Posterior fossa subdural haematoma associated with anticoagulant therapy

T. CAPISTRANT,¹ R. GOLDBERG, H. SHIBASAKI, AND D. CASTLE

From the Department of Neurology, St. Paul-Ramsey Hospital, St. Paul, Minnesota, U.S.A.

SUMMARY This is a report of spontaneous posterior fossa subdural haematoma associated with anticoagulation therapy. The possibility of posterior fossa lesions related to spontaneous haemorrhage is suggested by the combination of severe headache and increasing disturbance of consciousness associated with signs of brain-stem decompensation. A thorough neurological evaluation including appropriate contrast studies will help rule out a supratentorial lesion. This is a neurological emergency which can be successfully treated by early detection and prompt surgical decompression. This is the second reported case of spontaneous subdural haematoma of the posterior fossa occurring during anticoagulant therapy.

The physician who prescribes anticoagulant drugs strives for a point of balance in bleeding and clotting mechanisms. A significant decrease in the efficiency of these mechanisms must be achieved if therapy is to serve its purpose, but this must not proceed to the point of inducing haemorrhages.

This paper is concerned with a patient who developed a spontaneous subdural haematoma while receiving anticoagulant therapy. We are reporting this case because the lesion occurred in the posterior fossa, which is an uncommon site of subdural haematoma (Ciarla, 1913; Munro, 1934; McKissock, Richardson, and Bloom, 1960; Ciembroniewicz, 1965; Wright, 1966). A previous case of posterior fossa subdural haematoma complicating anticoagulant therapy was reported by Zenteno-Alanis, Corvera, and Mateos (1968). Diagnosis was difficult and evolved from a combination of clinical observations and emergency contrast studies. Ultimately diagnosis was confirmed by neurosurgical intervention.

CASE HISTORY

A 50 year old Mexican male laundry worker was admitted to St. Paul-Ramsey Hospital on 24 July 1969. He complained of sudden chest pain and blood-tinged sputum. The patient had noted swelling in the left leg and ankle the day before admission. He contracted and was treated for tuberculosis at 36 years of age. There was no history of head injury or alcoholism. The family and social histories were non-contributory. Physical examination was normal except for tenderness and swelling of

the left ankle and stasis skin changes in the lower left leg. There were no neurological abnormalities. Laboratory examination was within normal limits except for increased serum GOT to 230 (normal 10 to 40). Radiograph of the chest and electrocardiogram were within normal limits. A diagnosis of thrombophlebitis of the left leg and probable pulmonary embolism was made.

Anticoagulant treatment was started on 25 July with intravenous administration of 6,000 u. heparin every four hours. On 28 July prothrombin time was 25.9 seconds in the patient and 11.8 seconds in the control. On 29 July, the dosage of 6,000 u. was decreased to 5,000 u. heparin every four hours. Coumadin was initiated with 15 mg on 2 August and 10 mg on 3 August.

On the morning of 2 August, the patient complained of a severe, bitemporal, throbbing headache. He vomited once that afternoon. A complete neurological examination at that time was normal. The headache persisted. On 3 August, he seemed to fall asleep after lunch but was hard to arouse. He became progressively unresponsive and developed a high fever. At 2 a.m. on 4 August, the patient responded only to deep pain with slight jerking movements of both feet. The pulse rate was 76 per minute and regular; blood pressure was 150/56 mmHg; and the body temperature was 103° F (39.4°C). Breathing was normal. The optic discs appeared normal, but venous pulsations were absent. The left pupil was 3 mm and the right 2 mm in diameter, and both were non-reactive to light stimulation. The eyes were slightly divergent, and oculocephalic and ciliospinal reflexes were absent. Corneal reflexes were present. Muscle tone on the right was slightly reduced. There were no involuntary movements. Deep tendon reflexes and abdominal reflexes were absent. There were marked extensor toe signs bilaterally. Complete blood count, blood urea nitrogen, blood glucose, and serum electrolytes were within normal limits. Prothrombin time was 14.8 seconds with a control

¹Reprint requests to above address.

of 11.5 seconds; partial thromboplastin time was 38 seconds with a control of 31 seconds.

At this time a subarachnoid or intracerebral haemorrhage was suspected. Radiographs of the skull were normal. An echoencephalogram indicated no shift of midline structures. Heparin and coumadin were discontinued. The patient was given Decadron, 2 mg intravenously, followed by 4 mg intramuscularly.

At 8 a.m. on 4 August, the patient responded only with decerebrate posturing on supraorbital compression. Corneal and caloric responses were now absent. The left pupil continued to be slightly larger, and there was a mild decrease of tone on the right side. A left carotid angiogram (Fig. 1) was performed at 10 a.m. The lateral view showed moderate unrolling of the pericallosal arteries suggesting ventricular enlargement. There was no focal abnormality and no midline shift of the anterior cerebral vessels or the vein of Galen. The tentative diagnoses were intraventricular haemorrhage or haemorrhagic mass lesion in the posterior fossa. Air ventriculography was performed through bilateral occipital burr holes. The ventricular fluid was clear and colourless, ruling out intraventricular haemorrhage. As shown in Figs. 2a and 2b, there was moderate enlargement of the lateral ventricles and the third ventricle. The cerebral aqueduct and the fourth ventricle were not visualized.

An occipital craniotomy was performed and revealed that the dura mater was bulging posteriorly and had a brownish discolouration. Approximately 15 ml. of semiclotting blood was removed from the subdural space. The right cerebellar hemisphere was compressed forward, but there was no intracerebellar haemorrhage. The surface of the cerebellar hemispheres appeared normal and with adequate circulation. The dura mater was sutured primarily.

FIG. 1. Left percutaneous carotid angiogram showing unrolling of the pericallosal arteries suggesting ventricular enlargement.

Three days after surgery, the patient opened his eyes and moved his hands and legs to verbal commands. A right-sided extensor toe sign was present. One week after surgery the patient was alert and his speech was appropriate. At this time he had a fever, and *Pseudomonas aeruginosa* was cultured from the sputum. *Pseudomonas pneumonia* was the first of a series of complications which eventually resulted in the patient's death. The pulmonary infection was followed by *Pseudomonas meningitis*. The patient was treated with intrathecal polymyxin B and parenteral colymycin. Activation of pulmonary tuberculosis secondary to the general debilitation and high dosages of steroids was suspected. There was no change in neurological status during this time, except for somnolence associated with periods of high fever. On 2 September 1969, the 29th postoperative day, the patient suddenly expired. Permission for necropsy could not be obtained.

COMMENT

A 50 year old man developed headache and progressive coma one week after being given intravenous heparin for thrombophlebitis and probable pulmonary embolism. Although the neurological examination revealed some lateralization with anisocoria and diminished tone in the right extremities, neither echoencephalography nor left carotid angiography showed any evidence of a supratentorial mass lesion. Enlarged lateral ventricles were suggested by the angiographic appearance of the pericallosal arteries.

This could be secondary to (1) intraventricular haemorrhage, or (2) compression of the brain-stem by a posterior fossa mass. The first possibility was ruled out by the clear appearance of the cerebrospinal fluid obtained during air ventriculography. A posterior fossa subdural haematoma was confirmed by occipital craniotomy. The patient initially made a good recovery, but a series of infectious and metabolic complications resulted in his death on the 29th postoperative day.

The diagnosis of a haemorrhagic lesion in the posterior fossa is often difficult. The paucity of neurological localizing signs and the relative lack of specific diagnostic assistance provided by EEG, radiographs of the skull, echoencephalography, brain scan, and carotid angiography all contribute to the problem. A most important asset in making the diagnosis of a posterior fossa haemorrhage appears to be simply keeping the possibility of its existence in mind. The rapidity with which unconsciousness deepens necessitates swift action to avoid death or irreparable brain dysfunction.

Although an increased haemorrhagic tendency during anticoagulation is a well-known phenomenon, there still exists a controversy about the association of anticoagulation and central nervous system haemorrhage. Coogan (1965) followed 142

FIG. 2. Air ventriculogram showing lateral (left) and third ventricle enlargement without visualization of the cerebral aqueduct.

anticoagulated patients for 16 years and found only four who developed cerebral haemorrhage and doubted the causal effect of anticoagulation. Wells and Urrea (1960) discovered 14 primary intracranial haemorrhages including five subdural haematomas among over 600 patients receiving anticoagulants at the New York Hospital. Although they concluded that the overall mortality rate from intracranial haemorrhage among anticoagulated patients was not significantly higher than among untreated patients, they remarked on the relatively high incidence of subdural haemorrhage in the treated group. Wiener and Nathanson (1962) reviewed the histories of 50 consecutive patients with subdural haematoma at the New York Mount Sinai Hospital and found that 12% occurred in patients receiving anticoagulants. They concluded that this complication may occur more commonly than is reported in the literature. Lepoire, Montant, Renard, and Duplay (1964) reported two cases with subdural haematomas during anticoagulation in 1964 and collected 22 cases from the literature. They reported the relatively high mortality rate of 40% among these cases. It is noteworthy that the prothrombin

time of anticoagulated patients is not necessarily excessively elevated at the time of intracranial haemorrhage (Wells and Urrea, 1960; Wiener and Nathanson, 1962). Several authors propose that minor head trauma, unnoticed by the patient, may be the precipitating cause for subdural haemorrhage among patients on anticoagulants (Nathanson, Cravioto, and Cohen, 1958). A frequent precipitating factor for intracerebral haemorrhage in anticoagulated patients seems to be hypertension, although its relationship to patients developing subdural haematomas is not clear (Barron and Fergusson, 1959; Dooley and Perlmutter, 1964; Wright, 1966).

When confronted with patients on anticoagulant therapy who develop signs of intracranial disease, a spontaneous intracranial haemorrhage should be considered. Furthermore, the present case suggests that whenever a patient on anticoagulant therapy suddenly develops a severe headache and an increasing disturbance of consciousness without neurological focal deficits, the possibility of a haemorrhagic lesion in the posterior fossa should be kept in mind.

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