We describe a case of a 3-year-old girl with bilateral pontine and cerebellar infarction due to basilar artery thrombosis, related to a diagnosis of exclusion of severe iron deficiency anaemia who was successfully treated by mechanical thrombectomy.

Case Report Our patient presented with a one-day history of vomiting, and headache, on a background history of a viral prodrome the preceding week, and varicella infection three months earlier. Her haemoglobin was 4.6 g/dL, with a profoundly microcytic, hypochromic blood film. She received a blood transfusion with clinical improvement. At 36 hours after admission, she became irritable and developed a left divergent squint (NIHSS score 4). Neuroimaging demonstrated acute infarction of the pons, cerebellum and punctate lesions in both occipital lobes. MRA showed complete occlusion of right vertebral artery and basilar artery. Enoxaparin was commenced, but 12 hours later she developed left CNVII palsy and a dense left hemiparesis with hypertension. CT Brain showed worsening ischaemia. Given her ongoing clinical deterioration with major risk of significant morbidity and indeed mortality she underwent mechanical thrombectomy. Successful recanalisation of the basilar artery occurred with a distal left parieto-occipital thrombus remaining. At 9 months follow-up she has a mild left hemiparesis, left CN VII, III and IV palsies and right CNVI palsy, but mobilises independently and is on a normal diet (mRs 2); imaging shows established pontine infarction with gliosis.

Conclusion This case adds to the limited reports of mechanical thrombectomy in children in posterior circulation AIS, as a safe and effective treatment. It also highlights the importance of recognising severe iron-deficiency anaemia as a cause for AIS in children.

GP234 THROMBOLYSIS OF ARTERIAL ISCHAEMIC STROKE IN AN EIGHT MONTH OLD: THE FIRST IRISH CASE

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10.1136/archdischild-2019-epa.293

Paediatric arterial ischaemic stroke (AIS) is a potentially fatal condition with serious long term neurological sequelae. The rate of paediatric ischaemic stroke is 1.6 – 3.3 per 100,000 and 61% of these children are left with long term neurological difficulties. Thrombolysis guidelines for children were introduced in May 2017 by the Royal College of Paediatrics and Child Health (RCPCH). However, these guidelines were developed for children from the age of eight and up, and may be used on a case by case basis for children from the age of two to eight years. There is very limited data on the use of thrombolysis in children of any age, particularly in the under two age group. Here we present the first case of AIS thrombolysis in an under two year old in Ireland.

An eight-month old boy presented with a two-and-a-half-hour history of decreased movement of his left side. Importantly, his mother had noted the exact time symptoms commenced. His brain imaging showed a large right sided area of ischaemia in the territory of the middle cerebral artery (MCA), and a narrowed right internal carotid artery (ICA); no haemorrhage was seen. He was brought to intensive care and thrombolysis was commenced with alteplase and heparin. Methylprednisolone was also given as the narrowing of the

ICA was felt to be inflammatory. Other investigations, including echocardiogram, homocysteine level, thrombophilia screen and varicella titres, were all negative.

He has recovered well. He is back on full oral feeds and has learned to walk. He is right hand dominant, with no left sided neglect. He has no speech delay and has been discharged by both physiotherapy and occupational therapy.

Enrolling children in randomised controlled trials to assess AIS treatment and outcomes remains extremely difficult, as evidenced by the Thrombolysis in Pediatric Stroke (TIPS) study, which was closed by the National Institute for Health (NIH) after only thrombolysing one patient despite recruiting 43. The issues centre on delays in presentation (usually outside the thrombolysis window), and in delayed recognition of AIS in emergency departments by clinicians. This case highlights the need for continued research in this area as it demonstrates that successful outcomes are possible after thrombolysis of AIS.

GP235 COL4A1 MUTATION INHERITED FROM MATERNAL MOSAICISM IN AN INFANT PRESENTING WITH MICROCEPHALY, HAEMOLYTIC ANAEMIA AND CATARACTS

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10.1136/archdischild-2019-epa.294

Introduction COL4A1-related disorders have overlapping signs and symptoms as a result of fragile blood vessels. The COL4A1 gene located on Chromosome 13 is responsible for making the alpha one subunit of type IV collagen. Type IV collagen molecules form complex protein networks, which are the main component of basement membranes surrounding the body's blood vessels. Clinical presentation and the onset of symptoms are widely varied and largely dependent on the phenotype expressed. A number of phenotypes with overlapping features have been described. These include COL4A1 Brain Small Vessel Disease, AD Familial Porencephaly and HANAC (Hereditary Angiopathy with Nephropathy, Aneurysms and Muscle cramps) Syndrome. COL4A1 disorders are inherited in an autosomal dominant manner and the proportion of cases caused by *de novo* pathogenic variant is estimated to be 27%.

Case presentation A female infant was born at 36+2 weeks gestation via Emergency LCSC for IUGR and reduced fetal movements. Apgars were normal. BW was 1.88 kg (2nd centile) and Birth OFC 29.7cm (3rd centile). Initial exam was normal and the infant was admitted to the SCBU for Low Birth weight. She developed haemolytic anaemia week two of life and required three RCC transfusions on DOL 18, 26 and 35. Head growth was found to be static on week four of life and she was also noted to have absent red reflexes bilaterally. Cranial Ultrasound showed a large cystic abnormality in the right cerebral hemisphere. MRI Brain showed cystic encephalomalacia with a large right hemispheric cyst, simplified gyral pattern, significant volume loss and evidence of previous haemorrhage in left cerebral hemisphere. Ophthalmology confirmed bilateral cataracts. Extensive investigations including metabolic screen, infectious workup, Karyotype and microarray were all normal. Further genetic testing revealed that the child was heterozygous for novel COL4A1 missense