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Late Posttraumatic Epilepsy in Patients with Tension Pneumocephalus Complicating Head Injury: Report of Two Cases

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Abstract: Late posttraumatic epilepsy complicating traumatic pneumocephalus is rare and the pathogenetic association between these conditions is not clear. We report its occurrence in two adult men who developed intracranial hypertension, recurrent CSF rhinorrhoea and incontinence of urine and or faeces due to intra-cerebral and intra-ventricular pneumocephalus after apparent recovery from initial motorbike head injury. Cranial computed tomography revealed left frontal lobe pneumocephalus in one patient (Case 1), while skull radiography revealed pneumocephalus in the region of the left frontal lobe and lateral and third ventricles in the other (Case 2). Case 2 recovered fully while on initial conservative management but Case 1 required a frontal craniotomy for decompression and repair of cranio-dural fistula. Both patients developed generalised tonic-clonic epilepsy after eight months of follow-up. The scalp EEG recordings were normal and repeat radiological investigations revealed complete resolution of pneumocephalus. Both patients have been seizure free for 9 and 12 months respectively on phenytoin sodium. The possible association link between poattraumatic pneumocephalus and epilepsy is discussed.

Key words: Tension pneumocephalus, head injury, epilepsy, hypertension CSF

INTRODUCTION

Pneumocephalus, characterized by the presence of air or gas in the intracranial cavity is frequently caused by trauma and surgery (Jayaram *et al*., 2004). It occurs in 0.5 to 1.0% of head injured patients (Ersahin *et al*., 1999). Tension pneumocephalus occurs more rarely when air is trapped under pressure within the cranial cavity producing neurological deterioration and complications and sometimes death especially when diagnosis and treatment are delayed. Early Generalized Tonic-Clonic (GTC) seizures have been associated with tension pneumocephalus developing after craniotomy in supine position (Satapathy and Dash, 2000) ventriculoscopy (Saxena *et al*., 1999) cervical laminectomy (Schwarz and Tritthart, 1987) excision of fibrous dysplasia (Kanda *et al*., 2002) and epidural anaesthesia using the loss of resistance to air technique (Oliver and White, 2002; Van *et al*., 2002; Rodrigo *et al*., 1997). We are unaware of any report associating either early or late onset seizure with tension pneumocephalus following head injury. Nevertheless, late posttraumatic seizure is a recognized complication of head injury; the estimated incidence being 10-13% within 2 years after "significant" head trauma. It is therefore obvious that both head injury and pneumocephalus constitute risk factors either singly or in combination for seizure development. The patho-physiology of epilepsy following head injury is gradually being unravelled but the mechanism by which tension pneumocephalus produces epilepsy remains obscure. We present 2 illustrative cases because of the rarity of this association and in order to suggest the role of pneumocephalus in the development of late posttraumatic epilepsy. Further research is expected to yield better dividends in this respect.

CASE REPORTS

Case 1: A previously non-epileptic 57-year-old man was admitted after a cyclist head injury with bilateral cerebrospinal fluid rhinorrhoea, facial and scalp lacerations, peri-orbital ecchymoses, sub-conjunctiva hemorrhage and right facial nerve palsy. His post-resuscitation GCS was 10 and there were no focal deficits or lateralizing pyramidal signs. There was no familiar history of epilepsy. CSF rhinorrhoea stopped and he regained consciousness on bed rest, head-up nursing and intravenous mannitol, fluids and antibiotics. Four weeks

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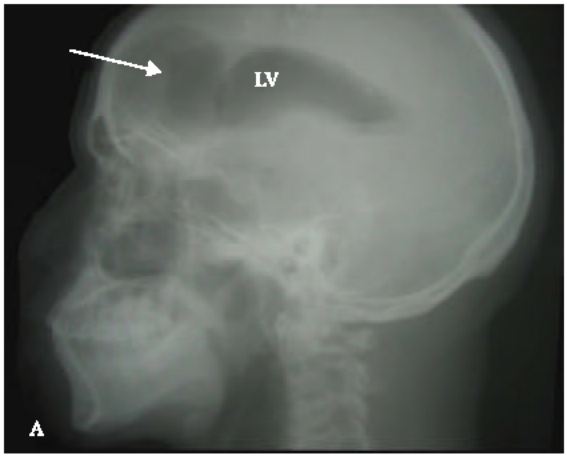

Fig. 1: Lateral skull radiograph showing pneumocephalus in the region of the frontal lobe and the lateral ventricles

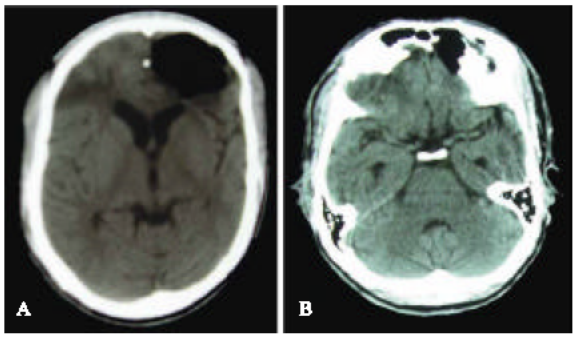

Fig. 2: Cranial computed tomography showing rounded air filled cavity (pneumocephalus) in the frontal lobe of the left cerebral hemisphere (A) and a fractured posterior wall of the left frontal sinus (B)

after discharge he represented with recurrent rhinorrhoea, headache, impaired memory, urinary incontinence and meningeal signs. His Glasgow coma scale score was 9. Head CT cysternography revealed a round air image in the left frontal lobe and a fracture of the posterior wall of the left frontal sinus (Fig. 1). He was commenced on culture sensitive ceftriaxone combined with metronidazole for meningitis and subjected to a craniotomy and extra-dural fascia lata repair of CSF fistula. He remained asymptomatic till after eight months of follow-up when he represented with multiple episodes of generalised tonic-clonic seizure. Scalp EEG recording was normal and repeat cranial CT showed complete resolution of pneumocephalus without evidence of a new intracranial lesion. He has been seizure free for 9 months on oral phenytoin sodium.

Case 2: A 49-year-old man without family or past history of epilepsy was successfully managed conservatively for moderate head injury (GCS = 10) and left CSF rhinorrhoea resulting from motorbike crash. His skull and sinus radiographs revealed an anterior cranial fossa basal skull fracture over the right orbital roof without intracranial air. Three weeks after discharge he represented with sudden onset headache, urinary and faecal incontinence, irrational speech and behavior and later coma. GCS at presentation was 8. Cranial CT was not done because of financial constraints, but skull radiographs showed air within grossly dilated lateral and third ventricles extending into the skull base and the region of the left frontal lobe (Fig. 2). His response to initial conservation with intravenous prophylactic antibiotics, mannitol and fluid therapy as well as intubation and hyperventilation with head-up (45°) nursing was prompt and sustained. He regained consciousness and other symptoms subsided while skull x-rays showed complete resolution of the pneumocephalus and pneumo-ventricle. He remained asymptomatic for eight and half months before onset of late posttraumatic generalized tonic-clonic seizures. The EEG examination was normal and a repeat skull radiograph revealed no recurrence of pneumocephalus. He has been seizure free for a year on oral phenytoin sodium.

DISCUSSION

It is difficult to conclude or confirm whether the late epilepsy observed in the two patients presented is a direct product of the primary head injury, or the complicating tension pneumocephalus or both. This is because both Tension Pneumocephalus (TP) and head injury have been independently associated with seizure occurrence, though the association of the former with epilepsy has been reported most often after surgical operations (Satapathy and Dash, 2000; Saxena *et al*., 1999; Schwarz and Tritthart, 1987; Kanda *et al*., 2002) or epidural anaesthetisia (Oliver and White, 2002; Van *et al*., 2002; Rodrigo *et al*., 1997) and none has been reported after trauma to the cranium. Though van den Berg *et al*. (2002) described a postpartum micro-vascular angiopathy on the brain MRI of a parturient who developed grand mal seizures secondary to pneumocephalus after the use of loss of resistance to air technique for epidural anaesthesia, generally the reason for epileptogenesis in

patients with postoperative or post epidural puncture pneumocephalus remains largely obscure (Oliver and White, 2002).

In head injured patients, Jennett (1975) identified common risk factors for delayed posttraumatic seizure which include early PTS, depressed skull fracture, intracranial haematoma, prolonged unconsciousness, low Glasgow coma scale score and prolonged posttraumatic amnesia, to which De Santis *et al.* (1992) added "documented cortical-subcortical brain lesions" which he opined represent the main risk factor for delayed posttraumatic epilepsy. In this context, a tension intra-cerebral and intra-ventricular pneumocephalus could be regarded or viewed as a cortical and subcortical lesion serving as an extra risk factor just as intracranial haematoma. The epileptogenesis of intracranial haematoma is due to the hemin and iron, breakdown products of hemoglobin. These products have been shown to have physiological effects on synaptic transmission that may lead to epileptogenesis (Yip and Sastry, 2002). The effects of iron are thought to be related to the formation of free radicals that cause direct injury to neuronal membranes and cell death and also the release of the excitatory neurotransmitter glutamate.

The mechanism underlining the genesis of an air induced seizure may be similar or completely different. Experience with animal models used in examining the consequences of head injury on epileptic activity lends support for the possibility of the effect of rapid air influx into the cerebral parenchyma or the ventricle. A popular model that is widely used is the fluid percussion injury model, where saline is rapidly injected into a closed cranial cavity, resulting in increased intracranial pressure and a brief displacement of neural tissue. This model has been used to demonstrate that damage to the hippocampus, an area of the brain often damaged in temporal lobe epilepsy, can result even after mild head injury (Hicks *et al.*, 1993). A rapid-air-influx through a cranio-dural fistula into the cerebral parenchyma which could occur during sneezing or coughing can produce similar damaging effect not only on the surrounding cerebral parenchyma but also on the hippocampus. It could also cause tearing of white matter that heals later with a gliosis that could subsequently act as an epileptogenic focus. It could also produce microvascular disruption with petechial hemorrhages or micro-bleeds that form micro-haematomas producing epileptogenesis through similar mechanisms described for intracranial haematoma above.

Although the inheritance of apolipoprotein E epsilon (ε) 4 allele has been associated with an increased risk of late posttraumatic seizures, this cannot be confirmed in our patients except their gene types at the APOE locus was determined by restriction fragment length polymorphism analysis (Diaz *et al.*, 2003).

CONCLUSION

It appears that both pneumocephalus and head injury have unique and probably independent roles to play in the pathophysiology of late epilepsy in the patients described though the definite roles are not certain. Systematic prospective human and experimental studies may be required to unravel the uncertainties.

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