Clinical Reports

Guillain-Barré syndrome following acute head trauma

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Summary: A case of classical Guillain-Barré syndrome following acute head trauma is described. The association of Guillain-Barré syndrome with head injury per se is not well recognized, and a possible immunological explanation is proposed.

Introduction

The Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy which follows a putative precipitating event in approximately two-thirds of cases. GBS has been described most frequently after non-specific viral infections, but it may also follow immunization, specific viral infections and various types of trauma, including intracranial and general surgical procedures, orthopaedic operations, and spinal anaesthesia. GBS following non-surgical head trauma per se, however, is not a well recognized event. We describe here a case of severe GBS developing after acute head injury.

Case report

A 61 year old man sustained a head injury after falling off a ladder. He was admitted to the Institute of Neurological Sciences that day, where he was noted to be drowsy and confused but not in coma. There was no focal limb weakness, and all limb reflexes were brisk. Plain skull X-rays showed a depressed right parietal fracture. Computed tomographic (CT) brain scan revealed extensive right fronto-parietal contusions, a small left posterior frontal contusion and a small right subdural haematoma. He was managed conservatively, improved rapidly and was discharged after 6 days, able to walk and talk normally. Nine days later he developed cramps in both legs and rapidly progressive weakness of all 4 limbs, as well as inability to swallow or breathe. There had been no recent viral illness. He was admitted to a local district hospital where he suffered a cardiorespiratory arrest from which he was resuscitated, and was then readmitted to the Institute of Neurological Sciences.

On examination he was apyrexial, with a tachycardia of 110/minute, blood pressure of 160/90 mm Hg and a respiratory rate off the ventilator of 55/minute. Air entry to the chest was generally reduced. He was fully alert and oriented. The fundi were normal. There was a left ptosis, a right lateral rectus palsy and bilateral lower motor neurone facial palsy. There was severe flaccid symmetrical weakness of all 4 limbs, more severe proximally. All tendon reflexes were absent and the plantar responses were flexor. There was distal impairment of sensation in all 4 limbs.

Investigations revealed normal routine haematology and biochemistry initially, but during his illness he developed transient hyponatraemia (plasma sodium 121 mmol/l) with the characteristics of inappropriate secretion of antidiuretic hormone (plasma osmolality 281 mmol/l with a urine osmolality of 951 mmol/l). Urinary porphyrins were not detected. A repeat CT scan showed no change from that performed at the time of his previous head injury. Electrodiagnostic studies revealed a severe generalized predominantly motor demyelinating peripheral neuropathy entirely consistent with the diagnosis of GBS. Cerebrospinal fluid examination was not performed because of the presence of the subdural haematoma, and the unequivocal establishment of the diagnosis by clinical and electrophysiological means.

During the first 4 days of his admission his respiratory function worsened, due at least in part to a right basal pneumonia, and a tracheostomy was performed. Both his limb weakness and bulbar function deteriorated further and over the next week he had five plasma exchanges. His condition then remained static for 6 days and he began to improve rapidly thereafter. Respiratory and bulbar function as

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well as his cranial nerve palsies recovered completely within a few days. Improvement in limb power occurred more slowly, but continued to show recovery when he was transferred back to the referring hospital 5 weeks after the development of his neuropathy.

Discussion

This patient showed the typical features of GBS with the rapid development of symmetrical flaccid limb paralysis, bilateral lower motor neurone facial weakness, respiratory and bulbar involvement, areflexia and distal sensory loss. The diagnosis was confirmed by the electrophysiological findings of a severe demyelinating neuropathy. The only clear precipitating event which could be identified was the severe head injury, which we infer to be the inducing stimulus for the development of GBS. Although the occurrence of GBS following trauma is well recognized, we do not believe that acute head injury by itself as a precipitating cause of GBS has previously been described. Postsurgical GBS generally follows 2 to 3 weeks after operation, which is consistent with the interval of 15 days between the head trauma and the development of symptoms observed in this case. The transient development of hypotraemia due to inappropriate secretion of ADH is well recognized in association with GBS, but it may also have been related to his chest infection, head injury or endotracheal intubation.

Plasma exchange has been shown to be effective in the treatment of GBS. The theoretical basis of the treatment consists of the finding of demyelinating factors in the plasma of GBS patients and, more recently, the finding of circulating antibodies to peripheral nerve myelin in such patients, the level of which decreases as clinical improvement occurs. The best characterized protein specific to nervous tissue is myelin basic protein. It is known to be immunogenic, inducing the demyelinating disease experimental allergic encephalomyelitis in a variety of animal species. The level of myelin basic protein in serum and cerebrospinal fluid of patients who have suffered head injury or undergone neurosurgery is raised. It is possible that, in the above case, myelin basic protein, or some other immunogenic component of nervous tissue released into the circulation following injury induced the production of anti-myelin antibodies, causing a demyelinating neuropathy. Invoking this mechanism raises the question of why GBS does not follow head injury or neurosurgical procedures more often. It is possible that because the potentially immunogenic factor originates in the central nervous system it is usually only weakly immunopathogenic for peripheral nervous tissue.

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References