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Case Report

Hemorrhagic Stroke on Vitamin K Antagonist

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Abstract

Hemorrhagic stroke may be subdivided into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Intracerebral hematomas represent 10% of serious haemorrhages under VKA. We report a case of a 43 years old patient, who had an intracranial hemorrhage one month after a heart surgery.

Literature say that the cerebral CT scan and Brain MRI make the diagnosis of intracerebral hematoma with very good sensitivity and specificity. The prognosis imposes an optimization of the treatments to act on the main determinants of the prognosis: the growth of the hematoma, to prevent secondary brain injury of systemic origin, to treat the systemic consequences of cerebral suffering, to prevent re-bleeding in the context of SAH, to identify and treat patients undergoing urgent surgery or radio-interventional procedures.

Keywords: *Hemorrhagic Stroke; Intracerebral Hemorrhage (ICH); Subarachnoid Hemorrhage (SAH)*

Introduction

Hemorrhagic stroke is associated with severe morbidity and high mortality. It may be subdivided into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Early diagnosis and treatment are essential [1]. It contributes to 10% to 20% of strokes annually [2].

Intracerebral hematomas represent 10% of serious haemorrhages under VKA. The combination of vitamin K antagonist treatment and aspirin doubles the risk of cerebral hematoma [3].

We report a case of a 43 years old patient, who had an intracranial hemorrhage one month after a heart surgery.

Observation

We report a 43 years old patient who presented with intracranial hemorrhage with a history of aortic valve replacement one month ago on acenocoumarol 4 mg per day.

The patient complained of brutal headache 6 hours before admission with sudden left hemiplegia, vomiting, without seizures.

On physical examination he had a Glasgow coma scale at 14 as he opened eyes to verbal stimuli, with left hemiplegia, pupils were equal and reactive, with a capillary glycemia of 1,4. The patient was stable with a heart rate of 85 bpm, a blood pressure of 14/8 cmHg, and eupneic it's a normal auscultation.

A CT scan was performed and showed a large compressive right frontoparietal intracranial hematoma with subfalcine herniation, intraventricular hemorrhage and minima subarachnoid hemorrhage (Figure 1).

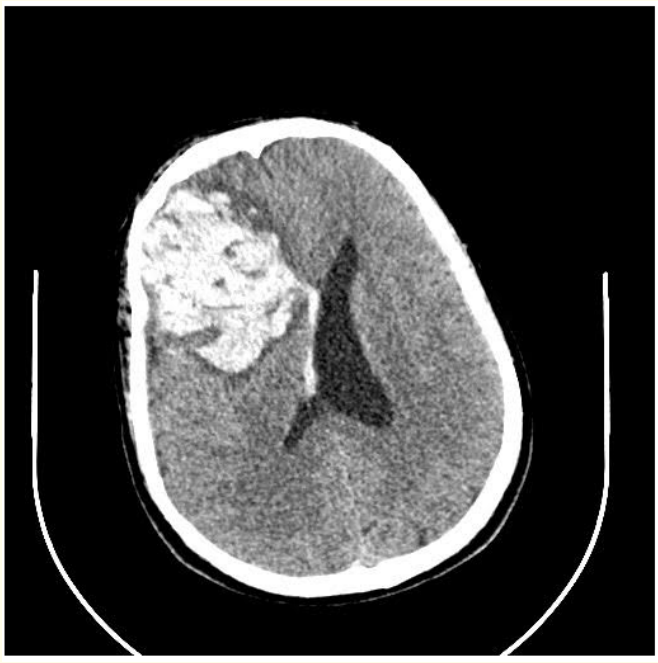

Figure 1: *CT scan showing a large compressive right frontoparietal intracranial hematoma with subfalcine herniation, intraventricular hemorrhage and minima subarachnoid hemorrhage.*

Since the patient was on antivitamin k a INR was ordered with a value of 7,46, aPTT ratio was 2,48. Hemoglobin rate was 13,3 g/dl and platelet count was 465 G/mg.

An EKG was performed showing a sinus rhythm with monomorphic ventricular extrasystoles and bigeminus, with left ventricular hypertrophy. A cardiac ultrasound showed left ventricular hypertrophy, a functional prosthetic aortic valve, preserved contractility with normal filling pressures.

The patient was taken to the operating room due to an accessible haematoma.

We took two peripheral venous lines, a right radial arterial line, then preoxygenated the patient.

Rapid sequence induction was performed Lidocaine 80 mg, Fentanyl 300 mcg, Propofol 120 mg and Rocuronium 80 mg. Arterial blood pressure remained 13/7 after intubation, with sinus rhythm on EKG. Sedation was maintained using continuous propofol and fentanyl infusion.

We performed lung protective ventilation while aiming for normocapnia. ABG showed a ph of 7,53, pCO_2 of 35, pO_2 of 153, Na^+ 141, glycemia 1,3.

The patient received 10 mg vitamins K in 20 minutes, tranexamic acid 1g in 20 minutes then 1g per 8 hours, and was transfused with 10 fresh frozen plasma. Control INR was 1,26, filling pressure remained normal without b lines on ultrasound.

Patient conditioning was completed with an right internal jugular central venous line and perfusion of Mannitol 80g, antibioprophylaxis with cephalothin 2g and potassium loading due to the arrhythmia.

The neurosurgery team performed hematoma evacuation, the surgery lasted 3 hours with a bleeding of 500 ml. Patient was transfused with two packed RBCs and had a diuresis of 2l.

Patient was transferred to the intensive care united intubated with a maintained sedation with midazolam and fentanyl. Patient received phenobarbital and depakine and his cerebral circulation was monitored using transcranial doppler. The only ultrasound window available was on the left side, it showed a pulsatility index of 1,19 and diastolic velocities of 35, with a pcO_2 of 35 and a MAP of 80 mmHg. The patient was put on enteral nutrition progressively reaching daily nutritional intakes goals, and physical therapy.

Sedation was arrested after 48 hours after depakinemia and phenolarbitemia were within the reference ranges and the patient maintained a good brain circulation on doppler. The patient presented with generalized seizures on day 3 so the patient was sedated again and clobazam was added to the regimen. A brain CT scan was performed showing without brain herniation. Anticoagulation with enoxaparin 40 mg was introduced on day 3 following the reassuring CT scan (Figure 2).

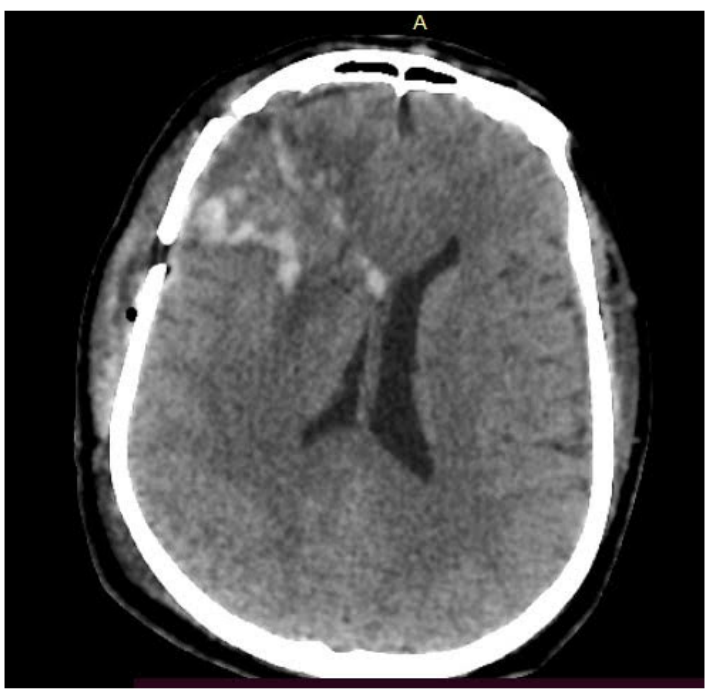

Figure 2: *CT scan showing the resorption of the hematoma with no brain herniation.*

Sedation was arrested after 24 hours, then the patient was evaluated neurologically. Early tracheostomy was performed on day 7. Daily neurological exams initially found GCS at 5 with decortication on the right and left hemiplegia on day 8. On day 12 the GCS was at 10 with a patient opening his eyes spontaneously and localizing pain. Therapeutic anticoagulation was then introduced with enoxaparin 60 mg twice daily. On day 15, the patient recovered a GCS at 15 with the left hemiplegic limbs progressively regaining strength. The patient was decanulated on day 20 and discharged on day 22 with a GCS at 15 and a muscular strength of 5/5 on the right and 4/5 on the left side, on enoxaparin 60mg twice daily and clobazam.

Discussion

Hemorrhagic stroke is due to bleeding into the brain by the rupture of a blood vessel. Hemorrhagic stroke is associated with severe morbidity and high mortality. Hemorrhagic stroke may be further subdivided into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Early diagnosis and treatment are essential [1]. Stroke, is the third major cause of morbidity and mortality in many developed countries. Hemorrhagic stroke contributes to 10% to 20% of strokes annually [1,2].

Intracerebral hematomas represent 10% of serious haemorrhages under VKA with an increase in the absolute risk of 0.6% to 2% per year. The combination of vitamin K antagonist treatment and aspirin doubles the risk of cerebral hematoma [3].

The natural evolution of hematomas in this context is responsible for a 54% increase in volume in the first three hours vs 35% for spontaneous bleeding [4] and particularly when the INR is high. This increase in volume is responsible for a very significant increase in mortality from 46 to 67% vs 30 to 40% for spontaneous intracerebral hemorrhages [5] and particularly in patients over 75 years old.

The cerebral CT scan makes the diagnosis of intracerebral hematoma with very good sensitivity and specificity. Recent hematomas are visualized in the form of hyperdensity from the first minutes of bleeding. A calculation of the volume of the hematoma has been proposed by taking the product of the widest diameter of the hematoma in cm, by the perpendicular diameter, by the thickness on which the hematoma is visible [6].

Easier access to MRIs makes first-line use relevant. Two prospective studies [7,8] have shown that MRI was as effective in detecting parenchymal hemorrhage lasting less than 6 hours as CT with 100% sensitivity. Finally, it allows a precise study of the cerebral parenchyma.

The prognosis imposes an optimization of the treatments to act on the main determinants of the prognosis: the growth of the hematoma, to prevent Secondary Brain Injury of Systemic Origin, to treat the systemic consequences of cerebral suffering, to prevent re-bleeding in the context of SAH, to identify and treat patients undergoing urgent surgery or radio-interventional procedures [9].

The prognosis of intracerebral hematomas is related to the increase in its initial volume, the risk of intracranial hypertension and the general neurological complications induced [9].

The urgent therapeutic strategy aims to normalize haemostasis urgently and quickly obtain an INR < 1.5. Every minute counts. Given the vital risk, after stopping treatment with AVK, an INR measurement will be carried out urgently in the first place, but the results of which should not delay the administration of prothrombin concentrate (CCP, expressed in IU/KG of factor IX), also called PPSB whose accelerated administration is possible in case of extreme emergency. In the absence of INR, 25 IU/kg of CCP (i.e. 1 ml/kg) will be administered. The plasma peak is obtained in 10 minutes by slow intravenous administration but can be administered in flash 10 then 10 IU/Kg at 2 minute intervals) allowing an INR < 1.5 to be obtained in all patients in 3 minutes [10].

The place of activated factor VII administration presented by the FAST study in which there was much hope to limit the size of hematomas by rapid action on hemostasis did not show any benefit on mortality [9].

The randomized STICH study [11] did not demonstrate superiority to surgery for hematomas versus conservative treatment except for the subgroup of lumbar hematomas < 1 cm deep and operated on within 12 hours as well as those of the fossa posterior > 3 cm. This data is found for patients who have ventricular compression with hydrocephalus justifying the establishment of an external ventricular shunt. Cerebellar hemorrhages benefit from emergency surgery when they are responsible for impaired consciousness or signs of brainstem compression. The STICH-2 trial, which is in progress and focused on lobar hematomas close to the surface, will make it possible to specify surgical strategies.

Conclusion

Intracerebral hematomas represent 10% of serious haemorrhages under VKA, especially if combined to aspirin. The cerebral CT scan or more better, the cerebral MRI make the diagnosis.

The prognosis imposes an optimization of the treatments to act on the main determinants of the prognosis: the growth of the hematoma, to prevent Secondary Brain Injury of Systemic Origin, to treat the systemic consequences of cerebral suffering, to prevent re-bleeding in the context of SAH, to identify and treat patients undergoing urgent surgery or radio-interventional procedures.

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