

Cerebral borderzone infarcts after phlebotomy

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Introduction

In polycythaemia vera both the red cell volume and the plasma volume are elevated¹. A high haematocrit may also result from a normal red cell volume in combination with a contracted plasma volume, a disorder referred to as relative or spurious polycythaemia^{2,3}.

In patients with cerebral ischaemia and a concomitant high haematocrit, phlebotomy is advised to lower the red cell volume. A decrease of the haematocrit will lower viscosity⁴ and increase the cerebral blood flow⁵, from which patients in the acute phase of cerebral ischaemia might benefit^{6,7}. Removal of whole blood in patients with a spurious polycythaemia may, however, be hazardous, as these patients have a decreased plasma volume. We describe the development of borderzone infarcts after venesection in a patient with cerebral ischaemia.

Case report

A 68-year-old left-handed farmer was referred to our hospital because of a 'weak sensation' in his right arm, difficulty in counting and an unsteady gait. The symptoms had started two days before, but details about the mode of onset could not be given. The patient had a long history of hypertension, which had been treated with 100 mg hydrochlorothiazide a day. The

Summary

We describe a 68-year-old patient with cerebral ischaemia who underwent a phlebotomy to lower an elevated haematocrit (0.61 l/l). The procedure was complicated by the development of a total hemiplegia after two hours. CT scanning showed two borderzone infarcts.

We conclude that the plasma volume of patients with cerebral ischaemia and a high haematocrit may be contracted to such an extent that venesection is hazardous. The most appropriate treatment in these patients is expansion of the plasma volume.

Key words: borderzone infarcts, phlebotomy, spurious polycythaemia.

past history and the family history were normal. The patient did not smoke.

On admission the pulse rate was 78, the blood pressure 190/110 mm Hg. On general examination there were no abnormal signs, apart from conspicuous plethora of the face. The patient was confused, was disorientated in time, unable to write and to tell his date of birth. He had right/left disorientation and finger agnosia. In simple calculations he made several mistakes. In the limbs there were no abnormalities.

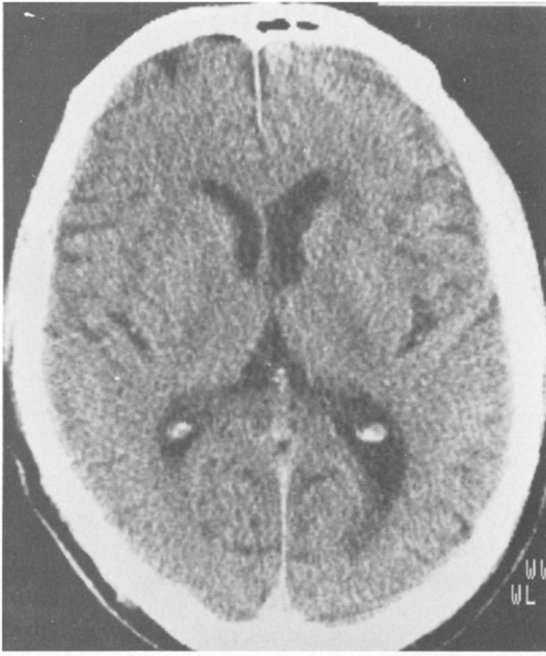
Laboratory analyses showed: haematocrit 0.61 l/l, haemoglobin 12.2 mmol/l, erythrocyte

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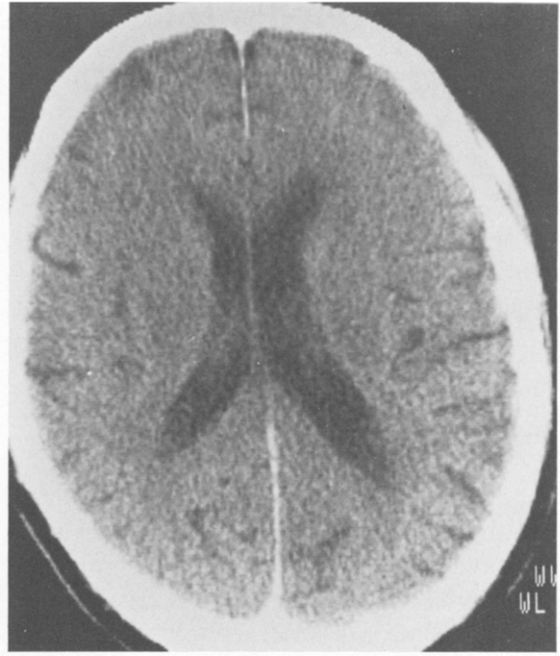
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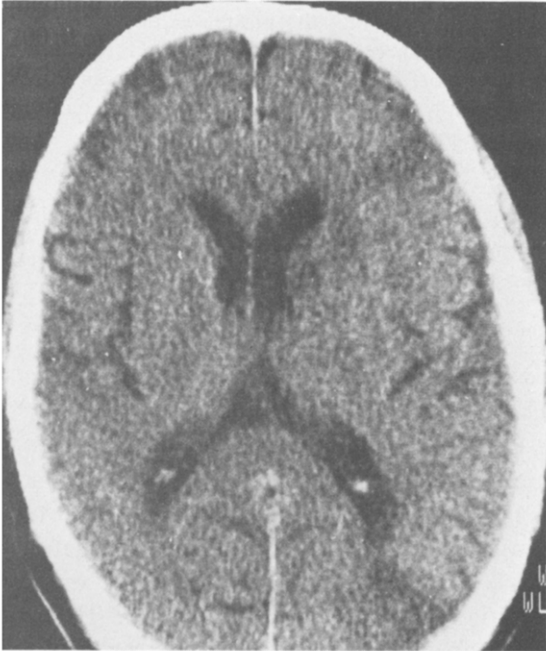
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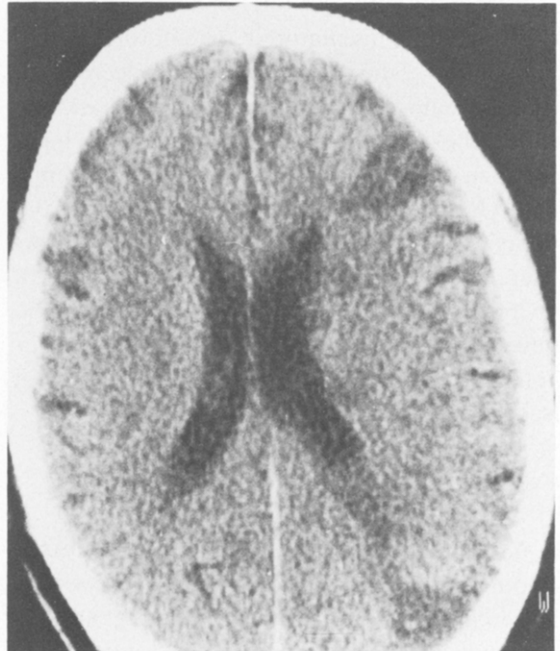
a



b



c



d

Fig. 1.
CT scan before (a + b) and after (c + d) phlebotomy.

count $6.6 \times 10^9/l$, MCV 83 fl, MCH 1680 fmol, MCHC 20.2 mmol/l, leucocyte count $8.2 \times 10^9/l$, thrombocytes $367 \times 10^9/l$, pO_2 kPa, pCO_2 5.2 kPa, bicarbonate 23.0 mmol/l, O_2 saturation 0.92, base excess -1.

Chest X-ray and electrocardiogram were nor-

mal. A CT scan was normal (Fig. 1a + b). Prolonged ECG monitoring and echocardiography showed no abnormalities. Doppler ultrasound examination, performed at a later stage, showed normal carotid arteries.

After admission the diuretic was stopped. 500 ml of whole blood was removed to correct the high haematocrit. Within two hours after completion of the phlebotomy the patient suddenly developed a total right-sided paralysis. Systolic

blood pressure had dropped 30 mm Hg (190 to 160 mmHg). A CT scan was repeated after three days and showed two borderzone infarcts (Fig. 1c + d). Fluid replacement was begun intravenously and haemodilution was thus continued until the haematocrit had decreased to 0.45. In the following weeks, the patient gradually improved. Residual deficits mainly consisted of a less skilful right hand and a slight weakness of the right leg. Extensive investigations into the cause of the polycythaemia were all negative. The hypertension was effectively treated with methyl dopa only. The haematocrit remained normal (0.47-0.49) during six months follow-up.

Discussion

We attribute the development of borderzone infarction in this patient to the removal of 500 ml of whole blood. These infarcts are caused by profound systemic hypotension⁸, which is usually found to be due to myocardial infarction, pulmonary embolus, internal bleeding or septicaemia. None of these conditions could be demonstrated in this patient. The removal of blood was advised because it was wrongly assumed that the high haematocrit was caused by primary proliferative polycythaemia. This was a very unlikely diagnosis, however, since the platelet count was within normal limits, as was the white cell count. Secondary polycythaemia was an equally unlikely diagnosis because of the absence of renal, pulmonary and cyanotic heart disease, and the presence of normal O₂ saturation. Therefore, the most probable diagnosis was spurious polycythaemia.

Since the mechanism of the original stroke of this patient is not clearly embolic, the initial ischaemia might also have been located in the borderzone area. The neurological examination on admission also fits such a localization. The sudden onset and extent of neurological deterioration of the patient shortly after venesection, however, imply that the aggravation cannot be simply regarded as a maturation of the original lesion, but that it was precipitated by the blood-letting procedure. An alternative explanation such as carotid occlusion could be excluded by Doppler ultrasound carotid evaluation.

Patients who present with cerebral ischaemia have a higher haematocrit than control patients⁹. In the weeks following the ictus the haematocrit shows a tendency to decrease spontaneously⁷. The elevated haematocrit might result from a contracted plasma volume rather than from an absolute increase in the red cell mass. The cause of the diminution of the plasma volume is unclear. It has been suggested that hypertension, stress and the use of diuretics might play a role^{2,3}. The patient described here used diuretics and after discontinuation the haematocrit remained within normal limits without any additional measures for at least six months. Thus, measurement of the plasma volume before venesectioning a patient is certainly the safest approach. It may be difficult, however, to await the results of such a time-consuming investigation before instituting a therapy in the acute phase of a stroke.

This case report demonstrates that the plasma volume may be decreased to such an extent that phlebotomy results in borderzone infarcts. To prevent the complications of phlebotomy it is advised to reduce the haematocrit by removal of small quantities of blood (250 ml) at a time and to replace an equal volume of fluid and to repeat this procedure until the desired haematocrit is reached^{5,10}. The plasma volume, however, will probably still remain contracted. The rationale for lowering the haematocrit in the acute phase of cerebral ischaemia is to improve cerebral blood flow by a reduction of blood viscosity. In addition, cerebral blood flow can be increased by a rise in cardiac output¹¹, but this will occur to a lesser extent if the plasma volume remains reduced. Therefore, it seems more appropriate to treat patients with a contracted plasma volume by expanding the plasma volume, since this both reduces the haematocrit and supports the increase of the cardiac output. Such a therapeutic regimen is currently being evaluated by a group of American investigators¹². In the near future it will be possible to compare their results with those of two European studies, the Scandinavian¹³ and Italian multi-centre trial, which both employ an isovolaemic haemodilution scheme.

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