SURGICAL APPROACH IN PRIMARY INTRACEREBRAL LOBAR HEMORRHAGE WITH PROGRESSIVE NEUROLOGIC DEFICIT

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Abstract: *Cerebral hemorrhage is the third most frequent cause of stroke, and it's most important risk factor is hypertension, followed by aneurysms and arteriovenous malformations. We present the case of a 63 year old male patient with a known medical history of intracerebral hemorrhage following a ruptured arteriovenous malformation, admitted for a sudden onset of a persistent confusional state. CT scans showed left frontal intraparenchymal hematoma. The initial therapeutic approach was conservatory, by administrating antihypertensive, diuretic and hemostatic agents, while simultaneously decreasing intracranial pressure. Forty-eight hours after admission, we found motor deficit of the right limbs and non-fluent aphasia, which justified immediate surgical removal of the blood clot. Following surgery, the patient's general and neurological status was noticeably improved. After extensive differential diagnosis, the current affliction was labelled as primary intracerebral "spontaneous" nhemorrhage, due to hypertension.*

INTRODUCTION

Of all the cerebrovascular diseases, brain hemorrhage has the most omninous and dramatic clinical picture. It is the third most frequent cause of stroke, being outranked only by atherothrombotic and embolic infarction.(1)

Primary intracerebral hemorrhage (ICH) refers to bleeding into the parenchyma of the brain as a result of spontaneous blood vessel rupture in the absence of trauma or surgery, and constitutes 10-20% of all strokes. Hemorrhagic strokes are more serious than ischaemic storkes and have a higher mortality.

The most important ICH risk factor is hypertension, followed by aneurysms and arteriovenous malformations.(2) Chronic hypertension is associated with bleeding into the putamen, thalamus, pons and cerebellum, while lobar hemorrhages have many causes.(1)

Lobar intracerebral hemorrhage accounts for 33% to 42% of all ICH. Lobar ICH refers to hemorrhages located in cortical–subcortical areas and follows a "lobar" pattern across one or less often multiple lobes of the brain.(3)

Many studies have reported a high rate of hypertension among cases of ICH. Significant independent risk factors for lobar intracranial hemorrhage included a first-degree relative with ICH, previous stroke, frequent alcohol use, as well as genetic factors.

Recurrent lobar ICH is a hallmark of cerebral amyloid angiopathy.(4)

As regarding the clinical picture, headache, acute hypertension and vomiting along side with a focal neurological deficit are the cardinal features of brain hemorrhage and serve most dependably to distinguish hemorrhage from ischemic stroke.(1)

The decision of surgical management of ICH is individualised based on the patient's age and neurologic condition, size and location of the hematoma.(5)

CASE REPORT

We bring forward the case of a 63 year old male patient, a non-smoker, with no history of alcohol intake, admitted to hospital through the emergency ward, for the sudden onset of a persistent confusional state during the previous 24 hours.

The patient had a known medical history of intracerebral hemorrhage following a ruptured arteriovenous malformation which needed surgical management nine years prior, but had no other known comorbidities, and was under no chronic medication.

Clinical examination on arrival revealed a confussional state, but no motor or sensory deficit, or other neurologic focal signs. Blood pressure was measured to be 180/100 mmHg.

Cranial CT was immediately performed in the emergency unit, which showed a left frontal intraparenchymal hematoma, with moderate surrounding edema and minimal ventricular seepage, as well as a right temporoparietal postoperatory sequelae (figure no. 1).

The initial therapeutic approach was conservatory, and consisted in administrating antihypertensive, diuretic and hemostatic agents, while simultaneously decreasing intracranial pressure.

The following day after admition, cerebral angio-CT was performed, which did not reveal any arteriovenous malformations and by comparison, no enlargement of the left frontal hematoma, indicating lack of continuous intracerebral bleeding. Therefore, we chose to continue the conservatory medical treatment (figure no. 2).

Hypertension, sinus arriyhmia and ischemic cardiomiopathy were diagnosed during hospitalisation, after a complete cardiological examination, and following the cardiologist recommendations, proper addition of indicated drugs was done for this coexisting conditions.

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Figure no. 1. Cranial unenhanced CT scan - Left frontal intraparenchymal hematoma, with moderate surrounding edema; right temporoparietal postoperatory sequelae

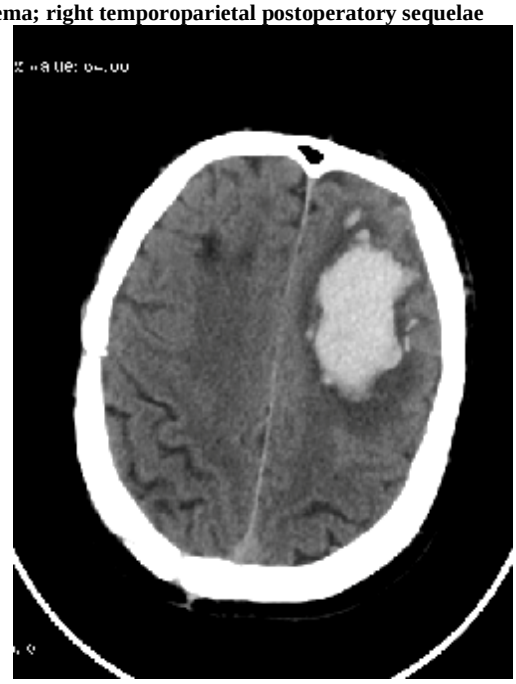

Figure no. 2. Cerebral Angio-CT scan - Lack of arteriovenous malformations

On a clinical reexamination 48 hours after admission, we found a newly developed motor deficit of the right limbs and non-fluent aphasia, which slowly but steadily progressed. Following this turn of events, we opted for immediate surgical removal of the blood clot.

This approach was proven to be optimal, since during postoperative recovery, the patient's general and neurological status were rapidly and noticeably improved, with nearly full remission of the right limbs motor deficit.

Gradually, the initial non-fluent aphasia took the shape of a minor language impairment. This status continued to improve without any complications, up to the discharge from hospital.

DISCUSSIONS

Nontraumatic bleeding into the brain parenchyma results from rupture of small penetrating arteries. In deep hematomas, this has been attributed to degenerative changes in the vessel wall associated with advancing age, chronic HTN, diabetes, and other vascular risk factors. Charcot-Bouchard microaneurysms and lipohyalinosis of small arterioles have been suggested as mechanisms.(3)

In this particular case, a sudden neurological deterioration in a patient with a known medical history of non-traumatic cerebral hemorrhage raised the suspicion of renewed bleeding. This assumption was immediately confirmed via CT scan, so that clinical diagnosis was fairly not problematic.

However, the cause of the bleeding was at question.

Lobar hemorrhage can have various causes, unlike bleeding in the deep structures of the brain, which are tipically caused by high blood pressure.(1)

Intracranial arteriovenous malformations (AVMs) are a common cause of stroke in younger patients, and often present as intracerebral hemorrhages, associated with 10 % to 30 % mortality. Patients who present with a hemorrhage from an AVM should be initially stabilized according to acute management guidelines for ICH.(6)

Given the medical history of our patient, we first took into consideration an underlying arteriovenous malformation or a saccular aneurysm as the cause of hemorrhage, but this hypotesis was swiftly disregarded due to brain imaging - AngioCT and previous cerebral MRI. Imaging tools also excluded any hemorrhaging cerebral tumoral masses.

Intracranial hemorrhage is the most feared and lethal complication of oral anticoagulation. Both patient factors and anticoagulation intensity importantly influence the rate of anticoagulation-related intracranial hemorrhage.

As the patient was under no chronic medication, we excluded anticoagulation therapy and vasopressor drugs as causes.

Intracerebral hemorrhage (ICH) is an unusual but serious complication of bleeding disorders. ICH is believed to follow thrombocytopenia, alterations in coagulation, and vascular fragility.(7)

As laboratory tests did not revealed any coagulation abnormalities, because of the age and gender of the patient, two main causes of this hemorrhage were singled out: hypertension and cerebrovascular amyloidosis.

Cerebrovascular amyloidosis manifests itself either as progressive dementia or as recurrent lobar hemorrhage, producing acute, focal neurologic deficits.(8) This diagnosis was definitely overruled by brain biopsy, which did not show any amyloid deposits in the cerebral arteries.

Intracerebral hemorrhage may be a complication of either acute or chronic arterial hypertension. Sudden and severe hypertension overwhelms the autoreglartory response of the cerebral vasculature, while chronic hypertension leads to lipohyalinosis and microaneurysms in the small penetrating arteries.(9)

All patients with intracerebral hemorrhage are hypertensive immediately after the stroke because of a general sympathoadrenal response. In our patient's case, it is unsure if the high blood pressure levels were long standing or if they had risen suddenly and recently.

Surgery has the potential to reduce the volume of intracerebral haemorrhage and there is clinical and experimental evidence that blood clot removal might reduce nervous tissue damage, possibly by relieving local ischaemia or removal of noxious chemicals.(10)

Moderate to large lobar hematoma close to the cortical surface indicate surgical management, especially when the neurologic symptoms are due to increased intracranial pressure or mass effect from the blod clot or the surrounding edema.(5)

In our particular case, the surgical approach was considered as soon as we found motor deficit of the right limbs and non-fluent aphasia, which slowly but steadily progressed. This decision had a clear positive outcome regarding the general and neurologic status of the patient

CONCLUSIONS

Hypertensive cerebral hemorrhage serves as a model for understanding and managing other cerebral bleedings. Although there is no general consensus regarding indications for surgery, it can prove successful when the patient's age and neurologic condition, size and location of the hematoma all allow this kind of therapeutic approach.

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