Primary Intracerebral Haematoma Evacuation: A Case Report

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Authors' contributions

This work was carried out in collaboration between both authors. Author TV designed the study. Author GB collected the literature and was the principal treating physician. Both authors read and approved the final manuscript.

Case Study

ABSTRACT

Spontaneous intracerebral hemorrhage is one of the most devastating types of stroke, leading to disability and high mortality rate. Besides blood pressure reduction and intensive medical and surgical treatment, immediate coagulopathy reversal is vital. On the other hand, the haemostatic disturbances may contribute to improve the recovery. We describe the evacuation of intracerebral hemorrhage with the insertion of external ventricular drainage in a patient suffering from deep hypertensive intracerebral haemorrhage and haematocephalus.

1. INTRODUCTION

Spontaneous Intracerebral Haematoma (ICH) is a frequent and well documented pathology, accounting for accounts for 20% of all stroke admissions [1-3]. Although advances in intensive care and haematoma management have improved the outcomes for patients with ICH, morbidity and mortality rate are still high. The 30-day mortality rate may reach as high as 50% and

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after one year, 75% of all patients have died or are severely disabled [2,4,5]. Rapid recognition and diagnosis of ICH as well as identification of early prognostic factors are therefore vital [4].

According to the aetiology, the ICH may be classified as primary or secondary [6]. The majority of haematomas, about 80%, are primary and the main risk factor is long-standing and uncontrolled arterial hypertension, causing comes from the spontaneous rupture of small vessels. These are usually located deep in the brain, in the internal capsule and the basal ganglia [2,3,6,7]. On the other hand, about 20% are secondary ICHs, more frequently located in cerebral lobes, cerebellum and pons [4,6]. They are associated with anticoagulant therapy or coagulation disorders, tumours and vascular abnormalities [8-10]. Often, intraventricular haemorrhage is associated with ICH and is considered as a poor prognostic factor due to Cerebrospinal Fluid (CSF) obstruction and the mass effect of haematoma, leading to hydrocephalus and raised intracranial pressure (ICP) [11].

We describe the evacuation of primary ICH with the insertion of external ventricular drainage in a patient with hypertensive deep intracerebral haemorrhage with haematocephalus.

2. PRESENTATION OF CASE

A 64-year old lady was admitted to the emergency department due to headache, nausea and progressive left sided arm and leg weakness. The symptoms started a few hours' before the admission and deteriorated gradually. A short episode of consciousness loss at home was reported by the relatives but no seizures were observed. No clear head trauma was recognized. In the past medical history, arterial hypertension and advanced hepatic cirrhosis of ethylic genesis were documented.

Neurological examination found slight disorientation in time and place and slowness. The Glasgow Coma Scale (GCS) was rated at 13. Testing of cranial nerves was normal. No meningeal signs were observed. There was left sided haemiparesis, more pronounced in the lower extremity. Here, the plantar response was extensor. No tremor was observed. Muscle tone was normal with no noticeable difference of reflexes in the upper and lower extremities. During Romberg testing, the lady was swaying with the tendency of falling backwards. Walking was not possible.

The laboratory findings revealed completely deranged coagulation and aggregation values. The International Normalised Ratio (INR) and Protrombine Time (PT) were lowered, 2.08 and 0.3 respectively. The platelet count was reduced to 57 and the platelet function tests were entirely disturbed. Computed Tomography (CT) showed an ICH with haematocephalus and developing brain shift due to cerebral oedema (Fig. 1). The CT angiography was negative, thus classifying the haematoma as a primary one.

The consciousness started to decline rapidly after imaging with GCS rated at 7. Urgent intubation was necessary. Immediately, coagulopathy correction started. Injections of vitamin K, protrombin complex, fresh frozen plasma, recombinant coagulation factor VIIa and platelet plasma were applied. Control haemostasis test were, however, even worse. This prevented any surgical method of haematoma treatment. Antioedematous therapy started. Only after 14 hours, the haemostasis values were acceptable, although far from ideal, for the EVD and ICP probe insertion. Liquefied blood and CSF, which were under pressure, were evacuated. The ICP values, initially 35 mmHg, have fallen to 10 mmHg and remained normal during the course of treatment. The control CT scan showed evacuated haematoma and normal ventricles with residual haematocephalus (Fig. 2). According to the improvement of the haematoma, the sedation was gradually discontinued after a week. In clinical status, a moderate left sided haemiparesis persisted, the GCS was rated at 12. The lady was transferred to neurosurgery ward where she was recovering slowly. Unfortunately, after 23 days of treatment on the ward she died due to complications of hepatic cirrhosis and pneumonia.

3. DISCUSSION

Despite the frequency of ICHs, there is still ongoing debate about the optimal patient care. The treatment strategies include medical, surgical and combined treatment, including emergent reduction of blood pressure and coagulopathy reversal, management of cerebral oedema, as well as surgical interventions [4,10,12-14]. The latter include External Ventricular Drainage (EVD) and hematoma evacuation [1,6]. Patients with anticoagulation therapy and intracranial haemorrhage require immediate coagulopathy correction in order to prevent haemorrhage expansion, limit tissue damage and impede surgical intervention [12,15,16].
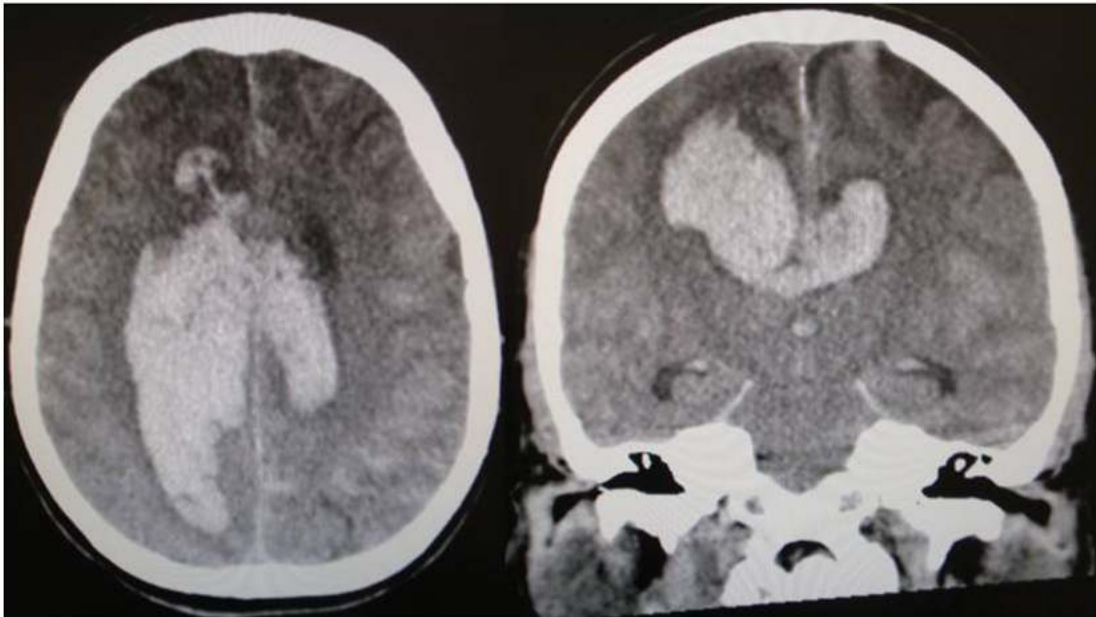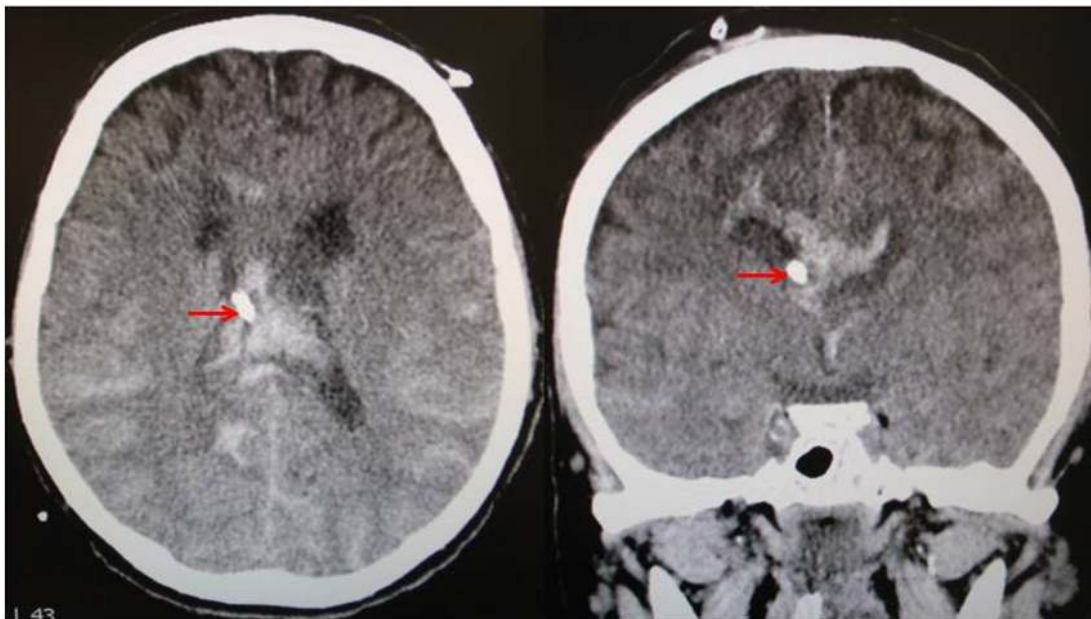
Fig. 1. An unenhanced CT scan showing ICH with haematocephalus and developing brain shift due to cerebral oedema

Fig. 2. The control CT scan showing evacuated ICH and normal ventricles with residual haematocephalus
EVD is in place (arrow)

The outcome reports of ICH treatment may differ among studies. Numerous factors influence the treatment outcome, the most important being the site of bleeding, age of patients and management of haematoma [9,13,17,18]. ICHs exhibit a heterogeneous clinical profile, depending on the bleeding site and the consequences vary accordingly. In intraventricular bleeding, for instance, the mortality approaches 44% [17]. Additionally, the

final recovery of younger patients is more favourable to older. Especially patients older than 85 years demonstrate worse treatment outcome due to higher complications-related in-hospital mortality and more severe neurological deficit at the discharge than younger [18]. While some claim better outcome after surgical evacuation, other studies found no difference between surgical and non-surgical group [9,13,19]. Secondary brain injury is smaller after surgical removal of ICH due to less oedema, less blood leftover, which needs to be resorbed in the brain, less scarring in the ventricles and therefore shorter intensive care treatment [6,8]. Haematoma in cerebral hemisphere differs from those with intraventricular location, as the latter may cause hydrocephalus and the need for EVD insertion. In the long term, many of these patients will require a ventriculoperitoneal shunt due to disturbances in CSF flow. Surgical strategies differ: Some reports describe minimally invasive haematoma evacuations, whereas others are in favour of craniotomies [2,14,16,20]. To our knowledge, no report about ICH successfully evacuated through EVD was found in the literature.

On our opinion, surgery has an advantage over conservative treatment and this strategy was adopted also in our patent, yet it must be used cautiously. As a result of severe liver failure, an immediate surgical evacuation of the haematoma was not possible due to coagulation and aggregation disturbances, nor was EVD and ICP probe insertion. It was necessary to wait until the haemostasis was corrected optimally. In patient with end liver failure, however; this is not an easy task to achieve. Immediate surgery was too risky, exposing the patient to even greater intracerebral bleeding. The EVD insertion and invasive ICP monitoring are contraindicated in disturbed haemostasis as well [1,4,6,15]. With continuous replacement of low numbers of non-functional platelets and coagulation factors in the form of fresh frozen plasma, protrombin complex and factor VII, the haemostasis finally improved, although the optimal values for surgery were not possible to attain. The EVD was first inserted and then ICP probe followed according to standard surgical method. Although risky, owing to possible new haematoma formation during the insertion procedure, this was the only method to lower the elevated ICP and remove the haematoma, thus saving the patient's live. There was some luck on surgical site. Due to disturbed haemostasis, the ICH remained liquefied and the clot did not form. Even though immediate

coagulopathy reversal is mandatory and was done so in or patient, normal values were not possible to reach due to liver failure, which on the other hand saved the patient with the prevention of clot formation. This enabled the liquefied blood to exit the intracranial space through the EVD and also to relieve the ventricles, this evacuating the ICH and reducing the haematocephalus, which was confirmed on the control CT and was evident thorough the continuous ICP monitoring.

Although the arterial hypertension and amyloid angiopathy are principal causes for ICH formation, the haemorrhage is accelerated in the presence of bleeding diathesis of various aetiologies [4,13]. Anticoagulant therapy, trauma and alcoholism are supplementary predisposing factors for ICH development. In this group of patients, risk is haematoma development and recurrences are highest [8,9,21]. Our lady had a clear history of arterial hypertension along with clinical signs of hepatic cirrhosis and therefore, the haemostatic mechanisms were ineffective. The bleeding started deep in the right hemisphere, near the wall of the lateral ventricle, rupturing it and penetrating into the cavities. It was accelerated by disrupted coagulation mechanisms; otherwise it may have been self-limiting or not as extensive as to cause clinically significant haematoma in case the haemostasis was normal. As blood in ICHs accumulates quickly and extensively, it damages the neurones. Many haematomas may increase in size causing additional damage and more extensive secondary brain injury with concomitant complications. The multicenter prospective randomized controlled trials have shown that slowing of the hematoma growth, removal of intacerebral blood, clearance of intraventricular hemorrhage and optimization of cerebral perfusion pressure may form the most important therapeutic goals to ameliorate secondary neurological damage, decrease mortality, and improve functional outcomes after ICH [1,4,5,15]. Immediate diagnosis with coagulopathy reversal, reduction of blood pressure and surrounding oedema may help to achieve hemodynamic stability, limit the hematoma expansion and prepare the patient for potential surgical intervention [6,8-10].

4. CONCLUSIONS

Spontaneous intracerebral haematoma is a frequent pathology, although treatment is not always clear and outcomes are usually poor. It

primary depends o the previous condition of the patient, location and extent of the haemorrhage and concomitant anticoagulation. Especially with the newer anticoagulant agents, the treatment is aggravated, as there is no effective antidote. In these cases, minimally invasive evacuation of haematoma is warranted.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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