A rare case of hemorrhagic mineralising angiopathy after trivial head trauma

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Abstract

Infarction of the basal ganglia after minor head injury is a well described entity. ‘Mineralising angiopathy’ is a term to describe mineralisation along the lenticulostriate vessels that predisposes infants to stroke, even after trivial head injury. The prognosis in these children is excellent. The supratentorial compartment is relatively mobile in comparison to the fixed infratentorial compartment offering greater shearing forces between the perforating vessels and the brain parenchyma following an acute trauma. The acute angulation of the lenticulostriate perforators is further responsible for its propensity of the basal ganglia for the insult. The current report is unique as it presents a young boy with hemorrhagic stroke in the basal ganglia after a trivial trauma and imaging findings suggestive of mineralising angiopathy.

Keywords: mineralising angiopathy, basal ganglia infarct, lenticulostriate vessels.

Introduction

Infarction of the basal ganglia after minor head injury is a well-described entity. ‘Mineralising angiopathy’ is a term to describe mineralisation along the lenticulostriate vessels (LSV) and is usually seen after a trivial head injury with a presentation as acute stroke [1]. The prognosis in these children is excellent. The supratentorial compartment is relatively mobile in comparison to the fixed infratentorial compartment, offering greater shearing forces between the perforating vessels and the brain parenchyma following an acute trauma [2]. The acute angulation of the lenticulostriate perforators is another postulate, suggesting the propensity of the basal ganglia for the insult [3]. In this case report, we present a young boy with hemorrhagic stroke in the basal ganglia after a trivial trauma and with imaging findings suggestive of mineralising angiopathy.

Case description

A nine-year-old boy presented with a history of trivial head injury over the left parietal area after a trivial fall while playing cricket. He had no loss of consciousness, nor bleeding from nose, mouth or ear. He did not sustain any external injuries. An hour later, he noticed sudden onset weakness of right upper and lower limb and deviation of angle of mouth to the left. His blood pressure when he was brought to the hospital was 110/70 mmHg. All his peripheral pulses were regular in volume and character and normal in rate and rhythm. Cardiac examination was normal. He had an upper motor neuron palsy of the right seventh nerve. The power was 0/5 in the right upper and lower limb according to MRC (medical research council) grading, with extensor plantar reflex and brisk deep tendon reflexes on the right. The rest of the neurological examination was normal.

Investigations showed hemoglobin of 12.5g/dL, a normal differential white blood cell count and platelets, and no evidence of coagulopathy. His vasculitic profile included the presence of titres in the serum antinuclear antibodies (ANAs), antibodies to extractable nuclear antigens (ENAs), anti-native DNA antibodies (anti-DNA), topoisomerase I antibodies (Scl-70), antibodies to cyclic citrullinated peptides (anti-CCP), rheumatoid factor (RA factor), C-ANCA (myeloperoxidase) and p-ANCA (proteinase 3) levels, which were normal. Serological markers, including both IgG and IgM antibodies against cytomegalovirus, toxo-
plasma, rubella and herpes, were negative. He was negative for HIV (human immunodeficiency virus) and the Hepatitis B surface antigen test. An electrocardiogram (ECG) and a two-dimensional echocardiography (2D Echo) were normal. A metabolic work-up test, including serum calcium, phosphorus, magnesium, thyroid, parathormone and vitamin D levels, was normal. A sickling test was negative.

A computerised tomogram (CT) of the brain revealed hypodensity in the left basal ganglia and a small speck of hyperdensity on the right (Figure 1). MRI (magnetic resonance imaging) confirmed the bleed in the left basal ganglia and showed no other parenchymal changes while the MRA (magnetic resonance angiogram) was normal. Possibilities considered were mineralising angiopathy with a hemorrhage in the left basal ganglia after a trivial trauma and an arteriovenous malformation. The patient was ad-
a causal association.

Competing interests
The authors have declared that they have no competing interests.

Author contributions
All the authors contributed to and also critically reviewed the manuscript. The final version of the manuscript was approved by all the authors.

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