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Cerebellar Infarction: Clinical and CT Findings

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✓ In this study, clinical and radiologic analysis of 16 patients with cerebellar infarct confirmed by CT, has been presented. Hypertension was the most common risk factor (68.7%). In the early stage, nausea/vomiting and dizziness were the most prominent symptoms. CT studies revealed the presence of eleven unilateral, two bilateral and one vermian infarcts. 2 patients who had impairment of consciousness from the beginning died (12.5%).

Key words: Cerebrovascular disease, cerebellar infarction

✓ Serebellar İnfarkt : Klinik ve BT Bulguları

Bu çalışmada, BT ile tanısı doğrulanmış 16 serebellar infarkt olgusunun klinik ve radyolojik analizi sunulmuştur. Hastalarda en çok görülen risk faktörü hipertansiyondur (%68.7). Erken dönemde en dikkat çekici semptomlar bulantı, kusma ve baş dönmesidir. BT incelemeleri 11 hastada unilateral, 2 hastada bilateral, 1 hastada da vermian infarkt bulunduğunu ortaya koymuştur. Başlangıçtan itibaren bilinç bozukluğu gösteren 2 hasta kaybedilmiştir(%12.5)

Anahtar Kelimeler: Serebrovasküler hastalık, serebellar infarkt.

INTRODUCTION

Infarction of the cerebellum represents approximately 0.5–1.5 % of strokes and has high mortality^(1,2). In one-third of the cases cerebellar symptoms are not apparent and the diagnosis may be overlooked. This patients are often misdiagnosed as labyrintihine or peripheral vestibular disorders because the early stage findings of cerebellar infarcts are dizziness, nausea and vomiting ⁽³⁾. The decision of cerebellar infarct could be established by evaluation of the clinical findings of the intermediate stage. the evolving of clinical findings to the intermediate stage. The diagnosis is also difficult in the late stage when stupor and coma develop⁽¹⁾.

MATERIAL and METHODS

16 patients who were hospitalized in Ondokuz Mayis University Medical Faculty, Neurology Department between September 1991 and April 1994 were taken to the study. Diagnosis were confirmed in all patients by brain CT. Patients who had mainly brain-stem ischemia and multipl infarcts were excluded. Stroke risk factors, early and late clinical symptoms and fin-

dings were evaluated as well as the tomographic location of ischemia and clinical evolution.

RESULTS

The mean age of 16 patients with cerebellar infarcts, 9 males and 7 females, were 58.7 years. The evaluation of patients regarding risk factors are listed in Table–I. Hypertension was the most common risk factor. It was present in 11 patients. Smoking, diabetes mellitus, hypercholesterolemia, obesity, atrial fibrillation and prior stroke history followed respectively. The symptoms and signs of the initial and late stage of cerebellar infarct are presented in Table–II. The most common symptoms were nausea, vomiting, dizziness, unsteady gait, headache and dysarthria.

The most common symptoms of the early stage were nausea, vomiting, dizziness and headache. In 9 patients with headache location of the headache was occipital in 4, periauricular in 4 and facioocular in 1. Ataxia was the major finding in both initial and late stages. The tomographic distrubi-

Table-I: Risk factors in 16 patients with cerebellar infarction.

	No	%	
Hypertension	11	68.7	
Smoking	8	50.0	
Ischemic heart disease	5	31.2	
Hypercholesterolemia	4	25.2	
Diabetes mellitus	4	25.0	
Obesity	3	18.7	
History of stroke	2	12.5	
Atrial fibrillation	2	12.5	

Table-II: Distribution of The Early and Late Stage Symptoms and Neurologic Signs of The Cerebellar Infarction. (n=16).

	Early Stage		Late Stage	
Symptoms	(First 2	4 hours)	(After 48	8 hours)
	No	%	No	%
Nausea/vomiting	11	68.7	_	_
Dizziness	10	62.5	4	25
Headache	9	56.2	-	_
Dysarthria	5	31.2	4	25
Gait disturbance	-	-	3	18.7
Neurologic Signs Ataxia	10	62.5	4	25
Nystagmus	4	25	2	12.5
Lethargia	_	_	3	18.7
Hemiparesis	***	_	3	18.7
Miosis		****	3	18.7
Coma	_	_	3	18.7
Anisocoria	_	_	2	12.5
Conjugate deviation		_	2	12.5
Facial palsy	_	_	2	12.5
Upward gaze palsy		_	1	6.25
Horner's syndrome	_	_	1	6.25

Table-III: Tomographic distrubition of ischemic regions.

	No.	<u></u>	
Unilateral	11	68.7	
Bilateral	2	12.5	
Unilateral+Vermian	2	12.5	
Vermian	1	6.2	

Table-IV: Clinical findings of PICA and SCA territory infarcts.

Symptom/Sign	PICA infarct	SCA infarct
Ataxia	9/10	5/6
Nausea/vomiting	8/10	4/6
Headache	7/10	2/6
Dizziness	6/10	6/6
Nystagmus	2/10	4/6
Dysarthria	1/10	4/6

tion of ischemic regions is presented in Table–III. CT revealed PICA (Posterior inferior cerebellar artery) territory ischemia in 10 patients and SCA (Superior cerebellar artery) ischemia in 6 patients. In Table–IV, the clinical findings of PICA and SCA territory infarcts are shown. In all patients antiedema and antiaggregant medication were given. Hypertensive patients received also antihypertensive therapy. 2 of the 4 patients who had impairment of consciousness at the onset died. These 2 patients had ischemia in the PICA territory, Only 1 patient with SCA ischemia had brain–stem compression findings.

DISCUSSION

Patients with cerebellar infarct have the usual risk factors: hypertension, a history of prior stroke, ischemic heart disease and atrial fibrillation. In our study, hyperten-

sion is the most common risk factor. The findings of classical SCA syndrome described by Mills and Guillain are ipsilateral dysmetria, ipsilateral Horner's syndrome, contralateral temperature and pain sensory loss and contralateral IVth nerve palsy⁽⁴⁾.

However, this syndrome results from the ischemia of the brain–stem region fed by SCA and is in fact very uncommon. In most prevalant SCA ischemia vestibular signs and headache are not seen frequently, gait and limb ataxia, dysarthria and horizontal nystagmus are prominent^(3,6,7). Contrary to Kase and Amarenco's series, in our patients with SCA ischemia verified by CT vestibular symptoms were prominent.

Patients with PICA territory infarcts frequently experience vertigo, vomiting, headache and gait imbalance at onset, with horizontal nystagmus and gait ataxia and less prominent ipsilateral limb ataxia⁽¹⁾. In our patients with PICA territory ischemia

ataxia, nausea, vomiting and dizziness were also the most common findings. Facial palsy was present in two patients in the late stage. CT showed ipsilateral cerebellar infarct and prominent edema. The presence of facial palsy in these patients can be explained by the compression of the swelled cerebellum to the pontocerebellar junction. However, in these cases the fourth ventricules were not compressed and there were no hydrocephalus.

In cerebellar strokes, it is well known that the level of consciousness varies greatly. Prognosis of patients who have impairment of consciousness from the beginning is poor⁽⁸⁾. This suggestion is strengthened by our study, as we lost 2 of 4 patients with impairment of consciousness onset. Patients who have not shown impairment of consciousness are expected to become stable or deteriorate within 24 or 48 hours ⁽⁹⁾.

Infarction of the cerebellum is uncommon. However, its clinical importance is considerable because it may initially present as a 'benign labyrinthine disorder' that can evolve into life-threatening brain-stem compression from postinfarct edema^(10,11). The key findings that point to the diagnosis of cerebellar infarction are related oculomotor signs. The subacute presentation of the case should prompt the physcian towards this diagnosis.

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