

CEREBELLAR HEMORRHAGIC INFARCTION AFTER PTERIONAL CRANIOTOMY: ANALYSIS OF PATHOGENETICAL MECHANISMS

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ABSTRACT

Objectives: Cerebellar venous infarction after supratentorial craniotomy has emerged as an infrequent, however, important and preventable problem in the recent literature. Cerebellar hemorrhage is secondary to venous infarction, however, exact pathogenetical mechanisms have not been demonstrated. In this study we evaluate possible etiological factors in 5 cases of cerebellar infarction and discuss them in the light of the recent literature.

Methods: This is a retrospective series analysis of 5 cases of cerebellar infarction. For each case several preoperative factors, treatment methods and outcomes were evaluated and discussed.

Results: No etiological factors common to all cases were found in our analysis except for the type of craniotomy and use of postoperative drains. However, no causative relationship could be established for either of these. For pterional craniotomy patients positioning and excessive CSF drainage may be suspected. Early and late outcome after treatment was good for all cases. Two cases required surgical treatment for cerebellar hemorrhagic infarction and related complications.

Conclusions: Peroperative and postoperative excessive cerebrospinal fluid drainage and the

position of the patient leading to hyperextension of the neck during surgery are the most important factors in the etiology.

Key Words: Cerebellum, Complication, Hemorrhagic infarction, Supratentorial craniotomy

INTRODUCTION

Intracerebral hematoma after craniotomy is one of the most common and important complications seen in neurosurgery practice (1). Generally, hematomas occur at the surgical site. However, remote hematomas can be seen as well. Hemorrhages can be seen in the intracerebral, cerebellar, epidural or subdural compartments. In his review Kalfas et al. (2), reported the incidence of postoperative hemorrhage as 0.8% in his series of 4992 craniotomies. However, cerebellar hemorrhages are rare after supratentorial craniotomies.

In the recent literature increased attention is paid to cerebellar hematomas in supratentorial craniotomy cases (3-7). Although rare, this type of hemorrhage is vital because of its localisation and clinical importance. This form of hemorrhage is shown to be secondary to venous infarction.

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Etiology is currently unknown. In this case series we evaluated possible causative factors in cerebellar hematoma after pterional craniotomy, which is the most common type of supratentorial craniotomy in modern neurosurgery.

MATERIAL AND METHODS

This is a retrospective analysis of five cases of cerebellar venous infarction, which occurred at Marmara University Neurosurgical Department. To determine common factors in this complication age, gender, type of craniotomy, side of craniotomy, primary pathology, duration of operation, timing of cerebellar venous infarction, mode of presentation, side of cerebellar infarction were analyzed. All preoperative and postoperative records, including vital signs were carefully reviewed. Coagulation parameters including PT, aPTT, INR and thrombocyte count were evaluated.

Required treatments were also analyzed along with immediate and long term outcomes. All cases were followed for at least two years and neurological examinations during the two year follow up were compared.

RESULTS

Four cases were male and one female. Ages ranged from 45 to 63 (median 51). All cases underwent pterional craniotomies. Four cases were operated from the right and one case underwent bilateral craniotomy. Five craniotomies were for aneurysms and one was for pituitary adenoma. Two aneurysms were located on the anterior communicating artery, two were located bilaterally on both internal carotid arteries and one was on the posterior communicating artery. Duration of the operation ranged from 3 to 6 hours (median 4 hours). Immediately postoperatively all cases were neurologically intact. Cerebellar hemorrhage was detected from 4 hours to the 7th day (median 1 day) after surgery. All patients had decreased levels of consciousness and all diagnoses were made with cranial computerized tomography (CT) (Fig.1). One patient had a magnetic resonance imaging (MRI) of the brain (Fig. 2). The hemorrhage location was on the left

cerebellum in 3 cases, in the right cerebellum in one case and bilaterally located in one case.

One case was managed with surgical decompression, one case underwent a ventriculoperitoneal (V-P) shunt placement procedure for secondary hydrocephalus, and 3 cases were managed medically with anti-edema drugs. All cases were followed for at least two years. At the two years follow-up one case had left sixth cranial nerve paralysis, two cases had minor ataxia and two cases were neurologically intact (Table I).

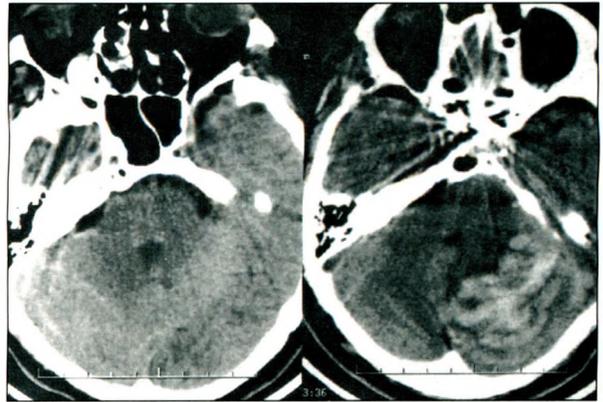


Fig. 1 : Preoperative cranial CT of the patient with anterior communicating artery aneurysm (left). Cranial CT 4 hours after the operation (right).

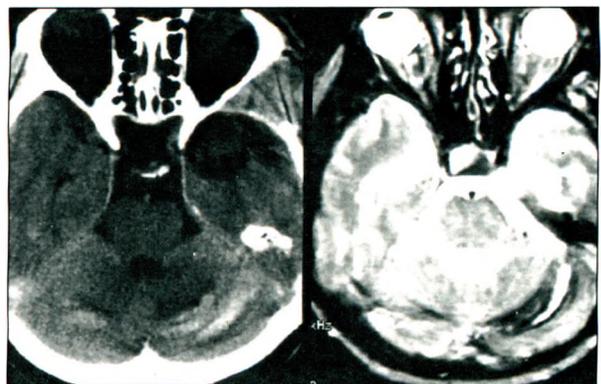


Fig. 2 : Postoperative cranial CT of the patient with pituitary adenoma (left). MRI at postoperative day 1 demonstrates venous infarction in the cerebellum (right).

Table 1: Presentation of clinical parameters and outcome for five patients.

TABLE 1					
Age/Sex	Primary Pathology	Time of Infarction	CT	Treatment	Result
45/M	Pituitary Adenoma	Postop. 7th day	Left cerebellar hemorrhage	Antiedema treatment	Normal
46/M	A. Com. A. Aneurysm	Postop. 1st day	Bilateral cerebellar Hemorrhage	V-P Shunting	Left VI.nerve palsy
51/F	P. Com. A. Aneurysm	Postop. 2nd day	Left cerebellar hemorrhage	Antiedema treatment	Ataxia
52/M	A. Com. A. Aneurysm	Postop. 1st day	Right cerebellar hemorrhage	Antiedema treatment	Normal
63/M	Bilateral internal carotid artery aneurysm	Postop. 4th hour	Left cerebellar hemorrhage	Suboccipital craniectomy and hematoma evacuation	Slight ataxia

DISCUSSION

Interest in remote hematomas after supratentorial craniotomies has grown in recent years. In Kalfas' (2) series distant hematomas were found in 7 cases in a group of 40. None of these cases had bleeding in the posterior fossa. In only 1 of the 4 remote hematoma cases, which are presented in Waga's (8) publication, the bleeding is located in the cerebellar hemisphere. In his literature review in 1996 Brisman (1) found 37 post-craniotomy distant hemorrhages. Of these cases, 17 were confined to the posterior-fossa.

A particular etiological factor could not be found for the post craniotomy distant hemorrhage. According to Brisman (1), since most of the cases became symptomatic within few hours after surgery, the bleeding is thought to occur during or just after the surgery. In a careful literature review one finds that the age, sex, pathology and type of intervention of the cases are quite heterogeneous. Post supratentorial craniotomy cerebellar hematomas are seen very infrequently. Early postoperative cranial CT have been performed more frequently in the recent years and it is possible that this is the reason why this entity is encountered more frequently.

However, several pathological mechanisms have been proposed, without adequate scientific proof. Postoperative excessive cerebrospinal fluid (CSF) drainage may cause inferior dislocation of cerebellum and tear of superior vermician veins. In 3 cases reported by Yoshida (9) it is claimed that drainage systems put in epidural space might have caused excessive negative pressure

and that might be responsible for the hemorrhage. All of our patients had postoperative epidural drains.

Peroperative patient positioning is one of the most commonly suspected factors. Several authors consider this pathophysiological mechanism. Especially in pterional craniotomies hyperextension of the patient's neck and subsequent turning of the head to the side can cause relative obstruction on the ipsilateral jugular vein. This is more important, if the dominant jugular vein is under pressure (5). All of our cases underwent pterional craniotomies, however no extensive neck hyperextensions were done, as evaluated both by the neurosurgery and anesthesiology teams intraoperatively.

Venous infarction is the primary pathology in all postoperative remote cerebellar hemorrhages. In his presentation of 4 cases Kuroda et al (3) demonstrated that the hemorrhage was on the tentorial surface of hemisphere in each case. Whereas in Toczek's (6) series hemorrhage was within cerebellar parenchyma in all cases. All cases in our series were located within the cerebellar parenchyma just below the tentorium.

In his review of 5 cases, Papanastassiou (4) pterional intervention was performed and all hemorrhages were detected within the contralateral hemisphere. In our cases, in 1 of them hemorrhage was ipsilateral and in 4 it was in the contralateral cerebellar hemisphere. In all 5 cases it was near the tentorial surface within the cerebellar parenchyma. This led us think that the cause of hemorrhage was injury to ascending

veins extending from the cerebellum to the tentorium.

Several other factors like arterial hypertension, anticoagulation therapy, coagulopathy, aneurysm or arteriovenous malformations were suspected (4). However, none of our patients had a history of hypertension, nor were any of them found to be hypertensive postoperatively. Coagulation tests were within normal limits in all of our patients and arteriovenous malformations were neither suspected in neuro-imaging nor found in decompressive surgeries.

Cerebellar hematoma is seen in 3.5-4.9% rate postoperatively in tissue excision cases like epilepsy surgery (6,7). However, cerebellar hematoma after craniotomy for other reasons is encountered in only 0.3-0.5% of the cases (4,10). In addition most of the cases reported in literature are aneurysms and parasellar tumors. In these cases sylvian cistern dissection and excessive CSF drainage is performed (7,10). This might be the reason for cerebellar shifts and stretching of the veins. After supratentorial craniotomy many factors are considered responsible for a cerebellar hematoma. None of the factors mentioned above are universally accepted and none is believed to be the sole etiological factor for cerebellar hemorrhage. Such hemorrhages are believed to be multifactorially mediated.

Small hematomas can be managed medically with serial imaging. However, severe mass effect in the posterior fossa related to the hemorrhage may necessitate decompressive surgery. Fourth ventricular compression with non-communicating hydrocephalus should be managed with CSF diversion procedures. However, diversion procedures for mass effect within the posterior fossa should be cautiously done and the threshold for surgical decompression should be kept low as brain stem decompression cannot be achieved neither with external ventricular drainage nor shunting. Immediate lateral 1/3 cerebellar resection should be performed, if

needed. Expanded suboccipital craniectomy and duraplasty are the procedures that decompress posterior fossa. Decompression was required in only one of our cases and was performed with excellent surgical results.

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