Case 8

16 year old male struck by car while inline skating without a helmet-post ABI seizures

8.1 Post-Traumatic Seizures

8.1.1 Classification of post traumatic seizures and epilepsy

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8.3.1 Typical Presentation of RLA-V

Q17. How would an RLA-V typically present?

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Q18. How should an RLA-V be treated?

Case Study 8

Case Study 8

The following patient was admitted to rehabilitation following a two week stay in an acute care facility. The patient, a 16 year old male, had been hit by a car while inline skating home from school (the teenager was not wearing a helmet). The patient underwent a craniotomy shortly after admission to hospital.

At time of injury-while being treated at the scene-the patient underwent a seizure lasting 3 minutes. Tests indicate there was an electrolyte disturbance. Patient had been previously diagnosed with ADHD and had been receiving 5 mg of Ritalin (BID). To control for seizures the patient was originally administered phenytoin (25 mg BID). Just prior to the patient being admitted to rehab carbamazapine was then prescribed as his seizures continued.

8.1 Post-Traumatic Seizures

8.1.1 Classification of Post-Traumatic Seizures and Epilepsy

Q1. Define the following terms: Seizure, Post-traumatic Seizure and Epilepsy.

Answers

Seizure: Discrete clinical events that reflect a temporary physiologic dysfunction of the brain characterized by excessive and hypersynchronous discharge of cortical neurons.

Post-Traumatic Seizure: An initial or recurrent seizure episode not attributable to another obvious cause after penetrating or nonpenetrating TBI. The term *post-traumatic seizure* is preferred over *post-traumatic epilepsy* because the former encompasses both single and recurrent events.

Post-Traumatic Epilepsy: A disorder characterized by recurrent late seizure episodes not attributable to another obvious cause in patients following TBI. Although the term *post-traumatic epilepsy* commonly has designated single or multiple seizures including early seizures, the term should be reserved for *recurrent*, late PTS.

8.1.2 Identification of the "High-Risk" Patient

Q2. What factors are predictive of an ABI patient at high risk of seizures?

Answer

1. Patient characteristics: age, alcohol use, family history
2. Injury Characteristics: Bone/metal fragments, depressed skull fracture, focal contusions/injury, focal neurological deficits, dural penetration, intracranial hemorrhage, more severe injury.
3. Early post-traumatic seizures.

There are several patient and injury characteristics that increase the likelihood for the development of late PTS. Some important patient characteristics include: increasing age, premorbid alcohol abuse, and family history. In terms of injury characteristics, markers of increasing injury severity such as penetrating injuries and depressed skull fracture increase the risks of late PTS. A seizure occurring immediately after the injury substantially increases the risk of late PTS. As the severity of the brain injury increases, the period of time for which a survivor is at risk of developing PTS also increases.

Discussion

It is important to identify patients who are at high-risk of developing post-traumatic seizures (PTS) since these patients would benefit greatly from its prevention or from the impediment of its possible reoccurrence. Yablon and Dostrow (2001) have proposed that methods that assess the clinical characteristics of the patient, the injury, and information obtained from neuroimaging and electrophysiologic assessment techniques can be used to identify such high-risk patients. Moreover, Yablon and Dostrow (2001) have identified some key risk factors which can aid in the identification of these patients (see Table below).

Table 1 Studies of Risk Factors for Late Post-traumatic Seizures as reported by Yablon and Dostrow (2001)

Risk Factor	Reference
Patient Characteristics	
Age	(Annegers et al., 1980; Asikainen et al., 1999; Hahn et al., 1988; Kollevold, 1979)
Alcohol use	(Kollevold, 1978; Evans, 1962; Heikkinen et al., 1990)
Family history	(Caveness, 1963; Evans, 1962; Heikkinen et al., 1990; Hendrick & Harris, 1968)
Injury Characteristics	
Bone/metal fragments	(Ascroft, 1941; Salazar et al., 1985; Walker & Yablon, 1959)
Depresed skull fracture	(Jennett, 1975; Hahn et al., 1988; Phillips, 1954; Wiedemayer et al., 2002; Wiederholt et al., 1989)
Focal contusions/injury	(da Silva et al., 1992; De Santis et al., 1992; Eide & Tysnes, 1992; Glotzner et al., 1983; Heikkinen et al., 1990)
Focal neurologic deficits	(da Silva et al. 1992; Jennett1975; Salazar et al. 1985)
Lesion location	(da Silva et al., 1992; Evans, 1962; Grafman et al., 1992)
Dural penetration	(Caveness & LISS, 1961; Evans, 1962; Salazar et al., 1985)
Intracranial	(Hahn et al., 1988; Glotzner et al., 1983)

hemorrhage	
Injury severity	(Evans, 1962; Jennett, 1975; Salazar et al., 1985; Walker & Yablon, 1961)
Other	
Early post-traumatic seizures	(Heikkinen et al., 1990; Jennett, 1975; Salazar et al., 1985)

8.1.3 Natural History of Post-Traumatic Seizures

Q3. Describe the natural history of post-traumatic seizures.

Answers

1. 55-67% of PTS patients will experience seizure within the first 12 months and 75-80% by the end of the second year.
2. Patients with moderate to severe TBI or penetrating TBI remain at increased risk for more than 5 years post TBI.
3. Approximately half of those subjects with PTS will experience a seizure recurrence.

Discussion

Yablon and Dostrow (2001) have noted that one-half to two-thirds of patients who suffer PTS will experience seizure onset within the first 12 months, and 75-80% will have seizures by the end of the second year following the injury (Caveness et al., 1979; da Silva et al., 1992; da Silva et al., 1990; Pohlmann-Eden & Bruckmeir, 1997; Salazar et al., 1985; Walker & Yablon, 1959; Walker & Yablon, 1961). After 5 years, adults with mild TBI no longer have a significantly increased risk relative to the general population (Annegers et al., 1998), whereas patients with moderate or severe TBI or penetrating TBI remain at increased risk for more than 5 years post-injury (Annegers et al., 1998; da Silva et al., 1992; Pagni, 1990; Salazar et al., 1985).

Seizure recurrence is an important factor in the determination of disability, employment likelihood, quality of life, and increased health care costs (Baker et al., 1997; van Hout et al., 1997; Yablon & Dostrow, 2001). Some studies have reported that following early seizures post-TBI, only one-half of the patients experienced a recurrence (De Santis et al., 1979; Kollevold, 1979) while another quarter experienced a total of only 2-3 seizures (Kollevold, 1979).

8.1.4 Complications of Post-Traumatic Seizures

Q4. List some of the complications of post-traumatic seizures.

Answers

1. Deterioration in cognitive and behavioural functioning.
2. Deterioration in overall functional status.
3. Negative impact on neurological recovery.
4. Status epilepticus
5. Mortality

Discussion

Seizures following TBI may themselves be a source of significant complications and morbidity and it has been noted that the recurrence of seizures is an important cause of nonelective hospitalization in patients with severe TBI (Cifu et al., 1999). Potential complications include deterioration in cognitive and behavioral functioning and overall functional status, influence on neurological recovery, status epilepticus and death.

Cognitive and Behavioral Function

Post-traumatic seizure disorders may lead to cognitive and behavioural disorders (Yablon and Dostrow 2001). Cognitive problems may arise during the interictal state in the absence of active seizures (Aarts et al., 1984; Aldenkamp, 1997; Binnie & Marston, 1992). Patients with PTS experience persistent behavioral abnormalities and a higher incidence of psychiatric-related hospitalizations even compared with patients with penetrating TBI who do not experience PTS (Swanson et al., 1995).

Influence on Neurologic Recovery

Post-traumatic seizures can influence neurological recovery (Hernandez & Naritoku, 1997; Yablon & Dostrow, 2001). Yablon and Dostrow (2001) have noted that in rodent models, brief and infrequent PTS occurring early after brain damage do not appear to impact functional recovery. However, more severe and widespread seizures occurring within the first 6 days post brain injury result in permanent impairments of functional recovery; while the same seizures occurring after the 6 day mark result in no change in somatosensory recovery (Hernandez and Naritoku 1997).

Functional Status

Recurrent PTS may exert a negative impact on functional status following TBI, an adverse effect independent of the severity of the injury (Barlow et al., 2000; Schwab et al., 1993). In the case of penetrating traumatic brain injuries, post-traumatic seizures have been reported to be an important and independent factor which affects both employment status and cognitive performance (Schwab et al. 1993). However, in the case of nonpenetrating TBI, the impact of PTS on functional prognosis and cognition is less clear (Armstrong et al., 1990; Asikainen et al., 1999). Haltiner et al. (1997) found no significant differences at one year as a consequence of later PTS in terms of neuropsychological performance and psychosocial functioning when adjusting for injury severity. Asikainen et al. (1999) found that patients with PTS did have poorer outcomes on the Glasgow Outcome Scale, although there were no significant differences in employment outcome associated with the presence of PTS.

Status Epilepticus

Status epilepticus can be defined as either more than 30 minutes of continuous seizure activity (Yablon & Dostrow, 2001) or two or more sequential seizures without full recovery of consciousness between seizures (Yablon, 1993). Status epilepticus is regarded as the most serious of the complications of PTS and may actually lead to additional neurological damage. Fortunately, clinically apparent status epilepticus is an infrequent complication of PTS (Kollevold, 1979).

Mortality

Yablon and Dostrow (2001) have noted that among patients with PTS mortality remains consistently elevated (Corkin et al., 1984; Walker & Erculei, 1970; Walker & Blumer, 1989). However, the contribution of PTS to increased morality is unclear and some studies have suggested that deaths among penetrating TBI patients with PTS are due to

complications of the actual injury and are not related to seizures (Rish et al., 1983; Rish & Caveness, 1973).

Yablon and Dostrow (2001) have noted that the complications associated with single later post-traumatic seizures are no different than those found in any seizure and are generally associated with minimal risk. However, the risks of increased mortality and increased morbidity in the form of worsened cognitive and functional prognosis are associated with increasing frequency and severity of the seizure disorder.

8.1.5 Seizure Prevention or Prophylaxis

Q5. What evidence is there to support the prophylactic use of anticonvulsants after ABI?

Answers

1. Based on meta-analysis and the findings of this review there is Level 1 evidence that anticonvulsants given during the first 24 hours post-ABI reduce the occurrence of early seizures (within the first week post-injury).
2. There is Level 1 evidence that anticonvulsants given shortly after the onset of injury, do not reduce mortality or persistent vegetative state or the occurrence of late seizures (> one week post-injury).

Discussion

Initially, retrospective and nonrandomized clinical trials in humans showed favorable results for the efficacy of anti-epileptic drug prophylaxis (Caveness et al., 1979; Heikkinen et al., 1990; Kollevold, 1978; Murri et al., 1992; Murri et al., 1980; Price, 1980; Rish & Caveness, 1973; Servit & Musil, 1981; Wohns & Wyler, 1979; Young et al., 1979). However, prospective investigations of chronic prophylaxis for late PTS have shown less impressive results (Glotzner et al., 1983; Manaka, 1992; McQueen et al., 1983; Pechadre et al., 1991; Temkin et al., 1990; Temkin et al., 1991; Temkin et al., 1999; Temkin et al., 1990; Young et al., 1983b; Young et al., 1983a; Formisano et al., 2007).

Overall, most published randomized control trials have failed to find any favorable evidence for prophylaxis of late PTS (Young et al., 1983a; Glotzner et al., 1983; McQueen et al., 1983; Temkin et al., 1990). In some of these reports, the occurrence of PTS was actually higher in patients treated with anticonvulsant medications (Young et al., 1983b; McQueen et al., 1983; Temkin et al., 1990; Formisano et al., 2007).

In contrast to late PTS, Yablon and Dostrow (2001) have reported that anticonvulsant drug prophylaxis consistently reduces the incidence of early PTS (Glotzner et al. 1991; Temkin et al. 1990; Temkin et al. 1999; Young et al.1983a). Similar to most of the studies on late PTS, there is no evidence that anticonvulsant drug prohylaxis of early seizures reduces the occurrence of late PTS or has any effect on mortality or neurologic disability (Schierhout and Roberts 1998). As mentioned earlier, patients who suffer severe penetrating brain injuries experience higher risks of PTS. However, due to the small number of patients with penetrating TBI in most reports, it is not clear if anticonvulsant drug prophylaxis has any effects, either positive or negative, on the occurrence of PTS is this subgroup of brain injury patients.

Schierhout and Roberts (2001) conducted a Cochrane review of anti-epileptic drugs for preventing seizures following acute traumatic brain injury. This review of six trials including 1218 randomized patients. The authors noted that all of the included trials were restricted to patients considered to be at high risk for PTS. Most of the trials excluded patients who already had one seizure with the intervention started within 24 hours of admission in four trials; the remaining two trials initiated the intervention between 4 and 8 weeks post head injury. Four trials used phenytoin, one trial phenobarbital and one trial carbamazepine. The medication was provided between 1-2 years in all the trials – duration of follow-up was two years in most cases. Loss to follow-up ranged from 5-72%. Four trials reported early and late seizure; two trials reported late seizures only (Schierhout and Roberts 2001).

The number of patients with early seizures (within the first week after injury) was available from 4 trials representing 890 randomized subjects. The pooled relative risk for early seizure prevention was 0.34 (95% CI 0.21, 0.54) with the number needed to treat to keep one patient seizure-free in the acute phase was 10. However, there was no evidence that this resulted in a reduction in mortality or a reduction in death or persistent vegetative state. The occurrence of late seizure was not reduced by anti-epileptic prophylaxis at any time. There was insufficient data to examine non-fatal adverse effect, with the exception of a trend towards an increased risk of skin rashes.

The authors Schierhout and Roberts (2001) note that, *"There is no evidence that prophylactic anti-epileptics used at any time after head injury, reduce death and disability. There is evidence that prophylactic anti-epileptics reduce early seizures, but this is not supported by a reduction in late seizures. Insufficient evidence is available to establish the net benefit of treatment at any time after head injury."* (pg 1)

There appears to be very little research to evaluate the efficacy of anticonvulsants given to treat seizures after they have occurred. We identified only one such study in this review. Wroblewski and Joseph (1992) reported on a collection of ten case studies of TBI patients treated with intramuscular midazolam for acute seizure cessation after other benzodiazepine drugs had failed. The authors reported that in all patients, seizures ceased within minutes of midazolam administration. Midazolam also prevented the onset of prolonged seizures or status epilepticus. Slight to moderate sedation were the only reported side effects.

Q6. Does methylphenidate used for the treatment of cognitive and behavioural problems post ABI increase the risk of seizures?

Answer

1. There is Level 4 evidence that methylphenidate for the treatment of cognitive and behavioral problems can be safely used in brain injured patients at risk for posttraumatic seizures as it is not associated with an increase in seizure frequency.

Q7. What concerns are there for the use of anticonvulsants on rehabilitation? What evidence is there for this?

Answer

1. There is Level 1 evidence that both phenytoin and carbamazepine have negative effects on cognitive performance, particularly on tasks with motor and speed components, which theoretically may have a negative impact upon learning during rehabilitation.

8.2 Heterotopic Ossification (HO)

Case Study 8 (continued)

The physiotherapist who is working on mobility reports that the patient's right hip is warm, swollen and painful; she is concerned about decreasing range of motion of that hip.

Q8. What would be in the differential diagnosis?

Answer

1. Heterotopic ossification
2. Fractured bone
3. Rarely infected hip joint

8.2.1 Defining Heterotopic Ossification

Q9. What is heterotopic ossification?

Answer

Process whereby new bone forms within tissues where bone formation does not usually occur.

Discussion

Heterotopic ossification (HO) is a process where new bone forms within tissues where bone formation does not usually occur (Watanabe and Sant 2001). The incidence of HO in TBI patients has been reported as ranging from 11% to 77% but the disease only reaches clinical significance in 11-35% of this group (Garland et al., 1980; Sazbon et al., 1981; Rogers, 1988). Skeletal trauma, spasticity, immobilization, and prolonged coma greater than 2 weeks are considered to be at highest risk (Gennarelli, 1988; Roberts & Pankratz, 1979).

HO is often quite painful, at times limiting a patient's ability to participate in rehabilitation. In addition, it can cause restricted joint range of motion and thereby further add to their disability by restricting mobility or functional abilities. HO therefore can potentially impede progress of patients towards their desired rehabilitation goals.

8.2.2 Formation of Heterotopic Ossification Post-Head Injury

Q10. Describe the pathophysiology of heterotopic bone formation post ABI.

Answer

1. The pathophysiology of HO is not well understood. It is believed that there is a neurogenic factor contributing to HO, although this mechanism is not yet understood
2. Initial formation of osteoid.
3. Progression to full calcification within weeks.
4. Calcified osteoid remodels into well-organized trabecular bone over the ensuing months.
5. The bony lesion has been found to have a high metabolic rate, with a rate of bone formation more than three times greater than that of normal bone and an osteoclastic density of more than twice the number of osteoclasts found in normal bone.

Discussion

The pathophysiology of HO is not well understood. It is believed that there is a neurogenic factor contributing to HO, although this mechanism is not yet understood (Pape et al., 2004; Hurvitz et al., 1992; Pape et al., 2001). Pape et al. (2004) noted that mesenchymal stem cells can generate cartilage, bone, muscle, tendons, ligaments or fat (Williams et al., 1999) and are thought to play a pivotal role in the development of HO. Pape et al. (2004) have noted that circulating factors promoting heterotopic ossification may be present in head injured patients. In one animal study, the serum from patients with head injuries has been shown to promote mitogenesis and cell division in a rat osteoblast cell culture model (Bidner et al., 1990). Trentz et al. (2005) have noted that many studies have shown enhanced osteogenesis in patients sustaining traumatic brain injury (TBI). Accelerated fracture healing and heterotopic ossifications are well-known phenomena in these patients (Bidner et al., 1990; Keret et al., 1990). Heterotopic ossification also has been reported after a wide range of central nervous system lesions, such as spinal cord injury, traumatic brain injury, cerebrovascular accident, encephalomyelitis, anoxic encephalopathies, poliomyelitis, tabes dorsalis, brain neoplasm, multiple sclerosis, syringomyelocele, and arachnoiditis (Pape et al., 2004; Jensen et al., 1987).

HO forms through a typical process beginning with the formation of osteoid to full calcification within a matter of weeks (Pape et al., 2001). Over the next few months, the calcified osteoid remodels into well-organized trabecular bone at which point it is considered to have matured (Pape et al., 2001). Several months after the initial trauma, these patients develop paraarticular and intramuscular bone formation and experience restricted range of motion, pain and ankylosis (Garland et al., 1980; Banovac & Gonzalez, 1997). The bony lesion has been found to have a high metabolic rate, with a rate of bone formation more than three times greater than that of normal bone and an osteoclastic density of more than twice the number of osteoclasts found in normal bone (Puzas et al., 1987).

8.2.3 Clinical Presentation of Heterotopic Ossification

8.2.3.1 Location of Lesion

Q11. Which joints are most often involved in HO post TBI?

Answer

1. The most commonly affected joints are the hip, then shoulders, elbows and rarely the knee.

Discussion

The most commonly affected joint is the hip, then the shoulders, elbows and rarely the knee (Garland et al., 1980). Hip involvement results in 18 to 37% restriction of range of motion (Sarafis et al., 1999). Total ankylosis of the joint occurs in 5-16% of affected hips (Stover et al., 1991). Sarafis et al. (1999) have noted that the distribution of HO around the elbow occurs most commonly either anteriorly in the flexor muscles or posteriorly in the extensors. Of the joints affected by heterotopic ossification after head injury, ankylosis is most likely to occur in the elbow and it usually occurs posteriorly (Garland et al.,1980). Sarafis et al. (1999) have noted that the knee is a rare site of heterotopic ossification following head injury. The most common site in the knee is the inferomedial aspect of the distal femur.

Q12. How common is HO following TBI?

Answer

1. The incidence of HO in TBI is 10-20%.

Discussion

Sarafis et al. (1999) have noted that the incidence of heterotopic ossification in TBI patients is 10 to 20% (Garland, 1988; Garland, 1991a) with the hip being the most frequent site of ossification.

8.2.3.2 Clinical Features of Heterotopic Ossification

Q13. Describe the clinical picture of HO post ABI.

Answer

1. Clinical features of HO include a warm, swollen and painful joint often associated with decreased range of motion.

Discussion

Pape et al. (2004) have noted that clinical examination may reveal a swollen, warm, painful joint which is often associated with a decreased range of motion. Watanabe and Sant (2001) have reported that the formation of HO generally precedes symptom onset with the earliest sign often being decreased range of motion in the involved joint. Other findings then include swelling, warmth, erythema, pain, palpation of a periarticular mass and fever (Varghese, 1992). It is therefore difficult to differentiate heterotopic ossification

from infection because of the association with fever (Garland et al., 1980; Garland, 1991a; Citta-Pietrolungo et al., 1992). The clinical picture may be confused with deep venous thrombosis (DVT), a local infection, local trauma or fracture (Buschbacher, 1992; Jensen et al., 1987).

8.2.4 Diagnostic Tests for HO Post TBI

Q14. Describe those diagnostic tests which can be helpful in positively diagnosing HO post TBI?

Answers

1. Plain radiographs are negative and remain negative until ossification occurs 4-6 weeks post injury.
2. Serum level of alkaline phosphatase and the ESR may become elevated early on.
3. Triple phase technetium-99 bone scan with increase uptake during the 1st and 2nd phases remains the gold standard, becoming positive about the same time as the clinical features occur.

Discussion

Watanabe and Sant (2001) have noted that HO is generally initiated within 2-3 weeks of the onset of the injury; however, the onset has been reported 1 to 7 months following the TBI (Sazbon et al., 1981). During the initial presentation, plain radiographs may be negative and will usually remain normal until ossification begins 4-6 weeks post injury. Serum levels of alkaline phosphatase and the erythrocyte sedimentation rate may become elevated early on. The triple phase technicium-99 bone scan with increased uptake during the first and second phases remains the diagnostic gold standard, becoming positive at about the same time as clinical features occur.

8.2.5 Treatment of HO Post-Head Injury

Q15. What prophylactic treatments are available for treatment of HO post ABI?

Answers

1. Range of motion exercises - There is Level 4 evidence that forceful manipulation under general anesthesia increases range of motion in patients with heterotopic ossification following brain injury.
2. Nonsteroidal anti-inflammatory medications
3. Low-dose radiation
4. Warfarin
5. Etidronate disodium There is Level 2 evidence that etridonate (EHDP) reduces the development of heterotopic ossification in severe head injury patients.

Discussion

Watanabe and Sant (2001) have noted that prophylactic treatment options include range of motion exercises, nonsteroidal anti-inflammatory medications (NSAIDs), low-dose radiation, warfarin, and etridonate disodium (EHDP)

Physiotherapy and Range of Motion Exercises
Range of motion exercises has been somewhat controversial with some earlier reports suggesting that physical therapy might actually contribute to HO (Chantraine & Minaire, 1981; Crawford et al., 1986). More recently there has been a trend towards utilizing physical therapy with range of motion exercises and even manipulation under anaesthesia of the involved joints (Garland 1991a; Garland et al. 1982) to help prevent ankylosis. Pape et al. (2004) have noted that for HO, careful and judicious use of physiotherapy involving assisted range of motion exercises and gentle stretching has been shown to be of benefit (Ellerin et al., 1999). However, Pape et al. (2004) caution that care should be taken not to move the joint beyond its pain-free range of movement as this can exacerbate the condition (Evans, 1991).

Garland et al. (1982) conducted a review of TBI patients who underwent forceful manipulation under anaesthesia and reported that it was useful in maintaining and increasing range of motion in TBI patients. Garland et al. (1982) reported that in their study, gains in motion were made in 23/28 (82%) of the joints treated with manipulation under anesthesia and 18/28 (64%) joints maintained or gained further motion with rehabilitation. Garland et al. (1982) suggested that "forceful manipulation of joints with pre-existing heterotopic ossification has a role in maintaining a useful arc of joint motion and in prevention of bone ankylosis. Moreover, the results of Garland et al. [1982] suggest that manipulation does not appear to speed up or worsen the process of ossification.

Garland and Varpetian (2003) have noted that because these patients frequently suffer from spasticity, intolerance to pain and voluntary muscle guarding, anaesthesia is needed to help differentiate between spasticity and ankylosis and allow sufficient muscle relaxation to perform the forceful manipulation.

Nonsteroidal Anti-Inflammatory Medications
The evidence for NSAIDs being used in prophylactic treatment of HO comes mostly from the use of indomethacin or ibuprofen prophylaxis against HO in patients following total hip arthroplasty (THA) (Kjaersgaard-Anderson & Schmid, 1986; Ritter & Sieber, 1985). Although it has been noted that these medications offer a significant benefit in prophylaxis of THA, the correlation of these finding in traumatic brain injuries is not known (Watanabe and Sant 2001).

EHDP (Ethylhydroxybiphosphonate)
Watanabe and Sant (2001) have noted that biphosphonates, in particular etridonate (EHDP) in the prophylaxis and treatment of HO is controversial. EHDP works by preventing the aggregation, growth and mineralization of calcium hydroxyapatite crystals which are essential for bone formation. In a small study Spielman et al. (1983) showed EHDP reduced the development of HO in patients with TBI. Most of the research has been on spinal cord injured patients. Finerman and Stover (1981) and Stover et al. (1976) reported that EHDP resulted in a significant reduction in HO in SCI patients while Garland et al. (1983) noted that EHDP failed to prevent HO in the hips of SCI patients being treated for HO already present in other joints (Watanabe and Sant 2001). Pape et al. (2004) have noted that there has been a lack of conclusive evidence to show that etridonate arrests the development of HO (Garland, 1991a; Citta-Pietrolungo et al., 1992; Shehab et al., 2002; Pelissier et al., 2002). Etridonate may potentially delay fracture healing, as long-term use has been associated with osteomalacia. EHDP can

lead to nausea, diarrhea and joint pain, which can be improved by dividing the daily dose (Spielman et al., 1983).

One prospective controlled trial examined the effectiveness of EHDP treatment for the management of HO following brain injury (Spielman et al., 1983). Treatment began two to seven days post injury and lasted for a period of six months. The group of patients treated with EHDP showed a significantly lower incidence of HO when compared with the control group. Further research assessing the benefit of EHDP for the treatment of HO following brain injury is needed.

8.2.6 Surgical Excision of HO

Q16. Does surgical excision of HO post ABI improve clinical outcomes?

Answer

1. There is Level 4 evidence that surgical excision of heterotopic ossification improves clinical outcomes.

Discussion

Surgical excision of the heterotopic bone has been suggested as a possible option for those in whom heterotopic ossification has generated marked functional impairment or ulcers in the skin due to deformity (Watanabe and Sant 2001). According to expert opinion, surgical treatment should be considered only after 12-18 months to ensure that the bone tissue has matured, and to reduce the likelihood of recurrence (Sazbon et al., 1981; Garland, 1991b).

There are some indications that EHDP and NSAIDs may be useful in preventing HO recurrence following surgical excision (Watanabe & Sant, 2001), although further studies are still needed to corroborate this claim. Watanabe and Sant (Watanabe & Sant, 2001) have reported that recurrence of HO following surgical excision usually occurs within 3 months post-operatively.

8.3 Rancho Los Amigos – Level V and VI (RLA 5-6)

8.3.1 Typical Presentation of RLA-V

Q17. How would an RLA-V typically present?(Woo & Nesathurai, 2000)(p58)

Answer

A patient who has been diagnosed at RLA-V (Confused Inappropriate) and VI (Confused Apporpirate) may:

- Continue to demonstrate inappropriate responses to environmental stimuli.
- Demonstrate reduced information processing capacity relative to the amount, rate and duration, and complexity of information provided.
- Not be able to perform elementary tasks such as dressing, bathing and feeding.
- Be vulnerable to outside variables and lack the internal mechanism to modulate responses.
- Sometimes react to the environment or situations is an exaggerated manner.

8.3.2 Treatment of RLA-V

Q18. How should an RLA-V be treated?

Answer (Woo & Nesathurai, 2000)

Treatment goals for an RLA-level V (Confused Inappropriate) or VI (Confused Appropriate) include:

1. Structure- oriented approach is most effective.
2. At this stage, discipline-specific goals can be established.
3. Tasks must structured by manipulating the stimulus parameters (i.e., amount, rate, complexity and duration of input).
4. Functional tasks must be simplified into sequential steps.
5. As cognitive capacity increases, structure can be gradually reduced.

Treatment strategies for patients include:

1. Environment should remain constant in terms of locations of objects.
2. Memory aids should be provided within the patients' vision.
3. Environmental distractions should be minimized.
4. The same staff should treat the patient where possible.
5. All personnel should provide orienting information at the beginning or end of each session.

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