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Thalamic Haemorrhage: Symptomatology, CT Findings and Prognosis

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✔ Thalamic haemorrhages still remains to be interesting subjects to study with their 30% incidence amongst intracerebral hematomas, highly variated clinical characteristics and relatively good prognosis. In our study, 21 haemorrhagic patients, 12 male patients and 9 female patients respectively, were examined for their clinical characteristics, CT findings and prognosis.

Key words: Intracerebral haemorrhage, thalamic haemorrhage.

✔ Talamik hemorajiler, intraserebral hematomlar içinde %30'a yaklaşan sıklığı, geniş varyasyon gösteren renkli klinik özellikleri ve prognozlarının görece iyi olması ile serebrovasküler hastalıklar içinde ilgi çekici olma konumunu sürdürmektedir. Çalışmamızda 12 erkek, 9 kadın olmak üzere toplam 21 talamik hemorajili hastanın klinik özellikleri, BT bulguları ve prognozları gözden geçirilmiştir.

Introduction

The thalamus is a large, ovoid mass of gray matter that forms the major part of the diencephalon. It is situated on each side of the third ventricle[1]. Blood is circulated to the thalamus by the posterior cerebral artery, posterior clinoid arteries and central cerebral artery, respectively[2]. Rupture in any arteries circulating blood to the thalamus may cause a limited hematom or because of the neighbourhood may include the internal capsule and cerebral peduncle . Clinical table has a wide spectrum ranging from deep coma to serious motor and sensorial deficits[3]. However, it is known as a characteristic of thalamic haemorrhages that they cause moderate hemiplegia/ hemiparesis or more serious sensorial deficits than motor deficits[4]. In thalamic haemorrhages, ocular deficits such as upward deviation of the eyes frequently occur. Skew deviation and anisocoria may also occur. In some series lateral gaze palsy have been reported[3].

Thalamic haemorrhages constitute 20-30% of the intraparenchimal haemorrhages of the brain[5,9]. The probability of early mortality is very high whereas some patients, especially those who have small hematomas, generally survive[10]. Permanent deficits may exist depending on the topographic localisation of haemorrhage[10,11]. In patients who survive (76%) the lesion is in the dorsolateral thalamic area. It is expected that prognosis is worse for one third of patients who have hemiparesis or hemianesthesia[12]. It is known that haemorrhage in the medial thalamus, medial temporal lobe, hippocampus and mamillary substances can cause amnesia[13].

It is claimed that if a hypertensive patient develops amnesia and moderate sensorimotor deficit a lesion in the medial thalamus which has a very good prognosis must be suggested.

Posterior thalamic lesions can cause upward gaze palsy. Neglect phenomena may be seen in lesions of non-dominant hemisphere. There may occur saccadic hypometry in the opposite of lesion and defective pursuit movements in the side of lesion. There also may occur Horner's syndrome depending on the compression of hypothalamus[15]. If the lesion is in the group of ventrolateral and anterior nucleus, it may cause resting and intention tremor affecting the afferents from intraluminal nucleus to striatum[16].

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It is expressed that the size and volume of bleeding, opening to the ventricle and the existence of ventricular dilatation may be related with the prognosis. The age and bilateral neurologic symptoms are correlated with bad prognosis[17]. Invasion to the hypothalamus and the deviation of the 3.rd ventricle from the midline with a length more than 2 mm. support the idea that prognosis may be worse[17,18].

MATERIAL and METHODS

In our study, 21 patients, 12 male and 9 female patients respectively, who were hospitalized in the Neurology Clinic of Ondokuz Mayıs University Faculty of Medicine between June 1991 and June 1992, were observed. The ages of the patients were between 37 and 72 (the average of the ages is 57.13).

In all patients primer hypertensive thalamic haemorrhage were diagnosed by CT. Routine biochemical tests, ECG and telecardiographies were made. Cerebrovascular disease risk factors were sought. Complete neurologic examination were performed. Motor deficits were classified as hemiparesis and hemiplegia. The assesment of consciousness was evaluated by modified Glasgow Coma Scale.

Volume of the hematomas were calculated by dividing the multiplication of width, length and height of the hematoma by two. The relation between the size of hematoma and the clinical state was defined[19].

RESULTS

The clinical characteristics, mostly seen, in the patients were hemiplegia and hemiparesis. 9 patients had hemiplegia whereas 8 patients had hemiparesis. 11 patients were recorded to have hypoesthesia

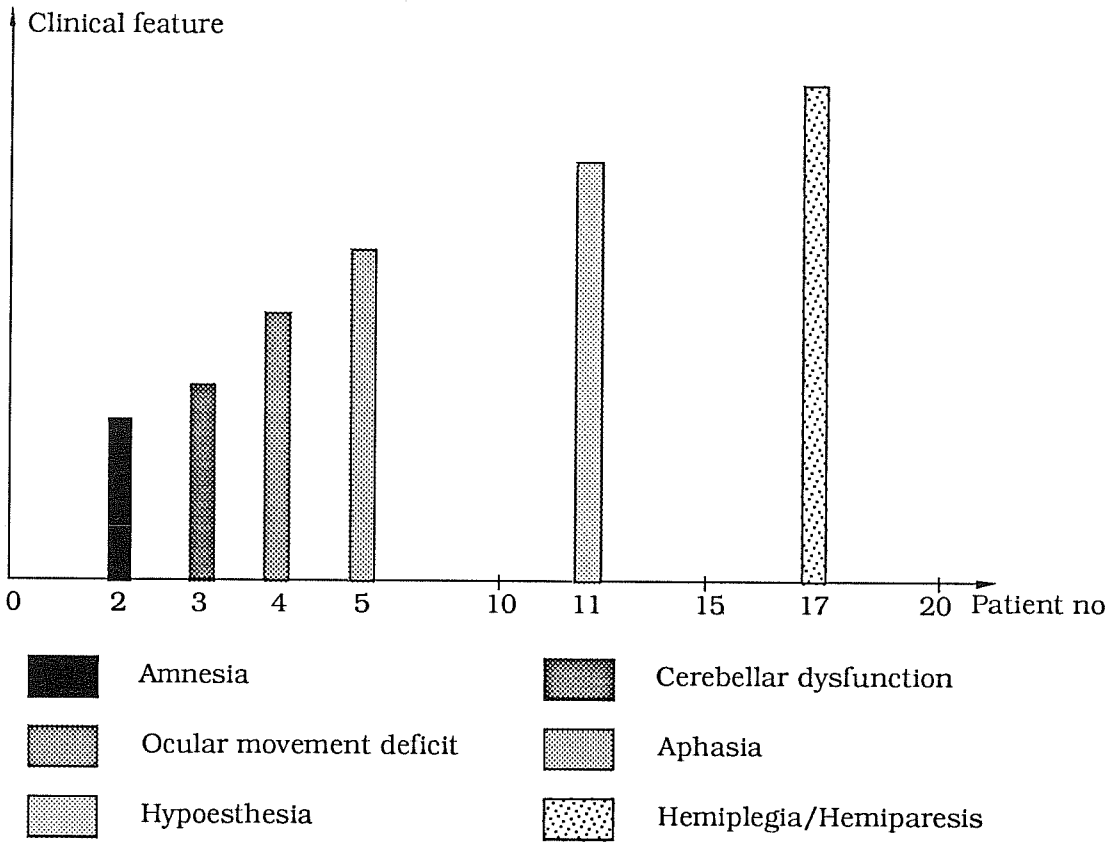


Figure-1

while 5 of them were recorded to have transcortical aphasia. Cerebellar tests of 3 patients were impaired. Neglect phenomenon were not seen in any patient. 4 subjects had ocular movement deficits such as downward and inward deviation of the eyes. 2 subjects had amnesia. (Figure 1).

By CT, in 4 patients hematoma volume was less than 30 ml. In 11 patients it was between 31 and 50 ml. In 2 patients it was more than 71 ml. In 6 subjects haemorrhage were opened to the ventricle.

All of the patients were medically treated. 2 of them died, 1 of them in the 7^{th} and the other one in the 16^{th} hospitalization day respectively. The end of the first month, 4 patients were recorded to have serious deficits while 11 of the patients were recorded to have moderate deficits. 4 patients completely recovered.

DISCUSSION

The most seen clinical characteristics of thalamic haemorrhages–with respect to their incidences-were hemiplegia, hemiparesis, aphasia, hypoesthesia and ocular movement deficits. Neglect phenomenon were not seen any of the patients. This may be because of the fact that minor hemisphere hematomas did not effect the corticolimbic reticular ring directly or the clinician might be concentrated on other major symptoms.

In 4 subjects, there occurred looking paralysis which were expected in posterior thalamic lesions. We did not record Fisher's gaze paralysis in any of the patients.

The greater the dimension of the hematoma, the more serious the impairment of consciousness will be. The volumes of hematoma for the 2 of the patients, who died, were about 44 ml. and 72 ml. It was confirmed by CT that each of the hematomas were opened to the lateral ventricles and caused hydrocephalus. Our another conclusion, that supports the general literature informations, is that motor deficits are less seen in hematomas which had volumes less than 30 ml.

One of the patients had not loss of pain at his face, like Hirosa and all's reported case. The ventroposteromedial nucleus of this patient had no defect while it is possible that ventroposterolateral nucleus may be effected by the hematoma.

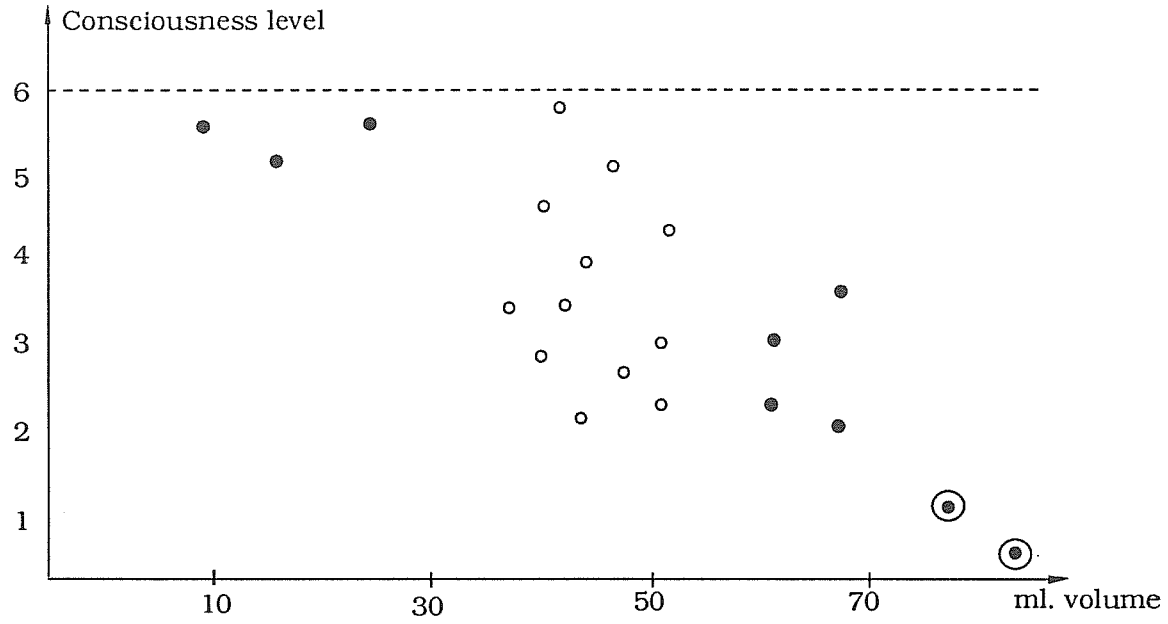
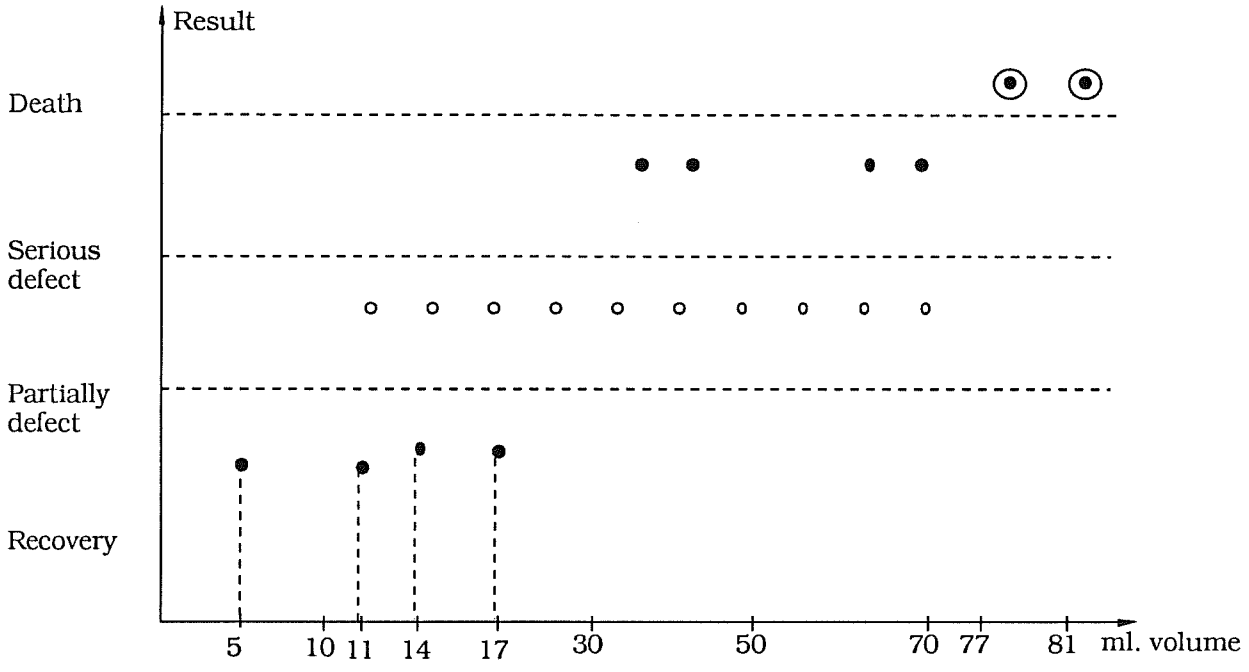


Figure-2



Figure–3

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REFERENCES

1. Snell RC. Clinical Neuroanatomy For Medical Students 2nd ed. USA, Little Brown and Company, 1987; 245 .
2. Walton J : Brain's disease of the nervous system. Oxford uni. press. New–York. 1977, 369.
3. Adams H, Biller J. Hemorrhagic intracranial vascular disease. In Joynt JR (ed). Clinical Neurology (5 th ed) Vol 2, Philadelphia, J.B. Lippincott Company, 1992; 8.
4. Fisher CM : The pathologic and clinical aspects of thalamic haemorrhage. Trans Am Neurol Assoc, 1959, 84: 56.
5. Kwak R, Kadoya S, Susiki T : Factor affecting the prognosis in thalamic haemorrhage. Stroke, 1983, 14: 493–500.
6. Dobato JL, Villanueva JA, Gimenez–Roldan: Sensory ataxic hemiparesis in thalamic haemorrhage. Stroke, 1990, 21, 1741–1753.
7. Walshe TM, Davis KR, Fisher CM : Thalamic haemorrhage: A computed tomographic–clinical correlation. Neurology, 1977, 27: 217–222.
8. Barraquer–Bordas L, Illa I, Escartin A et al: thalamic haemorrhage. A study of 23 patients with diagnosis by computed tomography. Stroke, 1981, 12: 524–527.
9. Weisberg LA: Thalamic haemorrhage: Clinical–CT correlations. Neurology 1986, 36: 1382–1386.
10. Kawahara N, Sato K, Muraki M et al: CT classification of small thalamic haemorrhages and their clinical implications. Neurology, 1986, 36: 165–172.
11. Azouvi P, Pappata S, Baron JC et al: Attaque sensitive pure par hematoma thalamique. Rev Neurol (Paris). 1988, 144: 212–214.
12. Piepras U, Rieger P; Thalamic bleeding: Diagnosis, course and prognosis. Neuroradiology. 1981, 22(2): 85–91.
13. Dennis c, Lewis S, Steven S, Burke P: Medial thalamic haemorrhage with amnesia. Arch. Neurol. 1983, 40: 611–613.

14. Ojemann RG, Mohr J: Hypertensive brain haemorrhage. Clinical Neurosurg. 1975, 23: 220–244.
15. Hirose G, Kosoegawe H, Saieski M, Kitagawa Y: The syndrome of posterior thalamic haemorrhage. Neurology. 1985, 35: 998–1001.
16. Schlitt M, Brown JW, Zeigner HE, Galbraith J: Appendicular tremor as a late complication of intracerebral haemorrhage. Surg Neurol. 983, 25: 93–95.
17. Fisher CM: Modern concepts of cerebrovascular disease. New–York, Spectrum, 1975, 1–41.
18. Pryse–Phillips W, Murray TJ: Essential Neurology, New–York, Medical Examination Publishing Co., Inc. 1978, 191–196.
19. Radberg JA, Olsson JE, Radberg CT: Prognostic parameters in spontaneous intracerebral hematomas with special reference to anticoagulant treatment. Stroke. 1991, 22, (5): 571–576.