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Letters to the editors

Transient global amnesia and high haematocrit levels

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Sir: Since being described by Fisher and Adams, transient global amnesia (TGA) has been defined as a loss of short-term memory characterized by an incapacity to form new memories, repetitive queries, retrograde amnesia and absence of other neurological signs and symptoms. Although TGA has been attributed primarily to a vascular aetiology, episodes of TGA have been related to different causes—hypoglycaemia, encephalitis, migraine, cerebral embolism, intracranial hematomas, and intracranial tumours—and some cases have been attributed to an epileptogenic mechanism [5]. Polycythaemia rubra vera (PRV) has frequently been associated with neurological signs and symptoms generally caused by cerebral vascular attacks. Pearson et al. [8] have shown that the incidence of vascular episodes in PRV depends on the haematocrit value (HCT). In order to minimize the chance of thrombotic episodes, the HCT should be maintained below 45.0% because cerebral blood flow is likely to be restored to normal when the HCT is reduced to this level [10]. To our knowledge only one previous case of TGA in a patient with PRV has been described [9]. A new case is described in this report.

A 56-year-old woman was admitted to out Haematology Department with a history of headaches. During the last 2 years she had complained of tinnitus and dizziness due to possible vertebrobasilar ischaemia. Physical examination showed only mild (4 cm) splenomegaly. Blood analysis showed: 7.7 RBC $\times 10^6$/mm^3, HCT 59.9%, haemoglobin (HGB) 19.9 g/dl, MCV 83 fl, MCH 28.9 pg, MCHC 33.5 g/dl, 10.4 WBC $\times 10^3$/mm^3, reticulocytes 18%, platelets 410 $\times 10^3$/mm^3, whole blood volume 5.468 l, plasma volume 2.050 l, and red cell volume 3.418 l (51.4 ml/kg). Bone marrow aspirate showed hyperplasia of all three series with no abnormal cells and leucocyte alkaline phosphatase of 140 (normal score 60). Bone marrow biopsy similarly revealed increased cellularity with megakaryocytic, erythroid hyperplasia with no adipose cells. Oximetry was 97%. Other complementary tests were normal. The patient was treated with 3.9 mc of ^{32}P. Two months later, after responding well to treatment, the patient experienced an hour-long episode of confusion following sexual intercourse. Her husband described preservation of long-term memory, repetitive queries such as where a certain object could be found, and absence of spatial disorientation. The patient had no headache during the attack and retrograde amnesia lasted approximately 1 h before the episode. Neurological examination on admission was within normal limits, as were EEG and CT scan. Blood analysis at this moment showed 5.8 RBC $\times 10^6$/mm^3, HCT 52.9%, HGB 16.1 g/dl, MCV 90 fl. High HCT level was thus found at the time of the episode.

This case fulfilled three major criteria of the Study Group for Diagnosis of Polycythaemia Rubra Vera [1]: (red cell volume over 32 ml/kg in women, oximetry over 92% and splenomegaly) and two minor criteria (thrombocytosis and increase of leucocyte alkaline phosphatase). Similarly, the patient fulfilled all criteria for TGA: (1) transient amnesic attacks with no direct relationship to cranial trauma or whiplash; (2) evidence given by a witness of inability to form new memories; (3) repetitive queries; (4) apparently normal behaviour and orientation; (4) evidence by witness of the normality of long-term memory; (6) presence of retrograde amnesia at least during the episode [5]. These criteria, although more restrictive than those demanded by other authors [2], eliminate other possible causes of the episode. The triggering of the episode by sexual intercourse, already described by Mayeux [7] and Fisher [2], and observed by ourselves [5], supports the diagnosis of TGA.

The Framingham data detected an association between high HGB level and the risk of cerebral infarction [4]. Toghy et al. [11] also observed that the incidence of cerebral infarction was higher with high HCT levels. A HCT of 50% or more was encountered in patients with carotid occlusion [8]. These reports have highlighted the possible role of a high HCT as a risk of cerebral infarction. We have conducted a prospective analysis of 42 patients of TGA, with a mean age of 59.2 years (16 male and 26 female). All criteria for TGA were present at the time of the attack. We have compared blood parameters with the levels found in healthy subjects. Student's *t*-test was used for statistical evaluation. A complete clinical assessment was made, with special regard to such risk factors as diabetes, hypertension, and migraine. Routine investigation included blood analysis, unidirectional Doppler study of extracranial cerebral arteries and CT scan. Migraine was found in nine patients (21.4%). Fourteen TGA patients were hypertensive (33.3%) and four had diabetes (9.5%). One patient had an internal carotid stenosis. Low density areas in the CT scan were observed in four cases (9.5%). The control group consisted of 68 healthy volunteers who had no history of blood or vascular disease, with an average age of 56.3 years (32 male and 36 female). We found HCT (control 41.42% ± 2.72, TGA 44.42% ± 4.24; $P < 0.001$) and HGB (control 13.71 g/dl ± 0.93, TGA 14.63 g/dl ± 1.53, $P < 0.001$) levels increased in patients with

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TGA compared with the control group. No differences were found in relation to RBC (control 4.77 $\times$ $10^6/mm^3$ $\pm$ 0.35; TGA 4.88 $\times$ $10^6/mm^3$ $\pm$ 0.50).

Many reports have suggested a vascular aetiology for TGA [5]. We have recently described an inverse correlation between high-density lipoprotein cholesterol to total cholesterol ratio levels and TGA, which supports a vascular mechanism for TGA [6]. Although PRV may be associated with TGA by chance, our case suggests a vascular pathogenesis, which is strengthened by our finding of elevated HCT and HGB in TGA patients.

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