Spontaneous mesencephalic hemorrhage: case report and physiopathological interpretation of its course

Tronci S., Congia S.
Cattedra di Patologia Speciale Neurologica - Università di Cagliari

We describe a case of spontaneous mesencephalic hemorrhage which evolved in 4 stages with complete clinical resolution. In the light of our findings and of published data we interpret the phenomenon in terms of the regional anatomy and vascularization.

Key-Words: Hemorrhage — mesencephalic — evolution

Introduction

Spontaneous mesencephalic hemorrhage is an infrequent clinical occurrence [2, 1]. As a rule sudden in onset, it is rarely due to vascular malformations [5]. It is often characterized clinically by headache and vomiting with disturbances of consciousness, no signs of meningeal irritation, alterations of vertical gaze and paralysis of the oculomotor nerves, disturbances of voluntary movement, of sensation and of control of movement (postural or intention tremor). The cases of spontaneous mesencephalic hemorrhage recorded in the literature [4, 5] all had a benign course, whatever the treatment.

We describe a case of mesencephalic hemorrhage evolving in four stages and seek to explain its favorable course in terms of the regional blood supply and anatomy.

Case report

A 35 year old normotensive man with no history of coagulation disorders or alcoholism complained of sudden frontal headache without nausea or vomiting. Within a few hours he had diplopia on upward gaze with left > right anisocoria and left palpebral ptosis. 2 days later he began to complain of paresthesias of the right side of the face and right hand and after a further 2 days mild right faciobrachial hemiparesis. He never had disturbances of consciousness. CT scanning on admission imaged a “hyperdense area of about 2 cm at midbrain level”. An angiogram of the posterior cerebral circulation a few days later revealed no vascular malformation of any kind; nuclear magnetic resonance imaged “... a 3 x 2.5 cm area of uneven signal hyperintensity with regular clearcut margins in the left cerebral peduncle...” (Fig. 1). After a first stationary phase the clinical symptoms began to recede: the patient began to walk again, at first with rightward cross-legged gait, but this soon normalized; the right arm weakness also disappeared at this time. ACT scan 3 months later showed complete reabsorption of the mesencephalic hemorrhage. On discharge the patient still had paresthesias of the right side of the face and right arm and complete palpebral ptosis with extrinsic and intrinsic ophthalmoplegia on the left side. At 6-month follow-up the clinical pattern had cleared completely with full recovery of extrinsic and intrinsic ocular movements of the left eye and no paresthesias on
Our case fits the stereotype of a midbrain tegmentum lesion, since the clinical symptoms denoted involvement of the surrounding structures (signs of cranial nerve III lesion, of involvement of the spinal and medial lemniscus and, due to distant compression, of the pyramidal tract), structures only partly detectable in a more caudal section.

On the evidence of previous cases [2, 1, 5, 3] this appears to be the most frequent site of spontaneous mesencephalic hemorrhage, for in the 9 cases of Scoville and Poppen and of Roig et al and in the 6 of Weisberg and in the case of Stern and Berneck impairment of vertical gaze and/or of cranial nerve III was constant, with accompanying symp-