Posterior fossa subdural hematoma mimicking intracerebellar hemorrhage

P. Miranda, R. Alday, A. Lagares, A. Pérez and R.D. Lobato
Servicio de Neurocirugía, Hospital “12 de Octubre”, Madrid, Spain

Abstract

Subdural hematomas of the posterior fossa are very rare and most cases are related to head injury. The influence of anticoagulation in cases of spontaneous development is well known. Although diagnosis is easily achieved by CT scan, atypical forms may lead to the wrong diagnosis of cerebellar hematoma. We present a case of a posterior fossa acute subdural hematoma occurring in an anticoagulated patient who was preoperatively misdiagnosed as an intracerebellar hemorrhage.


Introduction

Subdural hematomas rarely occur in the posterior fossa and the estimated incidence is less than 0.6% among intracranial subdural hematomas. Regarding its origin, head injury is the most frequent cause, and 30 to 50% of these traumatic cases show associated occipital skull fracture. Nontraumatic causes include rupture of an aneurysm or an arteriovenous malformation in the posterior fossa. Finally, spontaneous hematomas have been rarely described, anticoagulation being the main risk factor implicated in such instances.

Acute subdural hematomas in the posterior fossa usually manifest by a sudden decrease in the level of consciousness and rapidly progressing respiratory failure. Development of obstructive hydrocephalus may also be observed. Surgical emergent evacuation is generally indicated.

We report a new case of spontaneous posterior fossa subdural hematoma occurring in an anticoagulated patient and comment the radiological appearance and the clinical management of this infrequent entity.

Case report

A 74-year-old woman was operated to substitute a dysfunctional mitral prosthetic valve which had been placed five years before. Anticoagulation was changed from acenocumarol to heparin (1000 units/hour). The initial post-operative period was uneventful, but on the second postoperative day the patient experienced a progressive deterioration of consciousness: she was able to localize to pain but no eye or verbal response could be elicited after painful stimuli. She had to be re-intubated and mechanical ventilation was re-initiated. A cranial CT scan showed a left side posterior fossa hematoma measuring 4x3x3 cm (Fig. 1). The lesion was rounded and superficial, and in plain CT scan it appeared mainly hyperdense but exhibited a well demarcated hypodense superior area. The fourth ventricle was collapsed and displaced anteriorly. The patient was on heparin 20 IU/kg/h, PTT was 110 seconds (control = 37 sec.) and platelet count was 69,000/mm³. Heparin was
discontinued and mannitol and fresh frozen plasma were administered. Two hours later the PTT was 49 seconds and the patient was taken to the operating room with the diagnosis of intracerebellar hemorrhage.

A left suboccipital craniectomy was performed in the semi-sitting position. The dura appeared dark blue and it was under moderate pressure. After it was incised, a fresh subdural blood clot was aspirated. Underneath, the cerebelum appeared deformed and ventrally displaced. The surgical cavity was copiously irrigated with normal saline and no bleeding point or damage of the cerebellar cortex were observed. A control CT scan performed 6 hours later showed the complete disappearance of the subdural hematoma. One month later the patient was neurologically asymptomatic.

Discussion

Acute subdural hematomas of the posterior fossa in adults without a history of trauma are very infrequent. Less than 20 cases of spontaneous posterior fossa acute subdural hematomas have been reported up to date and they are usually related to the use of anticoagulation. Clinically they present with a progressive deterioration of consciousness that rapidly alerts the physician. In the past, diagnosis was made by ventriculography or angiography and some patients underwent operation on clinical grounds alone. With the aid of cranial CT scan diagnosis became much easier. However, it is still sometimes difficult to detect a subdural hematoma in the posterior fossa because: 1) the existence of bone artefact; 2) the occurrence of isodense clots, and 3) the possibility of atypical forms of presentation leading to the wrong diagnosis of intracerebellar hemorrhage. In this respect, it is useful to remind that hypertensive intracerebellar hemorrhage arises from the deep cerebellar nuclei that receive the greatest arterial supply.

Some recent studies have focussed on surgical indications in cases of cerebellar hemorrhage. D’Avella et al reported a clinical study on 81 cases of traumatic intracerebellar hemorrhage and pointed out that, in general terms, a conservative approach can be considered in noncomatose patients with intracerebellar clots measuring less than or equal to 3 cm, excepting the associated with other extradural or subdural posterior fossa focal lesions. On the other hand, surgery was recommended for all patients with clots larger than 3 cm. Kirolls et al developed a treatment protocol which was prospectively applied for the management of 50 patients with spontaneous cerebellar hematomas. The degree of the fourth ventricle compression and the presenting Glasgow Coma Scale score were considered the main factors conditioning the protocol schedule. Management of acute posterior fossa subdural hematomas follows these same general criteria. Surgical evacuation is generally indicated on an emergency basis and anticoagulation must be urgently reversed and discontinued for at least three days after surgery. Fresh frozen plasma and platelet transfusion may be required. Occasionally, conservative management may be indicated in patients with minimal clinical deterioration; spontaneous transformation of the acute lesion into an asymptomatic chronic subdural haematoma has been previously reported.

References


Correspondencia postal: P. Miranda. Servicio de Neurocirugía. Hospital 12 de Octubre Avda. de Córdoba s/n. 28041 Madrid