetic

Discussion

Patients who experience severe strokes are those that tend to have the greatest difficulty with incontinence (Nazarko 2003). Brittain et al. (1999) noted that non-neurological factors such as pre-morbid incontinence or stroke-related impairments, such as motor weakness, altered level of consciousness, cognitive impairment, ataxia and sensory lesions, in the presence of otherwise normal bladder function can contribute to the increase in incontinence. Normal age-related changes in bladder function may also independently affect recovery (Marinkovic and Badlani 2001). Jorgensen et al. (2005) identified depression, motor leg function and cognitive impairment as risk factors for incontinence. Gariballa (2003) found that patients with urinary incontinence at admission tended to be more undernourished and dehydrated, more impaired, at greater risk for infective complications and older then patients without urinary incontinence. A study of 935 acute stroke patients demonstrated that significant risk factors for post stroke urinary incontinence included age, severity of stroke, diabetes and comorbidity associated with other pre-existing disabling diseases (Nakayama et al. 1997).

E10.8 Consequences of Urinary Incontinence Post Stroke

Q9. Discuss the relationship between urinary incontinence and institutionalization.

Answers

Stroke survivors with persistent urinary incontinence tend to have greater disability, a poor prognosis, greater morbidity and mortality during their hospital stay and are more likely to be institutionalized.

Discussion

Brocklehurst et al. (1985) noted that while problems with bladder control and incontinence are common after stroke, they tend to resolve spontaneously in the majority of stroke patients. Recovery from post-stroke urinary incontinence is associated with less disability and lower rates of institutionalization than persistent incontinence (Patel et al. 2001). Bean et al. (2003) noted

an almost 2-fold difference in level of disability post-stroke among those who were incontinent versus those were continent ($p<0.001$). One study found that patients suffering from urinary incontinence on admission often had greater morbidity and mortality throughout their hospital stay and at 3 months post-stroke (Gariballa 2003). As noted by several investigators (Jongbloed 1986, Reding et al. 1987, Jawad and Ward 1999), recurring incontinence denotes a long-term poor prognosis for functional recovery. As Forster and Young (2002) note, "*treatment evidence specific to stroke is limited.*"

Brittain et al. (2000) reported that a significant higher proportion of community-dwelling persons who had experienced a stroke had more urinary symptoms compared to those that had never had a stroke (64% vs. 33%). The difference was statistically significant even after adjusting for age and sex differences between the groups. Stroke survivors were 1.77 times more likely to experience urinary symptoms than non-stroke persons. More stroke survivors reported a significant impact on lifestyle than did the non-stroke subjects. Twice as many stroke survivors than non-stroke persons reported that their urinary symptoms were moderate to severe.

Kolominsky-Rabas et al. (2003) examined the occurrence of urinary incontinence (UI) and the long-term effect UI had on subjects' prognosis and institutional status following stroke within a community-based population. Throughout the acute phase 41% of patients had full UI, 12% had partial UI and 47% had no UI (16%, 16% and 68% respectively at 12-months follow-up). In total, patients' institutionalized at 12-months follow-up included 45% of patients with UI compared to only 5% of patients without UI. The authors concluded that the risk of institutionalization 1-year post stroke is a "fourfold higher" for stroke patients with UI in the acute phase of rehabilitation.

E10.9 Urinary Tract Infection Post Stroke

Q10. How common are UTIs Post Stroke and what are risk factors for UTI?

Answers

1. UTI is the most common medical complication in stroke rehabilitation.
2. Risk factors include use of beta-blockers and high post-void residuals.

Discussion

Urinary tract infections (UTI) are the most commonly encountered medical complication associated with stroke rehabilitation (Roth et al. 2001). Although age greater than 65, male sex, a history of prior stroke, stroke syndrome, use of β-Block or antidepressants and a post void residual (PVR) of 150 mL or more have been reported as risk factors for the development of UTIs, Dromerick and Edwards (2003) only found an increased risk of UTI to be associated with β-Blocker use (OR & 95% CI: 4.01; 1.26-14.59) and PVR ≥ 150 mL (OR & 95% CI: 3.97; 1.24-9.53) among 120 stroke patients admitted to a rehabilitation service.

E10.10 Post-Void Residual Urine Testing

Q11. Discuss the importance of the post-void residual in diagnosis of bladder dysfunction post stroke.

Answer

1. PVR is able to determine if bladder emptying is complete.
2. PVR > 150 cc is generally regarded as abnormal.
3. Increased PVRs increases the risk of urinary tract infections.

Discussion

Borrie (1998) notes that "*A true void residual (PVR) urine test is critical to determine if bladder emptying is complete…although in/out catherization is the gold standard for determining the PVR urine, portable bladder ultrasound is practical, noninvasive and cost-effective (Chan 1993). A sterile culture from the catheter specimen would rule out urinary tract infection. Two consecutive residual urines of greater than 150mL suggest a significant degree of incomplete bladder emptying, and physical outlet obstruction should be ruled out by urology assessment with cystoscopy. There is no consensus as to what residual urine volume is definitively abnormal (Grosshans et al. 1993). Most would regard greater than 150 mL abnormal, but is depends to some degree on the volume of urine voided before catheterization.*"

E10.11 Diagnosis of Bladder Dysfunction

Q12. Provide a classification of bladder dysfunction post stroke.

Answers

1. Detrusor instability or hyperreflexia with urge incontinence.
2. Stress incontinence.
3. Overflow incontinence with incomplete emptying.
4. Mixed types of incontinence.
5. Functional incontinence, due to nonurinary factors.
6. Iatrogenic incontinence, due to drugs or restraints.

Discussion

Diagnosis of Various Bladder Dysfunction (from Borrie 1998)

History	Finding that May be Present	Residual	Pathophysiology Confirmed by Urodynamics
Urgency	Signs consistent with specific neurological disease	Low	Detrusor instability
Stress	Demonstrated during stress	Low	(Genuine) stress incontinence
Overflowing / incomplete emptying	Palpable bladder Enlarged prostate Urethral stricture Reduced anal sphincter tone Reduced anal sensation	High	Outlet obstruction and/or poorly contractile detrusor

Mixed	Variable	Variable	Mixed
Functional	Impaired mobility Impaired mental state Environmental factors	Low	Functional
Iatrogenic	Drugs Restraints	Variable	Iatrogenic

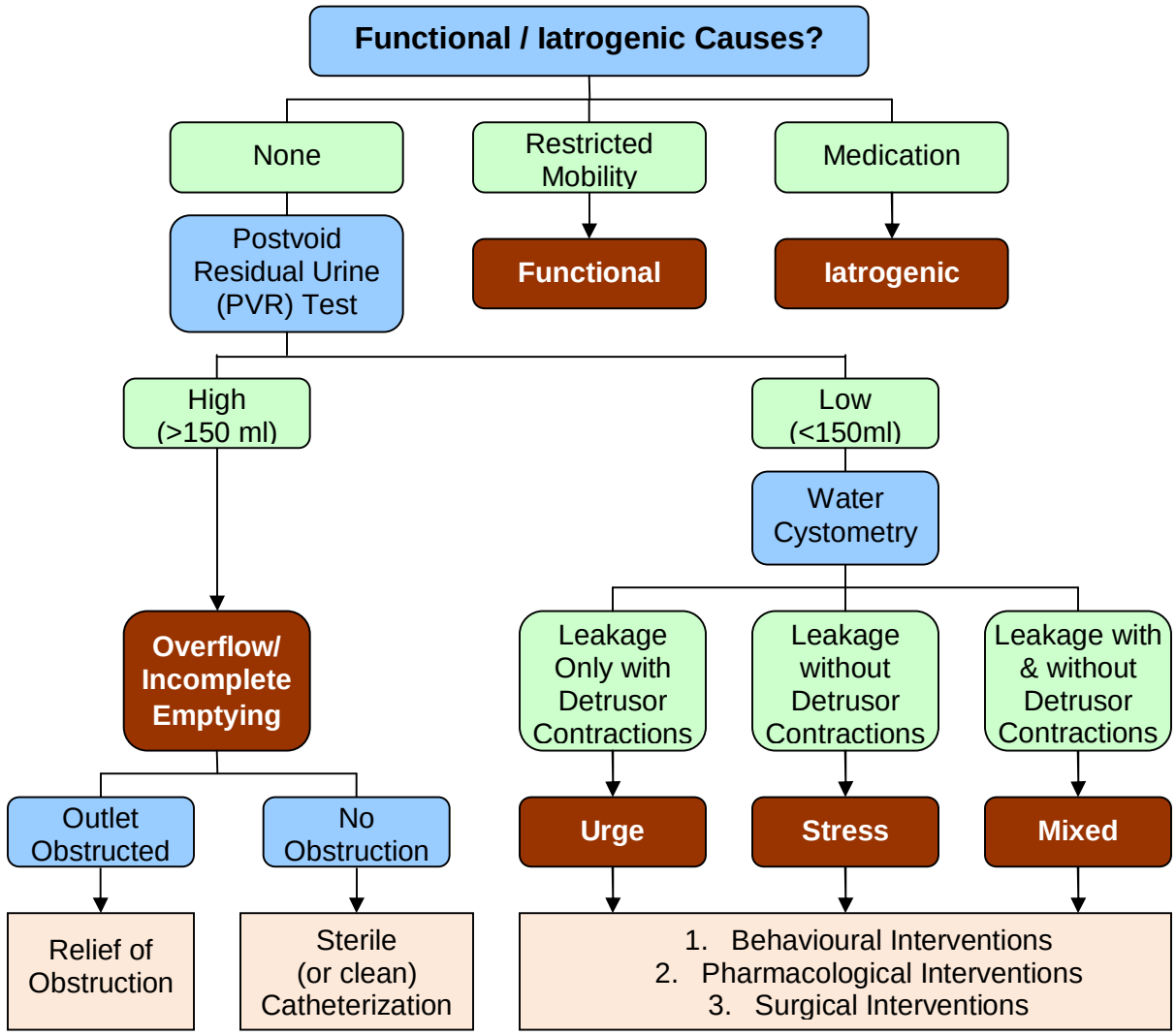
E10.12 Assessment and Management of Urinary Incontinence Post Stroke

Q13. Describe an algorithm for the assessment and management of urinary incontinence.

Answer

1. History of symptoms to determine which type of incontinence the patient suffers from (i.e., urge, stress, mixed, overflow, or functional).
2. Further assessment may include a physical examination as well as urodynamic investigations (such as a postvoid residual test or cystometry).

Assessment and Management of Post-Stroke Urinary Incontinence



E10.13 Treatment of Post-Stroke Urinary Dysfunction

Q14. What are the different treatment options for post-stroke urinary dysfunction?

Answers

1. Interventions used to treat UI depends on type of incontinence.
2. Initially behavioural interventions with scheduled voiding, pelvic floor exercises and biofeedback.
3. Pharmacological interventions
4. Catheterization.

Discussion

The intervention used to manage UI largely depends on the patient's history and type of incontinence. Due to the absence of strong evidence regarding treatment efficacy, Borrie (1998) has suggested using a stepwise approach, with behavioural interventions used as first line treatments and pharmacological and surgical interventions. This approach highlights the fact that patients should always be treated with the least invasive intervention option.

There are few RCTs evaluating the efficacy of bladder management to treat urinary incontinence post stroke, although the issue has been studied in other patient populations. The choice will often be dictated by the stroke patient's type of incontinence. In the absence of rigorous evidence, Borrie (1998) notes that a stepwise approach is best, beginning with behaviour intervention, progression to medication only when needed and considering surgical interventions only as a desperate last resort.

A recent Cochrane review (Thomas et al. 2008) investigating optimal methods for prevention and treatment of urinary incontinence after stroke in adults included the results from 12 trials (n=724). There was a wide range of interventions including behavioural interventions (timed voiding, pelvic floor muscles training), specialized professional input interventions (continence nurse practitioner care), complementary therapy interventions (acupuncture, moxibustion), pharmacotherapy (estrogen, oxybutynin, meclofenoxate) and physical therapy (sensory-motor feedback combined with timed voiding). A pooled analysis across all interventions combined was not performed. Two trials (Wikander 1998, Britain 2000b) offered some evidence supporting the use of input from specialized professionals using systematic methods to help evaluate, manage, and improve outcome of patients with continence complications. One trail (Brittain 2000b) suggested short-term and even long-term improvements in symptoms of urinary incontinence could be established through individualized care. While complementary interventions appeared to be effective compared with the placebo condition, small sample sizes and limited reporting of methodological details reduce the generalizability of the findings. While estrogen therapy was effective in reducing the number of incontinence episodes in a week, the therapy is generally contraindicated following stroke. There is limited evidence suggesting that the acute stage of rehabilitation has the largest impact on urinary incontinence following stroke. However, there is a paucity of evidence from studies done with stroke patients that helps direct specific practice guidelines. The authors concluded that further research is required.

There are a scarcity of trials that have evaluated urinary incontinence post stroke. Brittain et al. (1999) have highlighted the importance of distinguishing between urge incontinence and urinary retention, since the management of each is different. A wide variety of treatments is available in the management of incontinence following stroke. However, the pre-stroke continence status, small sample sizes and heterogeneity of treatments and outcomes assessed limit the generalizability of the findings. A single positive RCT was found for each of 4 differing treatments: Moxibustion, a program of prompted voiding, biofeedback assisted pelvic floor training and a FIM-focused rehabilitation unit.

E10.14 Behavioural Interventions for Incontinence Post Stroke

Q15. Discuss behavioural interventions for incontinence post stroke.

Answer

1. Scheduled voiding q 2-4 hours
2. Fluid restriction.

Discussion

Dumoulin et al. (2005) conducted a systematic review investigating the benefits of behavioural therapies used to treat urinary incontinence. The study included only four RCTs, a single cohort study and recommendations from three clinical practice guidelines. This study found limited evidence for the reduction of UI in male stroke patients using combination treatment including bladder retraining with urge suppression and pelvic floor exercises. The authors concluded that although there is increasing recognition of the benefits of using behavioral approaches as treatment for stroke patients with a high occurrence of continual UI, the evidence remains very limited for specific treatments used for stroke survivors with UI.

Bladder Training

Scheduled voiding programs follow a set schedule of voiding every 2-4 hours regardless of whether the patient "needs to go" because post stroke cortical awareness of bladder fullness is often reduced (Borrie 1998). Initiation of toileting in response to urgency, while shown to promote continence, often does not provide enough time to void especially when mobility is limited. Bladder training allows for lengthening of the voiding interval as the patient becomes consistently dry (Burgio and Paurgio 1986, Borrie 1998). However, Duncan et al. (2005) suggest that there is no evidence for or against a scheduled voiding program. These authors recommend an individualized bladder training program and the use of prompted voiding for incontinent stroke patients.

Scheduled Voiding programs follow a set schedule of voiding every 2-4 hours. Since cortical awareness of bladder fullness is often reduced following a stroke, voiding is attempted regardless of whether the patient "needs to go". Although initiation of toileting in response to urgency also promotes continence, it often does not provide enough time to void successfully, particularly when mobility is limited. Thus, scheduled voiding may be useful for treating patients with either cognitive impairment or urge incontinence. In a slight variation, bladder training also uses a voiding schedule but differs in that the voiding interval is lengthened as the patient becomes consistently dry.

Pelvic Floor Exercises are designed to strengthen the pelvic floor muscles through repeated contraction and relaxation of specific muscle groups. Although these exercises were originally developed to manage stress incontinence, rapid contractions have also been found to help patients suppress the "urgency wave" of the relax bladder contraction (Borrie 1998).

Biofeedback is an effective but time consuming intervention that uses either audio or video feedback to help patients identify the sensations of bladder fullness. The intension is that patients will later recognize these bladder sensations as an early cue that they need to void.

Fluid Intake

The total measurable fluid intake should be approximately 1500 – 1800 mL per 24 hours. The use of intravenous fluids or a feeding tube may result in fluid loads greater than 2L per day, which will in turn compromise bladder continence (Borrie 1998).

There is moderate evidence that prompted voiding significantly reduced the number of total incontinent episodes. There is moderate evidence that biofeedback-assisted pelvic training and behavioral therapy with weekly in-home visits from a nurse practitioner significantly reduces urinary accidents and incontinence. There is moderate evidence that a functionally oriented rehabilitation approach results in significantly less incontinence than a Bobath conventional approach. However, treatment of urinary incontinence post stroke has not been well studied. Further research is needed. There is moderate evidence that moxibustion can improve urinary tract symptoms. Prompted voiding and biofeedback-assisted pelvic training plus behavioral therapy and weekly in-home visits reduce incontinent episodes. Treatment with a functionally oriented rehabilitation approach vs. a Bobath approach for urinary incontinence post stroke has not been well studied.

E10.15 Pharmacological Interventions for Incontinence Post Stroke

Q16. Discuss pharmacological interventions for incontinence post stroke.

Answer

1. Pharmacologoical treatment will depend on the cause of the stroke.
2. The vast majority of bladder incontinence is due to detrusor hyperreflexia due to central loss of inhibition.
3. Drugs with anticholinergic actions are recommended in these cases, i.e Oxybutynin.
4. For overflow incontinence due to detrusor inactivity, a cholinergic such as Bethanecol is recommended.
5. For overflow incontinence due to outlet obstruction, an alpha-adrenergic blocker may be recommended.

Discussion

Drug therapy should be considered an adjunct to behavioural therapy and should only be used when behavioural interventions are either inappropriate or have had an adequate trial. The side effects of anticholinergic medications are often underestimated, particularly in the post-stroke population. Therefore, the goal of pharmacological therapy should be to use the lowest dose to achieve the optimal effect with the fewest side effects (Borrie 1998).

Borrie (1998) has noted that post stroke detrusor hyperflexia treated with drugs with various degrees of anticholinergic medications. These medications include Flavoxate, oxybutynin, propantheline and imipramine. These drugs should be started at a low dose and increased gradually over days, if not weeks.

Borrie (1998) has noted that **Flavoxate** is often worth trying initially because its direct smooth muscle action and limited cholinergic effect leads to fewer adverse side effects. **Oxybutynin** is an anticholinergic which is frequently used. **Propantheline** supposedly does not cross the blood-brain barrier, with a theoretical advantage over other drugs which can lead to confusion. **Tolterodine** is another anticholinergic which is said to have less influence on salivary gland function and therefore less likely to lead to dry mouth as a complication.

Borrie (1998) has also noted that for patients with poor detrusor contracture, **Bethanechol** may improve detrusor contracity (Sondra et al. 1979). It serves as an adjunct to intermittent catherization. Bethanechol is discontinued if residual urines do not decrease, there is excessive sweating, asthmatic attacks, congestive heart failure and abdominal cramps.

E10.16 Catheters for Bladder Dysfunction Post Stroke

Q17. Discuss the use of catheters for bladder dysfunction post stroke.

Answer

1. Catheters for bladder dysfunction should always be seen as a treatment of last resort.
2. Intermittent catheterization is the preferred option in urinary incontinence with overflow incontinence.
3. An indwelling catheter should be limited to those patietne with intractable urinary retention, continuous wetness (+/- skin breakdown) and those who have need of monitoring.

Discussion

Intermittent catheterization is the preferred management option for patients suffering from urinary retention with overflow incontinence. The combined voided and residual volume should not exceed 500 ml. and the frequency of catheterization should be monitored and adjusted if the residual volume changes. Long-term in-dwelling catheters are associated with several risks, such as chronic urinary bacteruria and bladder stones, and should only be used if intermittent catheterization is impractical.

Gresham et al. (1995) noted that "*use of indwelling catheters should be limited to patients with incontinence due to urinary retention that cannot be otherwise treated, severely impaired patients with skin breakdown, in whom frequent bed or clothing changes would be difficult or painful and in patients in whom incontinence interferes with monitoring of fluid and electrolyte balance.*" Brocklehurst et al. (1985) noted that 40% of patients regain continence during the first two weeks. The use of a catheter will inhibit that process. Again catheterization should be reserved for exceptional circumstances (Nazarko 2003).

Bjork et al. (1984), Sabanthan et al. (1985) and Wareen et al. (1982) observed the chronic use of indwelling catheters increases the risk of bacteria and urinary tract infection. Moreover, it has been shown that more than three-quarters of persons catheterized for three months or more will develop inflammatory bladder wall changes. While bacteremia can be identified by urine culture, treatment with antibiotics should be reserved for those patients with symptomatic urinary tract infections. Several investigators (Bennet and Diokno 1984, Maynard and Diokno 1984, Webb 1990) have noted that urinary retention can be safely managed with clean intermittent catheterization. Intermittent catheterization can be utilized when a stroke survivor is unable to pass urine and/or has a substantial amount of residual urine still in the bladder. To reduce the incidence of nosocomial UTIs, the use of silver alloy-coated urinary catheters has been recommended (Duncan et al. 2005). While the cost of these catheters is greater, they may be more cost-effective considering the reductions in cost associated with treating bacterial UTIs. The literature upon which this recommendation was based was not specific to stroke patients.

The use of indwelling catheters in stroke patients has not been well studied. There is consensus opinion that indwelling catheters should be limited to those patients with intractable urinary retention, skin breakdown, continuous wetness and the need for urinary monitoring. .

E10.17 Management of Bladder Retention

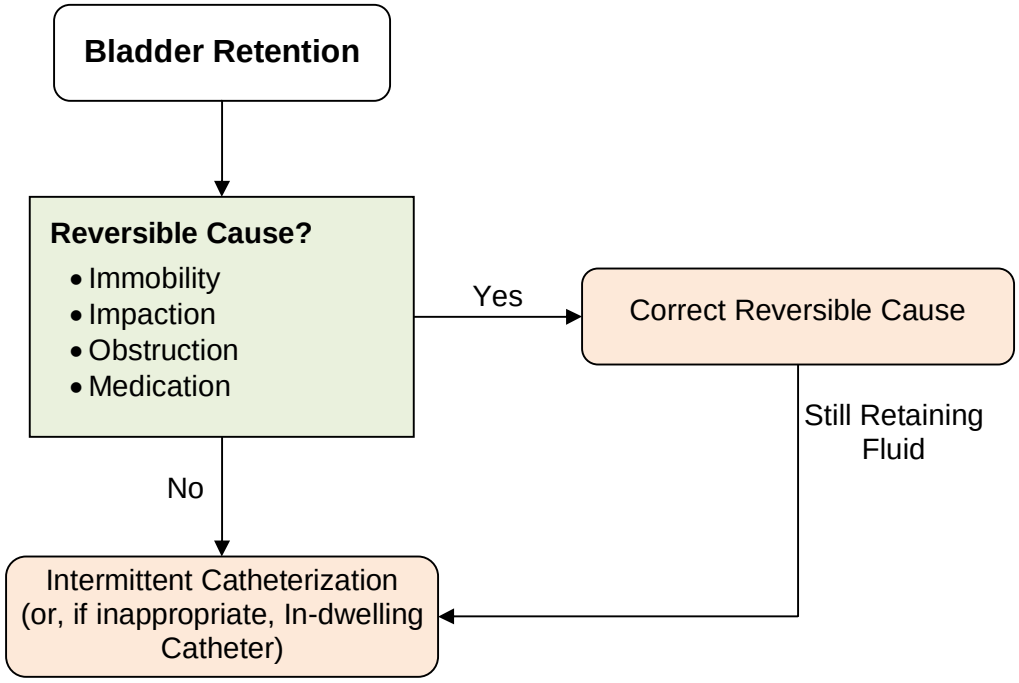
Q18. Describe an algorithm for the management of bladder retention?

Answer

1. Look for reversible causes of bladder retention and correct it.
2. If there is no reversible cause, need to perform urodynamic studies to determine whether it is a sphincter problem (not opening) or a detrusor problem (hypoactive bladder).
3. If sphincter problem consider alpha-blocker medications; if not successful consider botulinum toxin into the sphincter.
4. If detrusor problem consider cholinergic medication such as Bethanecol.
5. Once voiding begins monitor with post-void residuals.
6. If not successful catheterization is necessary; many stroke patient cannot do intermittent catheterizations which may require an indwelling catheter.

Discussion

Management of Post-Stroke Bladder Retention



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