

Tension Pneumocephalus and Acute Subarachnoid Haemorrhage Following Treatment For Chronic Subdural Haematoma: Case Report

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Abstract : This is a case of tension pneumocephalus with additional subarachnoid haemorrhage, occurring after chronic subdural haematoma. Possible mechanisms as causative agents discussed.

Key Words : Chronic subdural haematoma, Pneumocephalus, Subarachnoid haemorrhage.

INTRODUCTION

The reason for the poor post operative prognosis in chronic subdural haematomas can be listed as follows: Insufficient brain reexpansion, Tension pneumocephalus, recurrence of haematoma, intracerebral haematoma (2,4). There are reported cases of spontaneous, subarachnoid haemorrhage occurring with chronic subdural haematoma in the literature, yet there is no reported case of acute subarachnoid haemorrhage developing following surgery for subdural haematoma without any obvious cause.

In this paper, we report a case of tension pneumocephalus and acute subarachnoid haemorrhage following bilateral chronic subdural haematoma accompanied by neurological deterioration verified Computed tomographs (CT).

CASE REPORT

This 76-year old man, was admitted to hospital two months after a minor head trauma caused by falling from a high place. His neurological condition

deteriorated and he presented with progressive confusion, right hemiplegia and left hemiparesia.

CT, showed bilateral chronic subdural haematoma, slightly hyperdense on the left. There was ventricular compression bilaterally, and shift of the midline structures to the right. (Fig. 1).

These subdural collections were evacuated through multiple burr holes and drained by closed system drainage. The patient remained in a comatose state post operatively and a CT scan performed 4 hours after the operation, revealed a tension pneumocephalus bilaterally, being more obvious on the left (Fig. 2). Under local anaesthesia, bilateral air drainage was accomplished. During the post operative period, the patient deteriorated neurologically. A CT scan was performed 12 hours after the first operation and revealed a massive subarachnoid haemorrhage and also free air in the subarachnoid spaces as well as neural parenchyma (Fig. 3).

The patient died in a comatose state the second post operative day.

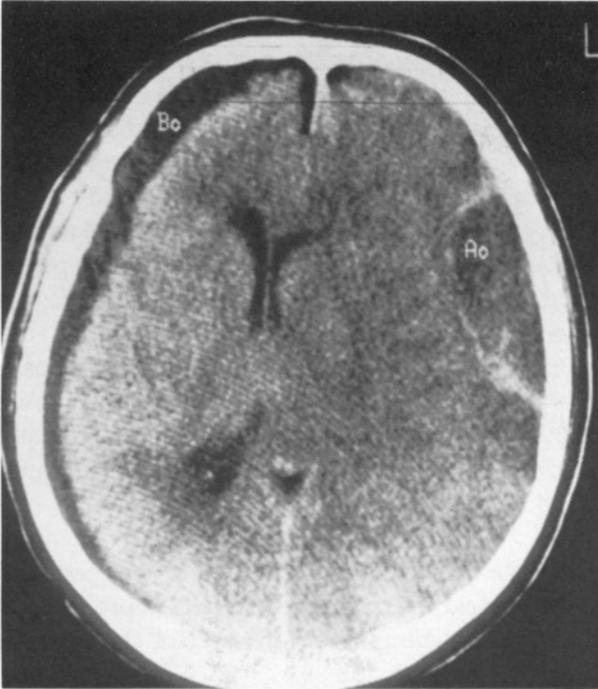


Fig. 1 : CT revealed ventricular compression bilaterally, and shift of the midline structures to the right.

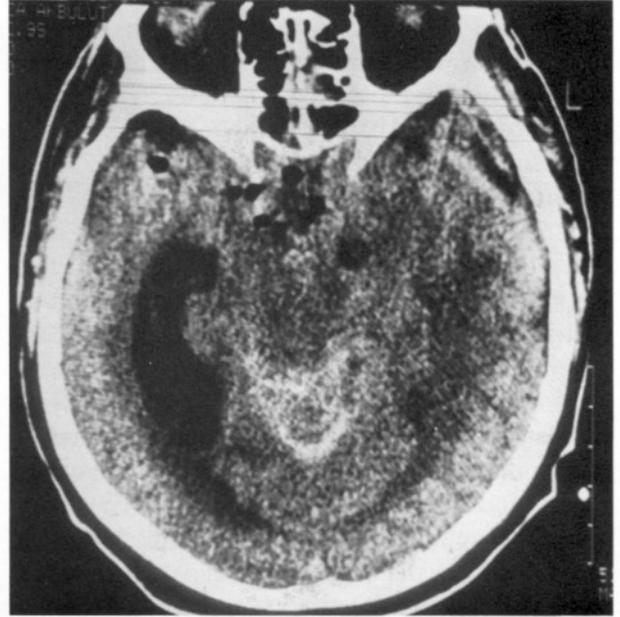


Fig. 3 : CT scan revealed a massive subarachnoid haemorrhage and also free air in the subarachnoid spaces as well as neurolept parenchyma.

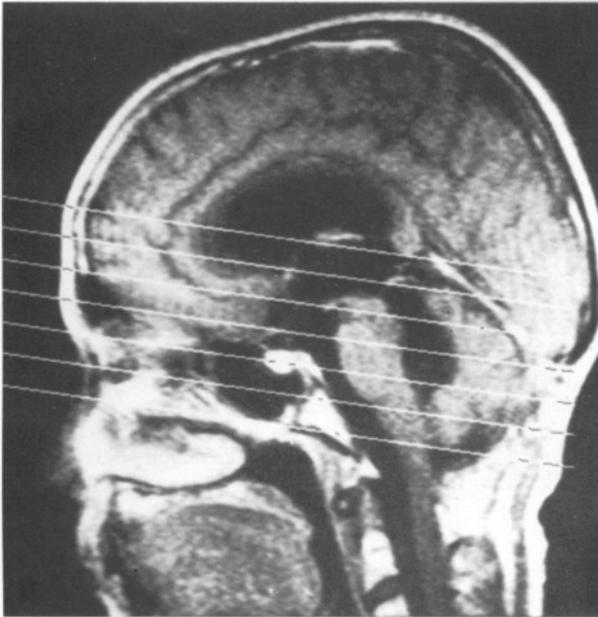


Fig. 2 : CT revealed a tension pneumocephalus bilaterally, being more obvious on the left.

DISCUSSION

There are different types of complication reported in the literature, occurring after surgical evacuation of chronic subdural haemorrhage. These include insufficient brain reexpansion, recurrence of haematoma and pneumocephalus but we have found no report so far, of massive subarachnoid haemorrhage as a complication (2,4).

It has been reported that late type subdural haematoma could be looked upon as proof of brain injury, being associated with subarachnoid haemorrhage (6). D'avella et. al. reported two cases of intracerebral haematoma, following evacuation of chronic subdural haematoma. One of which was associated with bifrontal pneumocephalus (2). In their paper, D'avella et. al. state that the rapid decompression of chronic subdural haematoma could be the possible cause for intraparenchymal haemorrhage and suggest rather slow decompression, with controlled reexpansion. Kotwica et. al. in 1985 reported 6 cases of chronic subdural haematoma, presenting with clinical findings of acute subarachnoid haemorrhage (5). These patients were all in the 2nd, 3rd decade and all clinical signs as well as lumbar puncture revealed SAH. Four vessel cerebral arteriograms

revealed no vascular abnormalities, but demonstrated unilateral extracerebral avascular areas, characteristic of subdural haematoma. Through a burr hole, the haematomas were evacuated and focal neurological symptoms returned to normal (5). Ktowica et. al. found three possible mechanisms responsible for SAH in their cases: 1. SAH was a result of vascular abnormalities not revealed on the arteriograms, 2. SAH was a manifestation of 'Spat Apoplexie' and the presence of haematoma was coincidental, 3. SAH was due to chronic subdural haematoma. The present authors of this paper discussed the first two possibilities and suggested that SAH was related rather to the chronic subdural haematoma and showed several possible mechanisms to explain this, but could not make a certain decision about which of these mechanisms was responsible for SAH in their case. a. The distortion and displacement of vessels, b. Pressure of the haematoma on the cerebrum causing degeneration in the wall of a vessel, leading to rupture and haemorrhage from proliferation of vessels in or adjacent to the capsule, c. A sudden increase of intracapsular pressure, leading to rupture of the arachnoidea with bleeding from the haematoma into the subarachnoid space.

Different authors have previously reported the possibilities of haemorrhage in the subdural space entering the subarachnoid space through rupture of the arachnoidea (7).

Our case was different from the above reported cases in that the patient was in the seventh decade, the lesion was bilateral, and SAH occurred after surgery. Following surgery for bilateral subdural haematoma, the patient deteriorated neurologically and CT taken revealed bilateral tension pneumocephalus. A second operation was then undertaken. The patient's comatose state was progressive and CT revealed massive subarachnoid haemorrhage and free air in the subarachnoid space as well as in parenchyma.

Eventhough no arteriograms were undertaken in the preoperative period to suggest the absence of vascular abnormalities, we believe that SAH occurred because of arachnoid membrane rupture, thus the haematoma entered the subarachnoid space. There were no signs or findings of 'spat apoplexie' with our case as proved by CT taken at the time of neurological deterioration (1,3), revealing SAH as well as free air in the subarachnoid space and in the parenchyma. It is possible that because of peroperative laceration and rupture of the arachnoidea the haemorrhage as well as free air in the subdural space could pass into the subarachnoid space. As far as we know, this is the first report to prove radiologically arachnoid membrane rupture in chronic subdural haematoma.

In the light of this knowledge, we suggest that subarachnoid haemorrhage can be a neurologically deteriorating complication in cases of chronic subdural haematoma.

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