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Associations with arterial ischaemic stroke in childhood

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Introduction: While risk factors for cerebrovascular disease in adults are rarely seen in children with stroke, there is considerable controversy about the relative importance of putative aetiologies, at least in part because most published series have been small.

Methods: At Great Ormond Street Hospital, 186 consecutive children with ischaemic stroke in an arterial distribution have been studied over the past 9 years. There is little evidence that inherited deficiencies of protein C or protein S are common although abnormalities are commonly seen acutely. This abstract reports our data on other possible associations.

Results: In our series 85% had cerebrovascular disease or MR or conventional angiography; 120 had transthoracic echocardiography, of whom 18 also had a transoesophageal study. Apart from the 25 with known cardiac disease, an additional anomaly was found in five (four PFO or secundum ASD, one cardiomyopathy), who only required the transoesophageal technique for diagnosis; 55/147 (37%) were anaemic, only 14 of whom had sickle cell disease; 19/104 (18%) children were homozygous for the thermolabile variant of the methylene tetrahydrofolate reductase gene, but this was not significantly higher than the control population (12%) ($p=0.3$). Four of the homozygotes and two of 26 negatives/heterozygotes also had raised homocysteine levels (>13.5 umol/litre), which might be reduced by folate supplementation. Seven of 104 (7%) were heterozygous for the factor V Leiden mutation and two of 102 (2%) for the prothrombin 20210 mutation, neither of which were increased compared with controls; 23/77 (30%) had raised factor VIII levels; 37/100 VIII levels; 37/100 (37%) had raised anticardiolipin antibodies, of which 14 of the 24 repeated were still raised at follow up. At least 17 had evidence for recent varicella zoster infection and seven of 78 (8%) had raised titres to *Mycoplasma pneumoniae*; 111 had at least one potential risk factor and 48 had at least two.

Discussion: It is possible that preventable infections and nutritional deficiencies are important risk factors for childhood stroke but small numbers in a single centre preclude a definitive answer. There is a good case for a large collaborative case–control study, which could be conducted in Europe. This should involve detailed analysis of stroke subtype and other factors, such as the presence or absence of cerbrovascular disease.

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Encephalo-duro-arterio-synangiosis (EDAS) for Moya-Moya disease

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Moya-Moya disease is rarely diagnosed in Europe because it is uncommon in the population and childhood stroke syndromes are underinvestigated. There is little published experience of operative management and outcome of the condition in Europe. The authors present a small series accumulated at a single paediatric neuroscience unit.

Material and method: Retrospective chart review of cases referred to University Hospital Nottingham 1987–98.

Findings: Five children aged from 2 years to 12 years were encountered; three were seen from 1995 onwards. Two were referred from other regions of the country on the advice of the local paediatric neurosurgeon because of lack of local experience. Age of onset of symptoms ranged from 1 year to 10 years. Duration of symptoms prior to referral was 1 year to 5 years. All patients suffered multiple ischaemic episodes and three had fixed neurological deficits. Two also had epilepsy and learning difficulties. Two children also had a patent ductus arteriosus and one had Down syndrome. Four patients had established infarcts on CT or MR. All had typical appearances of Moya-Moya disease on conventional cerebral angiography. All underwent bilateral EDAS. Three children had no further ischaemic events; one child has noticed fewer and less profound transient ischaemic attacks but remains on treatment with antiplatelet drugs and calcium-channel blockers and has developed choreoathetosis requiring treatment with tetrabenazine; one child has had a marked reduction in the duration and severity of transient ischaemic attacks. One child showed regression of a fixed deficit within 2 weeks of operation. No child has suffered a new infarct and there have been no complications of surgery. Follow-up ranges from 13 years to 15 months. Two children have shown either reduced ischaemia or increased perfusion on HMPAO SPECT or perfusion MRI respectively.

Conclusions: EDAS was of therapeutic value in the small population studied. Moya-Moya disease and the availability of effective surgery is still underrecognized. There is a place for a European survey of childhood stroke with management along an agreed strategy.

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Stroke in full term newborn: EEG as early detector

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Stroke is not uncommon in term newborn but diagnosis can still be difficult because of possibly silent clinical and ultrasound (US) examination. US scanning of the brain may show ischaemic damage beyond the end of the first week, even more powerful imaging techniques such as MRI can be silent in the first 4 days after the insult.