can not only show "some abnormality that encourages angiographic examination" but can also diagnose dissections involving the transverse, C6–C5 and C5–C4 intertransversar segments of the VA. The diagnosis is based on the association of a localised increase in arterial diameter with haemodynamic signs of stenosis or occlusion and/or decreased pulsatility and intravascular echoes at the same level. Furthermore, ultrasonic examination is an excellent tool for the follow-up of dissection.

6) Among other diagnostic procedures, the authors did not mention thin-section contrast-transmission angiography (or CT scan and MRI). Due to its sensitivity to both blood flow and thrombus formation, its multiplanar imaging capability, and its non-invasiveness, MRI (especially of the cervical region) has become an excellent tool for the follow-up of dissection in the cervical artery territory following cervical manipulation. Necropsy revealed 2 VA dissections, a recent one probably due to cervical manipulation and a second one, a few weeks old, accounting for the initial cervical pain. This case demonstrates that cervical pain precedes and motivates chiropractic manipulation may be the first symptom of a hitherto unrecognized spontaneous (or traumatic) dissection and illustrates the difficulty in classifying a cervical dissection with certainty whether dissection is spontaneous or traumatic.

Apart from trauma and fibromuscular dysplasia, other conditions implicated as risk factors for dissection include migraine, oral contraceptives, and chronic high blood pressure. In a case-control study, we found a significant positive association of dissection with migraine and current oral contraceptive use but not with hypertension. However, the mechanisms leading to this association remains speculative.

8) Finally, we agree that anticoagulants are not harmful in extracranial VA dissection and may even be of benefit although no conclusion can be drawn from the comparison of nonrandomised treatment groups.


Late onset globell cell leukodystrophy

I read with great interest the paper by Grewal et al.1 I would like to add a few comments. First, the authors suggest that their patient's late onset (at age 14) distinguishes his disease from globell cell leukodystrophy (GLD) distinct from the infantile and late infantile/young ages. This may be true for the first type, but the latter can occur within one family together with a later onset type. 

Second, it would be interesting to know whether the white matter hyperintensities on the MRI were diffused or rather restricted to the occipito-parietal white matter, as described in other late onset GLD.2 If so, this posterior white matter involvement on MRI would seem to be very distinct from GLD from other cerebral white matter diseases.

PETER VERDRU
Neurologie, Universitaire Ziekenhuizen, Vreeheuvel, B-3000 Leuven, Belgium

Aseptic meningitis associated with high dose intravenous immunoglobulin therapy

We read with great interest the report by Watson et al.3 and we would like to draw the authors attention to a similar case we published last year.4

Our patient was a seven year old boy with idiopathic thrombocytopenic purpura who had well-controlled episodes of acute aseptic meningitis on two occasions prior to the second intravenous dose of immune globulin. On these two occasions, the patient developed aseptic meningitis on day three; quite identical to the patient's patients reported by Watson, whereas Kato's patient developed the aseptic meningitis two days after a five day course of intravenous immune globulin therapy.5

In our patient the immune globulin preparation used was Sandoglobulin IV (Sandoz), which is a formulation prepared by cold ethanol fractionation. It was given at a dose of 0.4 mg/kg body weight infused over a 1 hour period.


8 Hinse and Thie reply: We thank Dr. May and colleagues for their interest in our recent paper,1 and we appreciate the opportunity to comment on a few of the issues raised by them.


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Late onset globoid cell leukodystrophy.

P Verdu

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