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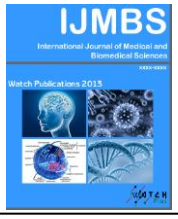

COEXISTING LEFT HEMISPHERIC INFARCT AND CHRONIC SUBDURAL HEMATOMA: A CASE REPORT

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Abstract

According to the Monro-Kellie hypothesis, the sum volumes of the brain, cerebrospinal fluid and blood inside the cranium should always be maintained constant. Any abnormality that may increase the intracranial pressure might lead to fatal brain herniation. Hence, this paper aims to present a case of chronic subdural hematoma eventually developing a left hemispheric cardioembolic infarct and how it was managed conservatively without developing the dreaded complications of increased intracranial pressure. This is a case of a 62-year old female patient, diagnosed with severe Valvular Heart Disease, Diabetes Mellitus and Toxic Goiter, who developed Chronic Subdural Hematoma from regular warfarin intake. However, she later developed a cardioembolic stroke after holding warfarin. She was managed medically with decompression, since surgical evacuation was deemed of very high risk for this patient. In a patient with multiple comorbidities presenting with concomitant chronic subdural hematoma and cardioembolic stroke, conservative management with an osmotic diuretic and corticosteroids, though still with scarce evidence in the literature, was found to be safe and may be beneficial in our patient.

Keywords: *chronic subdural hematoma, warfarin use, cardioembolic stroke, conservative management.*

INTRODUCTION

As a neurologist, one would usually encounter a Chronic Subdural Hematoma (CSDH) and a Cardioembolic Stroke (CES) separately. However, receiving a patient who has both of these conditions poses a very challenging task. This unusual presentation is rarely encountered in the literature, hence, there are only limited evidences regarding its management. In one reported case, a patient with CSDH developed an ipsilateral middle cerebral artery infarction which they attributed to compression of the brain with hematoma. The patient was successfully managed with burr hole drainage, later discharged without any neurological deficits (Wani et al., 2012). In another case series, they reported two cases of CSDH both developing occipital lobe infarction. The patients also underwent burr hole drainage, however, they both died due to herniation (Kanse et al., 2013). To our knowledge, there is no evidence yet regarding dealing with these two conditions concurrently with non-surgical interventions. Hence, this paper aims to discuss case of warfarin-induced subdural hematoma later having a left hemispheric cardioembolic infarct and how it was managed conservatively until the patient was discharged from the hospital.

CASE PRESENTATION

This patient is one of the few patients a physician would fear to encounter because of the presence of many medical comorbidities, making the management quite challenging. She is a 62-year old, married, Filipino female, diagnosed with severe Valvular Heart Disease with Atrial Fibrillation (AF) four years ago, advised surgical correction but opted medical management, hence maintained on Digoxin 0.25 milligrams and Warfarin 2.5 milligrams daily. Also, this patient was diagnosed with Diabetes Mellitus and Diffuse Toxic Goiter necessitating Metformin, Methimazole and Propranolol but these medications were recently stopped because of allegedly normal glucose and thyroid hormone levels.

The patient presented with a two-week history of intermittent severe, dull, left-sided headache initially given with pain medications but with only slight relief, associated with occasional post-prandial vomiting. There were no note of any neurological deficits. Due to the persistent headache, she was brought to a nearby local hospital where a plain cranial computed tomography scan showed CSDH involving the left frontotemporoparietal area. Since this patient was on Warfarin and a repeat clotting time was prolonged (PT-INR=8), it was discontinued. Two doses of intravenous Vitamin K was given. She was also allegedly advised for evacuation of hematoma, however, the patient declined and went home against medical advice.

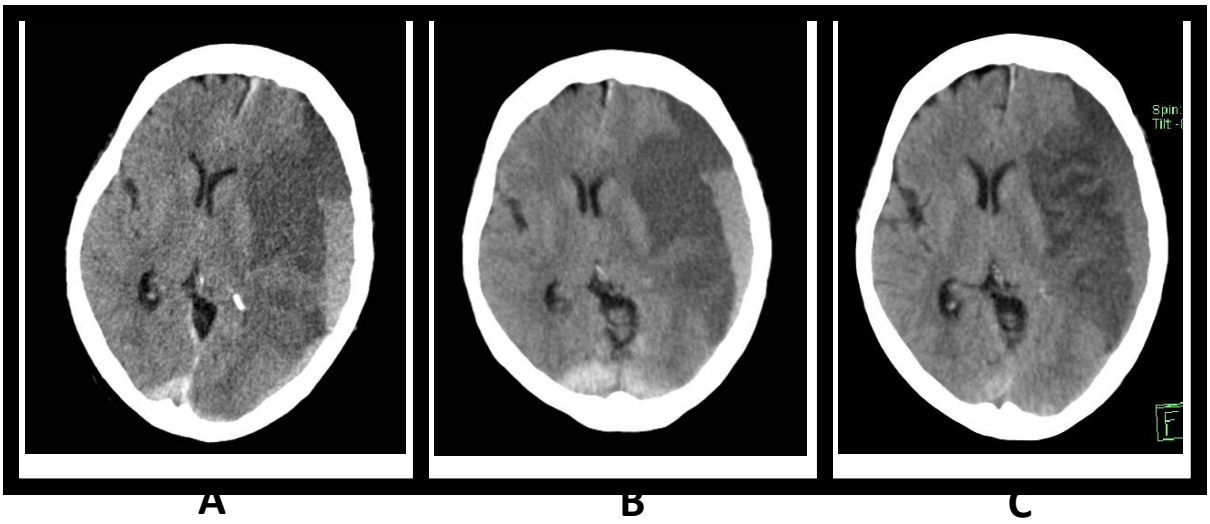

Figure 1: (A) Plain Cranial CT scan done upon admission showing a chronic subdural hematoma in the left frontotemporoparietal area with a maximum thickness of two centimeters with a large area of ill-defined hypodensity in the left hemisphere and a significant midline shift ~1 cm and diffuse cerebral edema; (B) Plain Cranial CT scan done three days after admission showed no progression of the CSDH and CES, no hemorrhagic conversion and a minimal decrease in the midline shift; (C) Cranial CT scan done after 14 days of hospitalization revealed some areas of reperfusion with a further decrease in the midline shift ~0.5 cm.

A few hours prior to admission in our hospital, she was noted to have sudden right sided weakness, associated with decreased verbal output, increased sleeping time and difficulty in arousability, hence brought in for evaluation. On examination, she was noted to have an elevated blood pressure of 140/80, tachycardic at 106 beats per minute with irregularly irregular rhythm. She was seen drowsy but with eye opening to tapping, no regard, no verbal output and did not follow any commands. This patient had preferential gaze to the left and right hemianopia. No papilledema was noted on fundoscopy. There was sluggish corneal reflex on the right eye. There was also note of right central facial palsy and right flaccid hemiplegia with only minimal withdrawal to pain. She had a good gag reflex and her tongue was deviated to the left. An extensor toe sign was present on the right. Upon repeat cranial CT scan, there was still subdural hematoma in the left frontotemporoparietal area with a maximum thickness of two centimeters, but this time, there is a large area of ill-defined hypodensity in the left hemisphere signifying the left middle cerebral artery territory with significant midline shift and cerebral edema (Figure 1A). Hence, this patient was referred to the Neurosurgery service for possible burrhole and evacuation of hematoma to relieve the increased intracranial pressure, however, the surgery was deferred because of the very high risk of cardiac complications.

Blood chemistry and thyroid function tests revealed normal findings. A repeat clotting time was noted to be normal at PT-INR=1.0. 2D-echo with Doppler studies revealed an ejection fraction of 46% with severe Aortic Stenosis, severe Aortic Regurgitation, moderate Mitral Stenosis, mild Mitral Regurgitation, mild Tricuspid Regurgitation and pulmonary hypertension. No thrombus was noted. Digoxin and Propranolol were continued at 0.25 milligrams once a day and 10 milligrams thrice daily respectively.

This patient was started on intravenous intravenous Dexamethasone 5 milligrams every 8 hours and low-dose Mannitol at 75 milliliters every six hours from the time it was decided that no operation will take place. Also, on the third hospital day, a repeat cranial CT scan revealed no hemorrhagic conversion with a minimal decrease in the midline shift (Figure 1B). A repeat clotting time was done (PT-INR=0.98). Warfarin was resumed at half her usual dose at 1.25 milligrams daily. Mannitol was gradually tapered off and was discontinued after 10 days. Dexamethasone was shifted to oral tablets after giving it intravenously for five days. A repeat clotting time done at Day 7 and Day 10 of hospitalization was PT-INR=1.8 and PT-INR 2.1 respectively. After two weeks of hospitalization, she was noted to be awake, with regard, still with no verbal output but occasionally follows commands. She also had some spontaneous movement of her right extremities (Grade 2/5 strength) already. A repeat Cranial CT scan done revealed a decrease in midline shift with some areas of reperfusion (Figure 1C). She was eventually sent home improved. This patient was on regular follow-up every two weeks for Warfarin titration and Rehabilitation exercises. On her last follow-up, she was noted to be awake, with regard, moans and nods to questions and occasionally follows commands. She still had right hemianopia but primary gaze already at midline. She still had right central facial palsy and right-sided

hemiparesis but she can already sustain sitting on a wheelchair. She was also able to tolerate trial of feeding per orem.

CASE DISCUSSION

This patient is on chronic warfarin therapy because of her high-risk valvular AF. In a study done by Kalra et al., patients with AF alone have a three to seven-fold risk of developing stroke and thromboembolism (Karla et al., 2007). The risk is even increased to 17-fold in patients with rheumatic heart disease and AF (Wolf et al., 1978). This justifies the use of warfarin in our patient. However, the risk of developing CSDH while on warfarin anticoagulation is around 42.5 times higher than if no anticoagulation is used, which could explain its occurrence in our patient (Rust et al., 2006).

Subdural hematoma is a collection of blood located between the dura mater and arachnoid. Being a dynamic lesion, the appearance of subdural hematoma in the imaging scans depends on its age. The lesion appears hyperdense compared to the normal brain in the acute phase, isodense in the next few weeks due to fibrinolysis of blood corresponding to the subacute phase and hypodense after a month because of the resorption of blood in the chronic phase (Ellis 1990). This patient developed CSDH; as opposed to acute subdural hematoma, it occurs more often in the elderly without any known damage to the brain such as trauma (Adhiyaman et al., 2002). This is because the bridging veins are more dilated and stretched, hence reducing the counterpressure of the brain to stop bleeding when it occurs (Markwalder 1981). The treatment of CSDH, especially if it's symptomatic, is surgical (Plaha et al., 2008). In a recent meta-analysis, the use of percutaneous bedside twist-drill drainage or burr hole evacuation were found to be equally effective and safe first-line management options in CSDH (Almenawar et al., 2014). In a retrospective study of 500 patients with CSDH, 89% of patients treated surgically were found to have complete recovery with a mortality rate of 2% (Mori et al., 2001). The role of conservative management still has very limited evidence (Miranda et al., 2011; Taussky et al., 2008). However, because of the high cardiac risk of surgery and the Grade 2 Markwalder Score of this patient, we chose to conservatively manage her with Dexamethasone known for its anti-angiogenic effect over the subdural clot membrane. In one retrospective study, the use of Dexamethasone in CSDH showed comparable favorable results as compared to surgical evacuation without putting the patient at risk of irreversible deterioration, reducing hospital stay and costs (Delgado-Lopez et al., 2009). In another study, the use of Dexamethasone in CSDH was found to be a good non-operative option for those with comorbidities because of its low mortality rate at 4% and low risk for significant complications (Sun et al., 2005). However, well-designed trials to prove this benefit are still lacking (Berghauser et al., 2012).

After discontinuation of warfarin, the patient developed CES. CES is one of the most severe stroke subtypes, having a high mortality rate of up to 27% (Ferro 2003). There are three main mechanisms for this, namely 1) local hemostasis causing thrombosis within a cardiac chamber, 2) embolic material from a valvular surface and 3) right-to-left shunts resulting in paradoxical embolism (Weir 2008). Atrial Fibrillation and Valvular Heart disease, which were both present in the patient, accounts for majority of the cases of CES (Han et al., 2007). Oral anticoagulation is indicated as soon as possible since the risk of stroke is 8% within a week, however, there is still no consensus on how early to start or resume anticoagulation (Cervera et al., 2007; Berge et al., 2000). In a study done by de Courten-Myers et al., reperfusion injury causing hemorrhagic transformation peaks within the first three days after CES (de Courten-Myers et al., 1992). Patients started on anticoagulation, either warfarin or warfarin with heparin bridging, were found to be around 13 times less likely to experience stroke progression as compared to antiplatelets (Camerlingo et al., 2005). In another study, patients with a PT-INR >1.6 at the onset of the CES is associated with better outcome (Wakita et al., 2002). However, there are conflicting evidences regarding the safety of anticoagulation (Paciaroni et al., 2007). In one study, there is a 2.5% risk of developing intracranial haemorrhage after starting anticoagulation compared to 0.7% risk of not starting it (Shahpouri et al., 2012).

Mannitol, being the first line drug for cytotoxic edema occurring in stroke, was also given. In a prospective study done by Kinjo et al., in the presence of CSDH, the use of intravenous Mannitol for two weeks contributed to the resolution of the hematoma as manifested in the imaging studies done on their patients, despite the aggravation of clinical symptoms during the first week (Kinjo et al., 1985). In an animal study using rats subjected to occlusion of the vertebral artery to induce ischemia, the combination of Mannitol and Dexamethasone as compared to Mannitol or Dexamethasone alone was noted to effectively reduce brain edema with reduction of further neuronal injury (Wang et al., 2003). However, a human retrospective study showed no significant difference in survival of patients with cerebral edema managed with Mannitol alone compared to combination of Mannitol and Dexamethasone (Canalese et al., 1982). Further studies are deemed necessary.

CONCLUSION

In a patient with multiple comorbidities presenting with concomitant CSDH and CES, the use of Mannitol and Dexamethasone, though still with scarce evidence in the literature, was found to be safe and may have a beneficial effect in our patient. Also, early resumption of anticoagulation after a CES is recommended.

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