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Acute ischaemic stroke (AIS) is an important cause of disability and death in children. It affects 1.2-8 children per 100,000 per year, and almost 70% of survivors have significant ongoing disability, resulting in reduced quality of life, increased care requirements for families, and cost[1]. Reperfusion therapies, which include intravenous thrombolysis and endovascular clot retrieval (ECR), are established as effective to improve long-term outcomes of adults with acute ischaemic stroke. Paediatric stroke consensus guidelines allow for reperfusion therapies in children, but access is currently limited due to diagnostic delays[2].

Keywords: neurology, emergency medicine, intensive care

Case Report

A 7-year-old girl presented to a paediatric emergency department (PED) at mid-day on a weekend with acute onset right-sided weakness. She had a background of tricuspid atresia with a hypoplastic right ventricle, and had undergone uncomplicated closure of an extracardiac Fontan fenestration using a 6mm Amplatzer Septal Occluder 6 weeks prior to her hospital presentation. She was taking prophylactic aspirin 100mg daily and no other regular medications.

At 10:30am she developed acute right-sided weakness and aphasia. Her parents were concerned about possible stroke and immediately contacted metropolitan ambulance services.

Ambulance staff suspected an AIS and pre-notified the PED of her symptoms. A ‘Code Stroke’ was activated prior to patient arrival.

On arrival to the PED at 11:56 am, she had a patent and protected airway, with no breathing or circulatory compromise, and normal vital signs. Her initial Glasgow Coma Scale score was 12; eyes spontaneously opening (4), making unintelligible sounds (2), and obeying motor commands (6). The Code Stroke team found her to have an expressive aphasia, a dense right-sided hemiplegia affecting the arm, leg and face, gaze paresis with eye deviation to the left and right-sided hemi-neglect. Her Paediatric National Institute of Health Stroke Score (PedNIHSS) score was 20 (range: 0 (minimum severity) – 41 (maximum severity)).

MRI under general anaesthesia performed at 12:50pm showed areas of restricted diffusion within the middle cerebral artery territory with involvement of the left caudate nucleus, putamen and pre-central gyrus. Time-of-flight MRA demonstrated complete proximal left middle cerebral artery (MCA) occlusion. Perfusion imaging demonstrated a large area of ischaemic brain involving almost the entire MCA territory (Figure 1).

Management of her AIS involved transfer via the Paediatric Infant Perinatal Emergency Retrieval service to an adult interventional radiology service at a separate adult hospital. She arrived at 14:07 pm. ECR was performed at 15:15pm, with single pass resulting in reperfusion of the M1 and M2 segments and Thrombolysis in Cerebral Infarction (TICI) Grade of 3 (range: 0 (no perfusion) – 3 (complete perfusion)). Onset to needle time was 4 hrs and 45min. Some distal non-occlusive thrombus remained at M3.

Post ECR angiogram demonstrated almost complete reperfusion of the left MCA. Repeat MRI 24 hours post ECR confirmed recanalisation of the left MCA with no residual thrombus and a stable distribution of diffusion restriction (Figure 2). Transthoracic and transoesophageal echocardiograms did not demonstrate intracardiac thrombus or thrombus in the Fontan conduit.

She was extubated uneventfully, commenced on heparin followed by warfarin, and transitioned to a rehabilitation service for functional recovery. PedNIHSS score was 5 at seven days post event. At one-month follow-up, she was eating independently, talking fluently, and mobilising independently. She has had near-complete resolution of her right hemifacial weakness. Her main ongoing deficit was weakness in her right (dominant) hand, with a Pediatric Stroke Outcome Measure (PSOM) of 1 (range: 0 (no deficit)-10 (maximum deficit)).

Discussion

Reperfusion therapy is a time critical intervention. ECR is beneficial for adults with AIS due to large-vessel occlusion if performed within 6 hours of symptom onset, though patients with a salvageable ischaemic penumbra may benefit from ECR up to 24 hours following symptom onset[3, 4]. The role of ECR in children with AIS is uncertain but a recent systematic review of retrospective data suggest that most children have successful recanalisation and good long-term neurological outcomes with low rates of intracranial haemorrhage (similar to those reported in adults)[5]. The PedNIHSS is a child-specific adaptation of the adult National Institutes of Health Stroke Score, and can be used to quantify AIS severity and recovery. The PSOM is a standardised neurologic outcome score that is predictive of long-term motor and adaptive behaviour outcome. The ischaemic penumbra on perfusion imaging performed

following AIS represents the volume of brain that is potentially salvageable with acute interventions such as thrombolysis or ECR. The TICI scale is a neuro-interventional radiology grade used to describe angiographic outcomes following ECR. Timely and complete reperfusion of the affected MCA territory prevents expansion of the core infarct and restores blood flow to the penumbral tissue, in this case preventing long-term aphasia, hemiplegia, and ongoing rehabilitation and care requirements.

Barriers to rapid *diagnosis* of AIS in children include: infrequency; delays in hospital presentation, with a median duration from onset of symptoms to hospital presentation of 24 hours; difficulties in differentiating childhood stroke from stroke mimics, such as migraine and seizure associated Todd's paresis; and delays to definitive imaging[6].

Children with complex cyanotic congenital cardiac disease are a high-risk population for AIS, with a lifetime risk of 1/100. Contributing factors include right to left cardiac shunt, increased systemic venous pressure, decreased cardiac function, altered flow dynamics, arrhythmias, polycythaemia, and foreign devices or materials[7]. Children with a Fontan circulation are at increased risk of neurological injury, including thromboembolic events, the risk of which may be reduced through closure of the fenestration. As seen in this case, however, AIS is a recognised, low incidence complication of fenestration closure procedures[8].

Hospital Code Stroke protocols sit within a broader system of acute stroke care that includes paramedic clinical practice guidelines, and predefined criteria for bypass to comprehensive stroke centres, clinical stroke recognition tools, rapid MR and CT protocols that include

perfusion imaging and adherence to standard adult inclusion and exclusion criteria for reperfusion therapies.

This case highlights the importance of developing code stroke protocols for children that facilitate rapid assessment and diagnosis of AIS to enable timely access to reperfusion therapies. Appropriate partnerships with adult neuroradiology interventional centres are essential given the relative infrequency with which ECR is performed in children. Prospective multicentre registries are also important to collect efficacy, safety and long-term outcome data[2].

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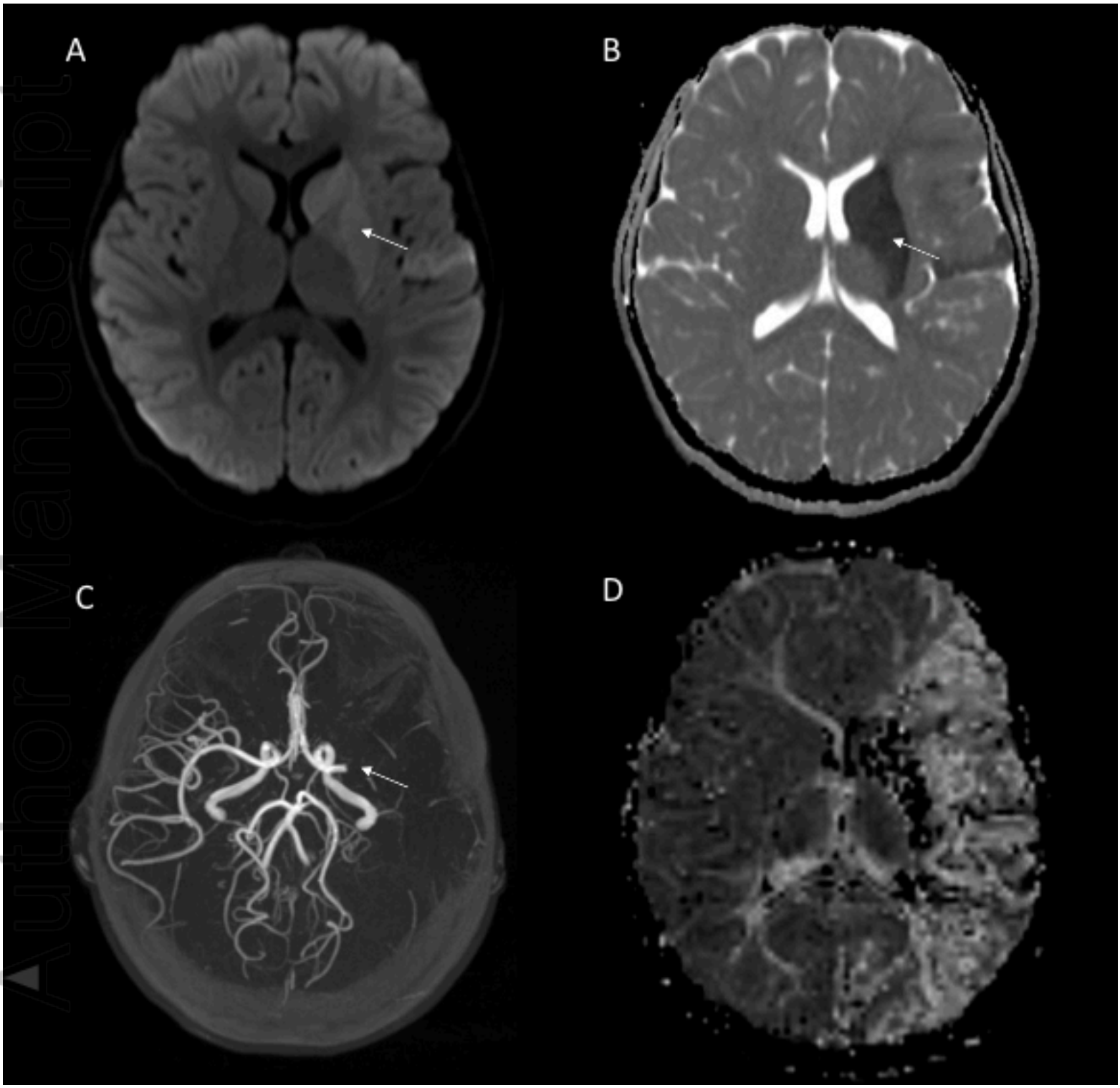
Figure Legends

Figure 1. Pre-intervention, diffusion weighted (A), apparent diffusion coefficient (B) Magnetic Resonance Imaging brain showing restricted diffusion in the left caudate nucleus and putamen. Time-of-flight angiogram (C) showing abrupt cut-off at the distal M1 segment of the left middle cerebral artery. Axial relation between relative cerebral blood flow (relCBF) demonstrating a large area of hypo-perfused brain. The ischemic penumbra (representing salvageable brain) is approximated by subtracting the diffusion lesion (core infarct) from the diffusion lesion (D).

Figure 2. Post-intervention angiogram (A) demonstrating absent flow past the M1 segment of the left middle cerebral artery. Post-endovascular clot retrieval angiogram (B) showing almost complete reperfusion of the left middle cerebral artery. Diffusion-weighted T2 axial Magnetic Resonance Imaging brain (C) performed 24 hours following endovascular thrombectomy showing expected evolution of previously seen changed in the left caudate and putamen but very little expansion of DWI changes to the rest of the left middle cerebral artery territory, and time of flight angiogram (D) performed 24 hours post endovascular clot retrieval showing restored perfusion to the left middle cerebral artery territory.



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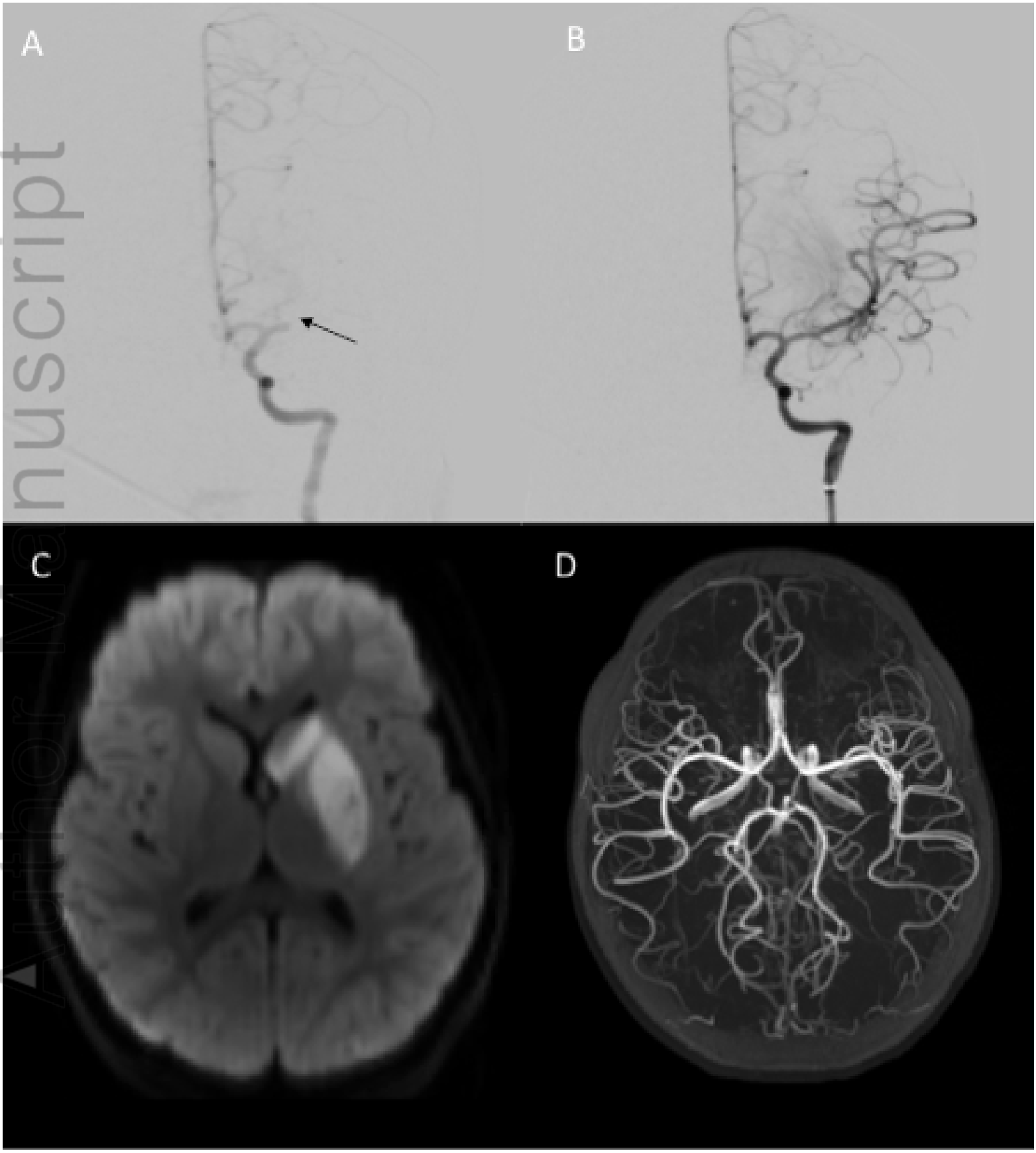

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