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Reversible Cerebral Vasoconstrictive Syndrome preceded by Minor Head Trauma

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Introduction

• Reversible cerebral vasoconstriction syndrome is a heterogeneous and under-recognised neurovascular disorder. Our knowledge with regards to specific syndrome triggers and optimal management is limited. The delay in diagnosis can be deleterious to the patient due to intracerebral sequelae causing temporary or permanent morbidity. Prompt identification of this syndrome is vital to reverse neurological deficits while appropriately managing and supporting patient recovery.
Case Report

A 52-year-old left handed woman with a background history of Type 2 Diabetes, hypothyroidism and hypertension was admitted after a book fell on her head. She developed progressive left-sided headaches and associated nausea, with no focal abnormality identified on CT brain during initial assessment. Three days after discharge, she experienced episodic focal seizures and an MRI and MRA showed localized subacute right frontal subarachnoid haemorrhage and normal intracranial vasculature. Over the course of the subsequent two weeks, she deteriorated becoming increasingly encephalopathic. Twenty-one days after the original minor trauma, she developed left sided hemiplegia, followed by a drop in her GCS necessitating intubation.
The patient was then transferred to the neurosurgical centre. CT brain showed multi-focal subacute frontal ischaemic infarcts (figures 1&2).

Figure 1. Axial Non-contrast CT Brain completed on transfer to the Neurosurgery Centre 21 days post original trauma identifying multi-focal subacute frontal ischaemic infarcts

Figure 2. Axial Non-contrast CT Brain completed on transfer to the Neurosurgery Centre 21 days post original trauma identifying multi-focal subacute frontal ischaemic infarcts
CT angiography (CTA) revealed severe focal narrowing in the A2 and A3 regions of the right Anterior Cerebral Artery (ACA), bilateral narrow but patent Middle Cerebral Arteries (MCAs) (figure 3&4).

Figure 3. Axial CT angiogram intracranial completed on transfer to the Neurosurgery Centre 21 days post original trauma identifying bilateral narrow calibre but patent Middle Cerebral Arteries (MCAs), with further focal narrowing along the right ACA.

Figure 4. Axial CT angiogram intracranial completed on transfer to the Neurosurgery Centre 21 days post original trauma identifying severe focal narrowing in the A2 and A3 regions of the right Anterior Cerebral Artery (ACA).
Digital Subtraction Angiography (DSA) showed diffuse vasoconstriction, greatest in the right anterior circulation (figure 5). Lumbar puncture and vasculitis screen were negative.

Figure 5. Initial Digital Subtraction Angiography: Right ICA injection lateral view showing diffuse vasoconstriction, greatest in the right anterior circulation 22 days post original trauma.
• The patient was diagnosed with RCVS with secondary ischaemic infarcts. No drug or over-the-counter medication was identified as a preceding trigger. The patient was subsequently admitted to the neurosurgical intensive care unit for close neurological and invasive blood pressure monitoring. The patient’s clinical status was highly dependent on blood pressure control, with mild hypotension resulting in deteriorating level of consciousness and worsening neurological deficit. The patient was managed with hypertensive therapy for vasospasm, nimodipine, anti-platelet agents and anti-epileptic medication, while avoiding all sympathomimetic agents. Repeat MR evaluation showed evolving left posterior cerebral artery (PCA), right ACA and posterior cerebellar artery territory infarcts, with some haemorrhagic component (figure 6).
• 27 days post original injury, the patient began to improve clinically. She was extubated, her headaches resolved and her left sided weakness gradually improved. Initial reduction of nimodipine caused an increase in headaches. Mean Arterial Pressures remained stable between 85 and 100mmHg. A repeat DSA showed persistent bilateral multifocal arterial irregularity with areas of narrowing and dilatation affecting the anterior and posterior circulation.

• DSA performed 3 months after the patient’s initial presentation identified near complete resolution of the previously beaded appearance of intracranial vessels (figure 7). The radiographic reversal of this patient’s previous vasoconstriction confirmed the diagnosis of RCVS.
Discussion

• Reversible Cerebral Vasoconstriction Syndrome (RCVS) is reversible multifocal narrowing of the cerebral arteries. Due to its similar clinical manifestation, this condition has been misinterpreted as primary CNS angiitis or vasospasm secondary to aneurysmal subarachnoid haemorrhage.
• Our patient presented symptomatically following a minor head trauma. No other potential causative mechanisms were identified. We postulate that the minor head trauma triggered the development of RCVS, with the earliest radiographic feature being cortical subarachnoid haemorrhage.
• Currently the literature proposes multiple potential provoking factors which could influence the activation of RCVS. Abnormal cerebrovascular tone has been suggested as a possible cause of this angiographic phenomenon however the exact pathogenesis of this condition is yet to be fully determined.
• The use of sympathomimetics and recent pregnancy or the postpartum state appear to be triggers. Disruption in hormonal regulation has also been linked to the phenotypic manifestation of this disease. Furthermore eucalyptus, indomethacin and the oral contraceptive pill have all been linked to sporadic onset RCVS.
• All documented SAH in the literature to date preceding a diagnosis of RCVS have been spontaneous, atraumatic and without aneurysms identified on CT angiography.
Conclusion

• This is the first documented case - to the authors’ knowledge - where a diagnosis of RCVS has been preceded by a minor head injury. This trauma is the only potential preceding incident that could have provoked this syndrome though a pure coincidence with a spontaneous onset cannot be ruled out. Our patient was female and within the reasonable age demographic for onset.

• This ambiguous pathology should be considered as a differential diagnosis in patients show atypical symptoms and fail to respond to standard management.