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Paraganglioma of the cauda equina

Sir: Paraganglioma of the cauda equina is a rare tumour which was first described in 1972.[1] Recently Anderson and Gullan[2] reported the occurrence of this tumour in a 63 year old woman and commented on the small number of reported cases. We would like to document 2 more cases of paraganglioma of the cauda equina.

Case A was a 50 year old man who was referred to the University Hospital of Wales, with an 8 year history of back pain. In the year prior to admission the pain had become associated with paraesthesiae in the buttocks and legs, and he had developed hesitancy of micturition. The only positive physical signs on examination were absent tendon reflexes, and pain on movement of the lumbar spine. A radiculogram revealed a complete block to the downward flow of contrast at the L3 level. At operation a vascular tumour occupied most of the lumbar spinal canal. The tumour was adherent to the roots of the cauda equina. The tumour was excised except for a small amount of capsule adherent to the nerve roots. Postoperatively he was given a course of local radiotherapy. He was last seen 2 years after surgery, and was well with no further back pain.

Case B was a 38 year old man, who presented at the Dundee Royal Infirmary with a 6 month history of back pain, which radiated down the back of the thighs. The only abnormal physical signs were a positive bilateral femoral nerve stretch test, and mild weakness of the left quadriceps. A myelogram showed a rounded intradural mass at the L1 level. At operation the dura was opened to reveal a vascular mass measuring about 2 cm, which was lightly adherent to the roots of the cauda equina. The tumour was completely excised. Post operatively he made a good recovery. He was last seen 6 months after surgery, and was well with no further back pain, and no neurological deficit.

Histological examination of the tumour from Case A included examination of a smear preparation at the time of operation. The smear revealed separate groups of round to oval nuclei with eosinophilic cytoplasm, and occasional rosette formation. The appearances were quite similar to smear preparations of an ependymoma. Paraffin sections of the tumours revealed a vascular stroma, with nests and sheets of round to oval nuclei, with eosinophilic cytoplasm. A very occasional mitosis was evident in Case A, and there was no mitotic activity seen in Case B. A Grimelius stain demonstrated numerous neurosecretory granules within the cellular cytoplasm in both cases. Using a peroxidase labelled antibody system both tumours showed a strong positive reaction for neuron specific enolase. Electronmicroscopic examination revealed the presence of cytoplasmic membrane bound granules which measured between 800–1500 Å. The diagnosis of paraganglioma was made in both cases.

There have been few descriptions of smear preparations of these tumours, and it is of interest that the smear preparation from our Case A, and of that reported by Gaffney, Doorly and Din[3] resembled an ependymoma. In this respect Anderson and Gullan emphasised the importance of making the correct diagnosis with regard to both prognosis and treatment. We have found 18 reported cases in the literature,[1–17] which with the addition of our two cases brings the total to 20. We observe that all of these cases appear to have followed a benign course, but documentation and follow up remains important in furthering our knowledge of the long term behaviour of paraganglioma of the cauda equina. Male predominance is suggested for these tumours, since 14 of these cases were males.

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Does the peripheral blood leukocyte count predict the risk of transient ischaemic attacks and strokes?

Sir: The peripheral blood leukocyte count has been shown to be a predictor of myo-

cardial infarction.[1] Some evidence suggests that this is to be explained by smokers' leukocytosis,[2] though other studies have shown an effect independent of smoking status.[3] The mechanism is debated but leukocytes affect flow in the microcirculation, and influence platelet aggregation,[4] and Light[5] has suggested their proteinases might affect the development of atheroma.

To investigate the possibility that the leukocyte count predicts clinical events in the case of cerebrovascular disease, we have reviewed a group of patients with transient ischaemic attacks (TIAs) and related their leukocyte count at presentation to their subsequent course. The clinical outcome of some of these patients has been reported previously,[6 7] many being from the study of strokes in young people.[7]

The case records of 68 patients (56 males and 12 females of average age 48 years) presenting to one of us (JM) with a history of recent TIAs were reviewed. Patients with a recent completed stroke were excluded as a leukocytosis may be a response to a recent infarct or haemorrhage. Patients with polycythaemia rubra vera were also excluded. Note was taken of conventional risk factors, age, sex, blood pressure and smoking status. The white blood cell count recorded at their first clinic or hospital visit was extracted from the notes. The presence or absence of subsequent TIAs or strokes during a follow up period of an average of 5 years was also noted.

Twenty three patients had had further TIAs or strokes. The age, sex, blood pressure, smoking status, duration of follow up and leukocyte counts at presentation of those with and without further events is shown in the table. There was no difference in sex distribution, the prevalence of a blood pressure over 150/90 mm Hg, the proportion currently smoking or in the length of follow up achieved, between the two groups. Those with subsequent cerebrovascular events were slightly older (51·8 ± 6·9 years cf 46·0 ± 9·5 years, $t = 2{\cdot}5$, $p < 0{\cdot}02$), and had higher leukocyte counts ($9{\cdot}74 \pm 1{\cdot}1 \times 10^9$/l cf $7{\cdot}774 \pm 2{\cdot}5 \times 10^9$/l, $t = 3{\cdot}25$, $p < 0{\cdot}002$).

Fifty three per cent of patients with leukocyte count over $8{\cdot}0 \times 10^9$/l had had further events whilst only 12·5% of those with a count of $8{\cdot}0 \times 10^9$/l or less had (Chi Square 10·5, $p < 0{\cdot}002$). Amongst the smokers there was still an apparent association between leukocyte count and risk. Thus the mean leukocyte count in smokers with subsequent events was $10{\cdot}55 \pm 1{\cdot}9 \times 10^9$/l, that in smokers with no further TIAs or strokes was $8{\cdot}05 \pm 2{\cdot}36 \times 10^9$/l ($t = 3{\cdot}72$, $p < 0{\cdot}001$). The chance of further events for a smoker with a leukocyte count over $8{\cdot}0 \times 10^9$/l was 61% with a count of $8{\cdot}0 \times 10^9$/l or less (n = 19), it was zero (Chi Square 15·5, $p < 0{\cdot}001$).

Relationship of risk factors including the leukocyte count to recurrence of TIAs or strokes

Risk factors	*Patients with further events* n = 23	*Patients with no further events* n = 45
Mean age (years)	51·8 ± 6·9	46·0 ± 9·5*
Sex	20 M 3 F	36 M 9 F
BP ⩾ 150/90 mm Hg	11 (48%)	19 (42%)
Smokers	17 (74%)	30 (67%)
Mean follow up (y)	5·2 ± 3·3	6·0 ± 4·7
Leukocyte count	9·74 ± 2·2	7·74 ± 2·5†

*$p < 0{\cdot}02$ Student's t test.
†$p < 0{\cdot}002$ Student's t test.

This small retrospective study of young patients with early evidence of cerebrovascular disease presenting with one or more TIAs suggests that the leukocyte count in the peripheral blood may be predictive of the risk of further cerebrovascular events. A similar trend was obvious in a study from Hiroshima though these were asymptomatic patients not at such a high risk,[8] as those with TIAs.

The data permit of no conclusion as to mechanism though the difference in apparent risk is clear within smokers as shown for myocardial infarction by Zalokar *et al.*[2] It is possible that the degree of smokers leukocytosis is a reflection in some way of the biological impact of smoking in the individual. Other risk factors may be involved since leukocyte counts are higher in women on an oral contraceptive.[9]

The evidence suggests that more investigation of the role of leukocytes in thrombosis,[4] on blood rheology,[10] and in atherogenesis is warranted.

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Correction

Cysticercosis in the UK (*J Neurol Neurosurg Psychiatry* 1987;**50**:1080). The title of this letter should have been Cysticercosis in Birmingham and the authors C Shieff, ER Hitchcock, SP Valsangkar.