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ONLINE CASE REPORT

Cerebellar Mutism Following Closed Head Injury in a Child

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الخرس الدماغي عقب إصابة الرأس المغلقة عند طفل

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ABSTRACT: Cerebellar mutism is a rare occurrence following paediatric trauma. Although it is quite common after posterior *fossa* surgery in children, this phenomenon has rarely been reported following other insults, such as trauma, and its pathophysiology remains poorly understood. We report a seven-year-old child who presented to the casualty department of Sultan Qaboos University Hospital in Muscat, Oman, in May 2013 with a traumatic right cerebellar contusion. The child presented with clinical features of cerebellar mutism but underwent a rapid and spontaneous recovery. The possible mechanism of this occurrence is discussed.

Keywords: Head Injury; Mutism; Paediatrics; Complications; Case Report; Oman.

الملخص: يعد الخرس الدماغي نادر الحدوث عقب الرضح عند الأطفال. وعلى الرغم من أنه شائع بعد عمليات الجراحة في الحفرة الخلفية عند الأطفال، إلا أنه لم يسجل إلا نادرا عقب مختلف أنواع الأذية مثل الرضح. وما زالت فيزيولوجيته المرضية غير مفهومة تماما. وهنا نسجل حالة طفل في السابعة من عمره أحضر لقسم الحوادث في مستشفى جامعة السلطان قابوس في مسقط بعمان في مايو 2013م مصابا برضة رضحية في يمين الدماغ. وأظهر الطفل علامات سريرية للخرس الدماغي، إلا أنه سرعان ما شفا بسرعة وبتلقائية. ونناقش هنا آلية حدوث هذه الحالة.

مفتاح الكلمات: إصابات الرأس؛ الخرس؛ الأطفال؛ مضاعفات؛ سجل حالة؛ عمان.

Cerebellar mutism (CM) was first described by Rekate *et al.* in 1985 following posterior *fossa* surgery in children;[1] since then, it has increasingly been reported, mainly occurring as a postoperative complication. It has also been reported in both children and adults following several other cerebellar insults, including vascular events, infections and trauma.[2] However, post-traumatic CM has only rarely been reported.[3–6] Its unique clinical features, heterogeneous anatomical localisation and unclear pathophysiology make it a curious clinical entity.

Case Report

A seven-year-old right-handed boy was brought to the casualty department of Sultan Qaboos University Hospital in Muscat, Oman, in May 2013. He had fallen off a cupboard which he had been climbing and the cupboard had subsequently fallen on top of him. On admission, the emergency room physicians recorded his Glasgow Coma Scale (GCS) as 8/15; however, the analysis of different components of the score was not documented. An examination showed bilaterally equal and symmetrical reactive pupils, without evidence of any unilateral motor deficit. The patient subsequently vomited and had one seizure episode. He was quickly intubated and ventilated.

A plain computed tomography (CT) scan of the brain showed a comminuted fracture of the right *occiput* with a small underlying cerebellar contusion and minimal blood in the fourth ventricle [Figures 1A & B]. A repeat CT scan the next day showed no increase in the cerebellar haematoma or any new lesions. He was extubated after 48 hours of elective ventilation. A neurological examination soon after extubation revealed a GCS of 10/15, no vocalisation and the presence of mild right lower motor neuron facial nerve palsy and mild weakness in the upper extremities. Magnetic resonance imaging (MRI) of the brain revealed right cerebellar oedema and a resolving small right cerebellar contusion without supratentorial lesions [Figures 2A & B]. By the sixth day, he was obeying commands fully and his facial weakness and upper limb weakness had resolved. He started crying by the 10th day but still could not speak. He could walk with minimal *ataxia* at 13 days after admission. He was discharged two weeks after admission once he had spoken a few words. No emotional lability was noted at any time.

The patient did not return to the hospital for his

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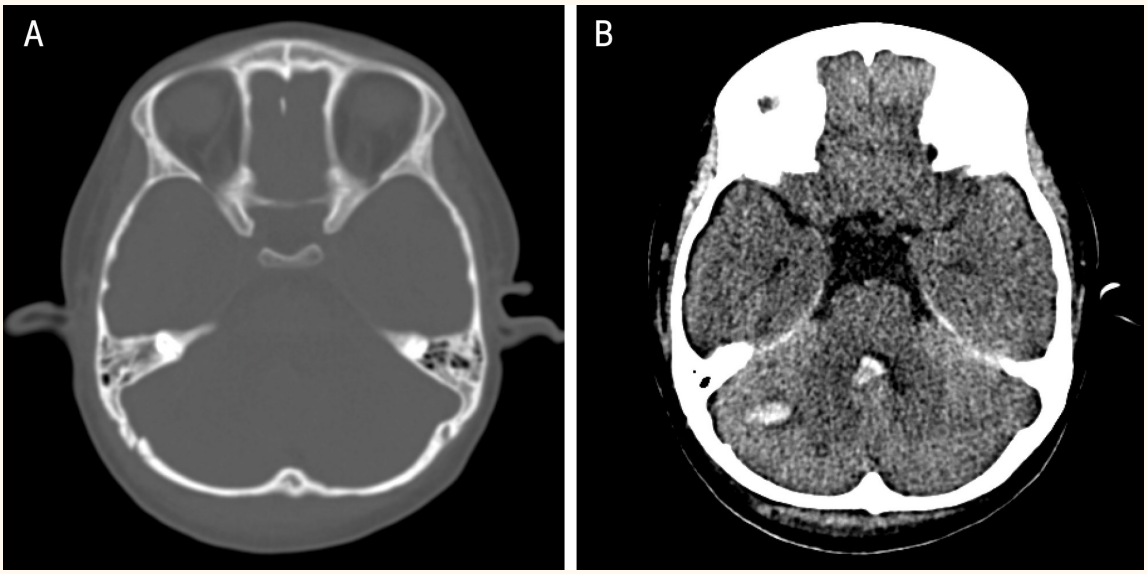

Figure 1A & B: Computed tomography scans of the brain showing (**A**) the right occipital fracture and (**B**) the right cerebellar contusion with intra-ventricular haemorrhage.

one-month follow-up. However, when contacted by telephone, his family reported that he had regained normal speech. At his first review eight months after discharge, the child was back at school and taking part in normal activities. He had no speech, cranial nerve, motor or cerebellar deficits. A detailed neuropsychological assessment was suggested but not performed.

Discussion

Mutism is defined as the inability to speak in an otherwise cognitively alert patient.[4] This can occur due to lesions in several locations of the brain, including Broca's area and the supplementary motor cortex, thalamus and mesencephalic reticular formation regions, as well as due to bilateral hemispheric lesions.[4] Mutism is more commonly seen following posterior *fossa* surgery, predominantly in children, with a reported incidence varying from 2–40%.[7] It has also been reported uncommonly in other cerebellar pathologies including trauma, vascular events and infection.[3–6,8,9] In a recent review, several terms were found in the literature to describe associated features of mutism: CM, transient cerebellar mutism, mutism and subsequent dysarthria, CM syndrome and posterior *fossa* syndrome.[2] These encompass a spectrum of neurological deficits from mutism alone to *ataxia*, hypotonia, cranial nerve palsies, haemiparesis and emotional lability.[2]

In its typical form, CM has a delayed onset (1–6 days) and limited duration (between one day and four months) followed by a variable period of recovery.[2] In this particular patient, it was not possible to identify the precise time of onset due to the low GCS score at presentation and the subsequent 48-hour ventilation period. Nevertheless, other features of the case—the failure to speak for 10 days, the subsequent gradual return of verbalisation and the rapid recovery within one month—were typical of the usual course of CM.

The anatomical substrate of CM is reportedly variable. To date, damage to the midline cerebellar structures have been described in more than 90% of paediatric and 70% of adult CM cases.[8] The most commonly implicated regions are the *vermis*, dentate nuclei and the cerebellar peduncles—particularly the superior and middle cerebellar peduncles.[2] These findings are mainly based on postoperative imaging of patients with midline posterior *fossa* tumours. In the current case, the *vermis* and the peduncles appeared intact on the MRI scan. The right cerebellar hemisphere was predominantly involved, as indicated by the diffuse oedema of the entire right hemisphere and the small contusion close to the cerebellopontine angle. Neurocognitive studies have suggested that the right cerebellar hemisphere has a dominant role in language tasks.[10,11] However, even left cerebellar contusion has been known to produce CM.[5,6] It has therefore been suggested that any bilateral disruption of the dentato-thalamo-cortical pathway, regardless of the specific location, can result in CM.[2]

In addition, the pathophysiological mechanisms responsible for CM are also unclear, with vasospasm, oedema and the resultant cerebello-cerebral *diaschisis* being variously attributed.[2] Oedema of the right cerebellar hemisphere was the prominent feature in the MRI scan of the presented patient. The delayed

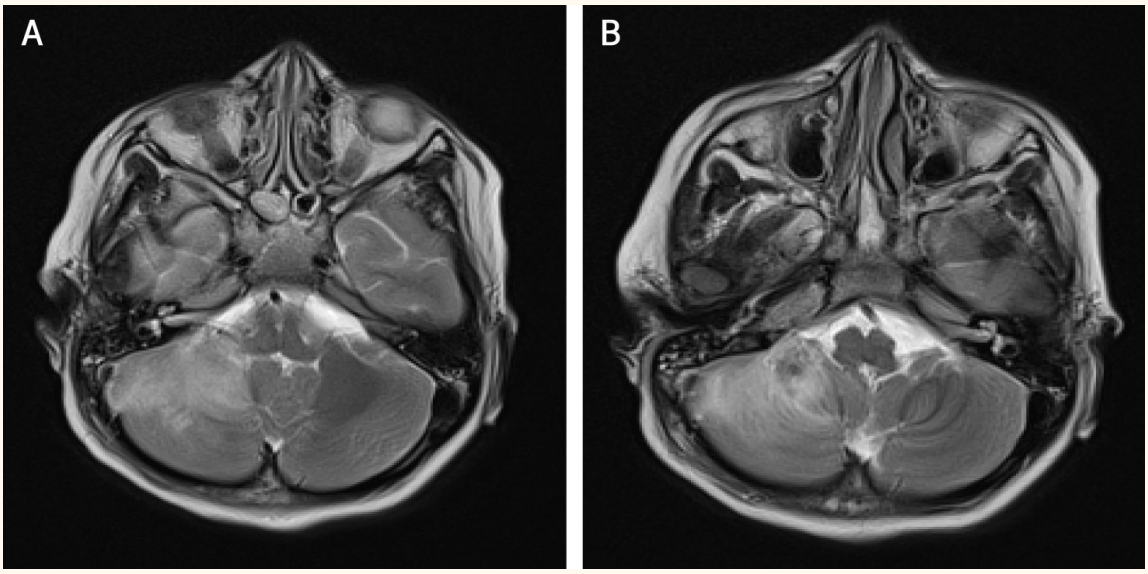

Figure 2A & B: Magnetic resonance imaging scans of the brain showing **(A)** the right hemispheric oedema with minimal mass effect and **(B)** the resolving right cerebellar contusion.

onset of symptoms correlates approximately with the onset of oedema; however, the duration of symptoms outlasts the oedema, especially in severe cases.[2] With less severe injuries, as in this case, the oedema and the consequent mass effect and midline shift could cause a transient disturbance of the bilateral dentato-thalamo-cortical pathway.

Most reports of cases with postoperative CM mention long-term residual deficits including dysarthria, *ataxia* and behavioural changes.[7] In this case, the child most probably had a full recovery due to less severe cerebellar involvement. However, minor residual abnormalities, including language, memory and learning disabilities, may not have been discovered in the absence of a detailed neuropsychological evaluation.

Only five cases of post-traumatic mutism have been reported in the literature, although this entity is probably under-reported.[2] Of these cases, two were due to right cerebellar contusions, two to left cerebellar contusions and one was due to a right posterior *fossa* acute subdural haematoma.[3–6] This further highlights the heterogeneous anatomical localisation of CM. Prognosis for these patients, as in the current case, was excellent; however, residual dysarthria was mentioned in one patient after six months.[3]

Conclusion

Although CM is fairly common following posterior *fossa* surgery in children, its mechanism remains poorly understood. Its occurrence in other pathologies, especially trauma, may help in understanding its pathophysiology.

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