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Case Report

Seizure and embolic stroke secondary to patent foramen ovale in a migrant girl

Joseph V Turner[1,2]

[1]Rural Clinical School – School of Medicine, University of Queensland, Brisbane, [2]Queensland Health, Australia

ABSTRACT

Episodes of loss-of-consciousness in school-aged children are not uncommon and include metabolic, cardiovascular, neurological, and psychiatric/psychosocial causes. This case describes a first episode of seizure in an 11-year-old migrant girl. History and language issues contributed to the diagnostic dilemma. Ongoing neurological symptoms prompted transfer to a tertiary care facility where investigation revealed patent foramen ovale (PFO) with likely paradoxical embolus. Prevalence and management of this condition is briefly reviewed in the context of the current presentation.

Key words: ASD, embolic stroke, ischemic stroke, paradoxical embolus, right-to-left shunt, PFO

Introduction

Episodes of pre-syncope and syncope in school-aged children are not uncommon. Differential diagnoses are broad and include metabolic, cardiovascular, neurological, and psychiatric/psychosocial conditions. These can be approached using either/both emergent and probability diagnostic strategies. One uncommon cause is paradoxical embolus secondary to a patent foramen ovale (PFO). While PFO-related ischemic events have been described in the literature, the case described here presents a diagnostic dilemma with confounding factors including history, ethnicity, and issues of rurality.

Case Report

An 11-year-old girl from non-English speaking background (NESB) was brought into the Emergency department at 10:30 am with apparent seizure-like activity at school mid-morning during the summer. It was reported that at school assembly she fell backwards when trying to rise from sitting with upper limb twitching and hand-clenching, as well as drooling and left-sided facial droop. On arrival she was responding to verbal stimuli and did not look unwell although was also complaining of headache and nausea. Breakfast consisted of only a glass of milk and she did not describe any other unwell symptoms pre-morbidly. Communication was difficult due to her very limited English.

On examination her Glasgow Coma Scale (GCS) was 14 (eyes 4, verbal 4, motor 6) with occasional dysrhythmic jerking movements of her upper limbs and torso. Vital observations and blood sugar were all within normal limits. Physical examination revealed a left facial droop with tongue deviation to the left and midline uvula. There was no photophobia or meningism. The left upper limb strength was decreased with respect to the right but there were no other abnormal findings. Lower limb tone and reflexes were normal and there was no clonus. More detailed examination was not possible due to the communication barrier.

The patient was admitted for neurological monitoring. Over the course of the day symptoms and signs improved and she was eating and mobilizing independently. Apart from hypochromic microcytic RBCs, her blood tests including full blood count, urea, electrolytes, creatinine, and liver function tests were all normal.

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Address for correspondence:
Dr. Joseph V Turner, Rural Clinical School – School of Medicine, University of Queensland, c\- Locked Bag 5009, Toowoomba DC, QLD 4350, Australia. E-mail: josephvturner2110@gmail.com

Review in the evening revealed new left ankle clonus and left knee hyper-reflexia. Facial droop was still mildly evident. The new and persisting neurology necessitated patient air-evacuation to a tertiary center for further investigation.

CT and MRI head demonstrated abnormalities in the posterior right putamen extending superiorly to the right periventricular region, and also in the posterior frontal cortex just superior to the insular and a small area of anterior parietal cortex. These areas demonstrated diffusion restriction and were associated with a minimal mass effect. The appearance was in keeping with ischemia.

Subsequent echocardiogram showed a small PFO with positive contrast study consistent with paradoxical right-to-left shunt across the interatrial septum on straining. This right-to-left shunt put her at risk of paradoxical embolism.

Thrombophilia blood investigations were unremarkable.

Discussion

The prevalence of PFO on autopsy is around 25% and is generally regarded as a common and benign condition.[1] The prevalence decreases with increasing age, with a peak of 34% in the first three decades.[2] In patients under the age of 55 years with cryptogenic stroke, a PFO has been found on transesophageal echocardiograpy in 40-50% of cases.[3] One meta-analysis found a significant association between PFO and cryptogenic stroke, and that the size of the right-to-left shunt was also an important contributing factor.[4] It is thought that a PFO enables paradoxical right-to-left shunting and may permit paradoxical emboli arising from the venous system to enter the arterial system, including the intracranial circulation. Shunting may have increased during exertion upon rising from sitting in the case of this 11-year-old girl. She did not fulfill the criteria for migrainous cerebral infarction.[5]

There is no current agreement in recommending primary prevention of stroke in people with asymptomatic PFO. Options for secondary treatment of cryptogenic stroke associated with PFO include antiplatelet therapy, anticoagulation, surgical and transcatheter closure of PFO. It has been suggested that percutaneous closure may provide better outcomes than medical treatment[6] although the margin of potential benefit of PFO closure in preventing stroke recurrence may be narrow.[7] In considering management strategies it is worth noting that quality randomized controlled trials in this area are lacking and that there is insufficient evidence to recommend warfarin and/or PFO closure.[8] The functional prognosis of cryptogenic PFO-related strokes is better than for non-PFO related strokes.[9]

Management for the patient reported here consisted of PFO closure with an Amplatzer device. She was anticoagulated with enoxaparin subcutaneously for 6 months given her acute ischemic brain injury and neurological signs. A tertiary center review at 3 months post closure showed PFO device in good position with no residual flow and complete resolution of symptoms. She was discharged following a 1 year post-event review.

Tertiary centre review necessitated travelling more than 8 h by road or 2 h by plane. Organizing transport and communication during review appointments were made more difficult by the NESB of the parents. The year prior to this, her parents had their other child evacuated from the rural hospital for reasons they could not understand. As a result, they were particularly upset and fearful when their 11-year-old girl had to fly out.

Initial diagnosis of the PFO upon presentation of this patient was hampered by communication issues and confounding history. Fortunately aeromedical retrieval services facilitated rapid transfer of the patient to a tertiary center where definitive diagnosis was achieved.

Conclusion

Given the high prevalence of PFO, uncommon causes of seizure such as embolic stroke secondary to PFO may occur in any setting. Differential diagnoses must be accounted for according to an emergency diagnostic model first, with due consideration of probability diagnoses. A strong suspicion for an embolic or neurologic cause should be present if there are persisting neurological symptoms or signs.

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