

SIMULTANEOUSLY DEVELOPED BILATERAL OCCIPITAL HEMATOMA DURING ANTICOAGULANT TREATMENT

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SUMMARY : *The occurrence of intracerebral hemorrhage in patients treated with anticoagulants is a serious complication, often fatal. Patients with mechanical prosthetic valves require anticoagulation to prevent embolic events. In case of cardioembolic stroke, the safety of continuing anticoagulation is still controversial. We present a report of a patient with a prosthetic mitral valve, who was on anticoagulant treatment and supposed to have developed posterior cerebral artery (PCA) embolic infarction first, then bilateral occipital hematoma due to continuing anticoagulant treatment. Although the risk of recurrent embolism is high in cardioembolic stroke, continuing anticoagulant treatment is hazardous in the acute stage.*

Key Words : *Bilateral Occipital Hematoma, Anticoagulation, Cardioembolic Stroke.*

INTRODUCTION

The principal rationale for using anticoagulation (AC) during an embolic stroke is the prevention of a recurrent embolism. There is limited information on the effects of AC on promoting hemorrhagic infarction (HI) or hematoma formation in unselected series and this remains to be a controversial issue (2, 8, 12, 14).

We describe a patient who simultaneously developed bilateral occipital hematoma as a serious complication of anticoagulation.

CASE REPORT

A 40-year old woman was admitted to our hospital in September 1993 because of deep coma. She had a history of rheumatic heart disease and had a mechanical prosthetic heart valve in the mitral position since 1984. She had been on warfarin AC for 9 years. She had no stroke-like episode during this

period. Ten days before, she suddenly developed vertigo, vague blurry vision and visual field defect which was poorly defined. She continued her medication which was Warfarine 5 mg on a time daily and was not seen by a physician. Her symptoms remained unchanged till she had a generalized tonic clonic seizure and then became unresponsive.

On admission, the pulse rate was 140/ min and rhythmic, and blood pressure was 100/80 mmHg. She was comatose, her pupils were dilated and poorly reactive, oculocephalic responses were absent and decerebration occurred with painful stimulation. She had respiratory insufficiency, so she was intubated and mechanically ventilated. General laboratory work up was unremarkable. CT scan of the head showed bilateral temporo-occipital acute hematoma with surrounding edema compressing lateral ventricle posteriorly, perimesencephalic suprasellar cisternal obliteration and transtentorial herniation (Fig 1). Her prothrombin time (PT) and acti-

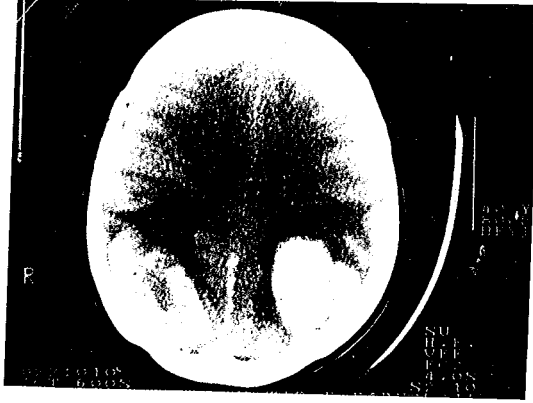


Fig - 1 : CT scan showing bilateral temporo-occipital acute hematoma with surrounding edema compressing lateral ventricles posteriorly.

vated partial thromboplastin time (aPTT) values were 17 and 50 seconds and her platelet count was 283 thousands. Standard 12-lead EKG showed sinus rhythm, inferolateral ischemia with ST segment depression. Echocardiographic examination revealed thrombus formation beyond the mitral positioned mechanical prosthetic valve.

Despite for hyperventilation to a PCO_2 of about 25 mmHg and intravenous Mannitol administration for the management of elevated intracranial pressure, she rapidly deteriorated and died on the second day of admission.

DISCUSSION

The risk of embolism is 2-4 % in patients with mechanical prosthetic heart valves, rates are higher for those in the mitral than in the aortic position (7), so they need life-long anticoagulation to prevent thromboembolism. The favorite sites for lodgment of cardiac emboli are the main trunk and branches of the middle cerebral artery (10). About 10 percent of cerebral emboli enter vertebral-basilar circulation, where they lodge mainly in the top of the basilar artery or in the main trunk or one of the branches of the posterior cerebral arteries (13). Isolated PCA stroke syndromes are relatively uncommon (10).

Our patient had obvious heart disease that was the potential embolic source, did not have TIAs and clinical stroke deficits were maximal at onset. Those clinical features suggest a cardioembolic PCA infarction but unfortunately she was not seen by a

physician during this period, so PCA infarction could not be demonstrated by CT scan. Therefore, we could only suggest that she had developed embolic PCA infarction first, then bilateral occipital hematoma which was demonstrated by CT. In the series reported by Pessin et al (13), embolism was found to be the major mechanism of infarction in the PCA territory and associated clinical features were similar to our patient's.

Cardioembolic strokes have a propensity for secondary hemorrhage transformation. Studies with serial CT scans show spontaneous hemorrhage transformation in up to 40 % of patients with presumed cardioembolic source. Large embolic infarcts are especially prone to secondary hemorrhage (3, 7, 8). One could have difficulty in concerning either continuing or interrupting AC treatment in acute stage of embolic stroke who had been readily anticoagulated because of a potential cardioembolic source.

Bleeding secondary to heparin or warfarin AC accounts for up to 9 % - 23 % of intracerebral hemorrhage (ICH) and about 1 % of the bleeding complications of warfarin are ICH. Bleeding usually occurs when anticoagulation is excessive or the patient has an underlying lesion such as acute cerebral infarction especially embolic in origin (1, 4, 5, 6, 9). Safety of continuing AC treatment during acute stage of cardiovascular stroke is still controversial. Even though some authors suggest that AC treatment can safely be used continuously in patients with hemorrhagic infarction (14), others still recommend to interrupt the treatment for up to 10 days (5, 6, 7, 11).

Our patient continued her AC treatment and simultaneously developed bilateral occipital hematoma despite for her non prolonged PT and aPTT values. To our knowledge, this is the first reported case of a patient with simultaneously developed bilateral occipital hematoma due to AC treatment. We suggest that continuing AC treatment is hazardous in acute cardioembolic PCA infarction.

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