Cerebellar Haemorrhage: Clinical, CT Findings and Outcome

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✔ **SEREBELLAR HEMORAJİ: Klinik, BT Bulguları ve Prognoz**

Bu çalışmada Bilgisayarlı Tomografi (BT) ile tanı konulan 13 serebellar hemoraji (SH)'li hasta sunulmaktadır. Yedi olguda (%53.8) etyolojik faktörün hipertansiyon olduğu düşünülmüştür. En sık görülen başlangıç bulguları vertigo, bulantı, kusma ve baş ağrısıdır. Altı hastada sol serebellar hemisfer, beş hastada sağ serebellar hemisfer ve iki hastada da vermian hemoraji saptanmıştır. SH'li hastaların üçü kaybedilmiştir. İlginç olarak, vermian büyük bir hematomu bulunan hasta sekelsiz iyileşmiştir. Hastaların klinik gidişi değerlendirildiğinde, başlangıçtaki bilinç durumunun, hemoraji boyutu ve lokalizasyona göre prognoz için daha belirleyici bir parametre olabileceği sonucuna varılmıştır.

Anahtar Kelimeler: İntraserebral hemoraji, serebellar hemoraji.

✔ We studied 13 patients with cerebellar haemorrhage diagnosed by computed tomography (CT). Hypertension was accepted to be the etiological factor in 7 patients (53.5%). The most common presentation findings were vertigo, nausea, vomiting and headache. In 6 patients haemorrhage was located to left cerebellar hemisphere in 5 to right cerebellar hemisphere and in 2 to vermis. 3 patients were lost while a patient with a large vermian hematoma improved without any sequel. It is decided that the prognosis mostly depends on the level of consciousness from the beginning rather then the localization and the diameter of the hematoma.

Key words: Intracerebral haemorrhage, cerebellar haemorrhage.

INTRODUCTION

Cerebellar haemorrhages constitute about 10% of all intracranial bleeds and 2% of all stroke patients[1,2]. The etiological factor is hypertension in two third of the cases. Arteriovenous malformations, blood dyscrasias, trauma, anticoagulation and neoplasms are the others. In one fourth no etiological factor can be demonstrated [3,4]. Cerebellar haemorrhages mostly occur in one of the hemispheres. In Fisher's cases right hemisphere has been involved twice than the left[5].

Vertigo, vomiting, headache and gait ataxia are the prominent symptoms. Tinnitus and hiccups can rarely be seen[6].

In examination ataxia, ipsilateral conjugate gaze palsy, cranial nerve signs (peripheral facial palsy, nystagmus, miosis, sixth nerve palsy, skew deviation, trochlear palsy, absence of pharyngeal reflex), hemiparesis, Babinski response and neck stiffness can be detected[7]. In 25% of cases there is no detorioration of consciousness. In 40% drowsiness, in 9% stupor and in 26% coma has been reported[8].

In cerebellar haemorrhages the clinical outcome has not always been correlated with the diameter and localization of the hematoma as the level of consciousness at onset[1]. The development of hydrocephalus and the deteriorating level of consciousness have been reported as the indicators of bad

prognosis. Patients with vermian haemorrhages have also been accepted to have a poor outcome[6].

MATERIAL AND METHODS

13 patients with CT evidence of cerebellar haemorrhage seen at Ondokuz Mayıs University, Medical Faculty, Neurology Department from September 1991 to February 1994 have been reviewed.

The diameter of the hematomas have been measured in milimeters. Biochemical tests, ECGs and telecardiographies of all patients have been performed. CT of five patients have been repeated a month later. Patients have been treated against brain edema and the hypertensive ones against hypertension. All cases have been reviewed according to their clinical findings at onset, early and late neurological features, the diameter of the hematoma, the presence of hydrocephalus, hypertension, diabetes mellitus and cerebrovascular disorders and the clinical outcome.

RESULTS

The mean age of the thirteen cases; seven men (54%) and 6 women (46%) was 95.6 ranging from 30 to 75. The clinical and radiological findings are summarized in Table I. Eight patients had been hypertensive for at least three years. Blood pressures of four were high on admission. Two of the three patients with high blood glucose levels had been traced as diabetic before. Five patients had been smokers for at least ten years. Three of the thirteen patients died on the second, third and sixth days of their illness (patients, 9,10,12) and two of them had been obtunded on initial examination.

Two of the three patients with hematomas greater then 30 mm in diameters (patients 2 and 9) were lost. Two patients had vermian hematomas (patients 1,13). The diameter and localization of the hematomas are shown in Table II and III.

DISCUSSION

The female male ratio of the 13 cases was 6/7. The same ratio in Fisher's cases is 13/8[6]. Other series give ratios like 5/14, 1/9 and 26/30[6].

Cerebellar haemorrhages mostly originate from distal branches of the superior cerebellar artery or from PICA[9]. Like Fisher and Mc Kissok's cases left cerebellar hemispheral predominence was noted in our patients.

The most frequent etiological factor for approximately 70% of the cases has been reported to be hypertension[3,4]. In our seven patients (53.8%) the etiological factor was hypertension. Vomiting was the initial symptom in our ten patients. In Ott's cases 7 has been noted in 42 over 44[8]. The frequency of vertigo was the same with vomiting (9/13). In the previous reports about cerebellar haemorrhage vertigo has been noted as 24/44, 8/21 and 4/12[8,10].

Headache is also a frequent finding. Although it is expected to be occipital in localization it can be frontal or lateral[6]. One of our patients (patient 4) had frontal headache. Though headache is not a spesific symptom of cerebellar haemorrhage it has to be taken seriously. By this way it can be differentiated from labyrinthine diseases with which it can easily be confused.

It is difficult to predict the prognosis in cerebellar haemorrhages[6,7,11]. The patient can become comatose in the first 48 hours without any warning sign. It is valid for the first two weeks as well. The clinical course can change even after a month[12].

It has been reported by Ott et all that 50% of the patients with cerebellar hae-

Table-I: The Distribution Clinical Findings of Cerebellar Heamorrhage (n=13).

Clinical Findings	Patient No	%
Vomiting	10	76.9
Vertigo	9	69.2
Headache	9	69.2
Dysarthria	6	46.1
Ataxia	6	46.1
Nausea	4	30.7
Nystagmus	2	15.3
Right extensor plantar respons	2	15.3
Equal reactive pupils	2	15.3

Table-II: Size of Haemorrhage

Size	Patient no	%
0–10 mm	2	15.3
11–20 mm	3	23.0
21–30 mm	5	38.4
> 30 mm	3	23.0

Table-III: Location of Haemorrhage

Location	Patient no	%
Right cerebellar haemorrhage	5	38.4
Left cerebellar haemorrhage	6	46.1
Vermian haemorrhage	2	15.3

morrhage become comatose in the first 24 hours and 75% in the first week[8].

Level of consciousness seems to be the most important indicator of the prognosis. The mortality rate among the responsive patients is 17% when it is 75% among the unresponsive ones[13]. The diameter of the hematoma is also important. Cerebellar heamorrhages with a diameter smaller than 3 cm are not responsible for the deteriorating level of consciousness or brain stem compression. Brain stem compression and neurological detorioration is frequent if the diameter is greater than 3 cm with ventricular enlargement[13,14].

Blood seen in the fourth ventricule is also an indicator of poor prognosis[15]. In 2 of the 3 patients we lost deteriorating level of consciousness had been noted in the first 24 hours. Though the diameter of the hematomas were greater than 30 mm. patients no

1 and 6 responded well to medical therapy. There are cases in literature with cerebellar haemorrhages greater than 40 mm in diameter showing neurological recovery without surgery[1,2]. The most important indicator for surgery appears to be the development of hydrocephalus[11,16].

In autopsy series vermian haemorrhages constitute 6% of all cerebellar haemorrhages. They are mostly due to anticoagulation and have a poor outcome[6]. Our patient with vermian haemorrhage (patient 13) had been hypertensive for fifteen years and under antihypertensive medication for three months. Anticoagulation was not the case. He responded well to anti-oedama therapy.

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