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Traumatic Subarachnoid Haemorrhage: Analysis of 89 Cases

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Summary

Between 1985 and 1990, 2056 patients with head injuries were treated in the Department of Neurosurgery at the Izmir State Hospital. Among them, 89 patients with traumatic subarachnoid haemorrhage (TSAH) were analysed retrospectively. It was noted that focal or global contusion accompanying TSAH was the most common pathology which could be detected in computer tomographic (CT) imaging. CT did not show any other intracranial lesion in 13 cases.

Vasospasm developed in one patients and hydrocephalus in two others in the acute stage.

Considering the relationship between the severity of a subarachnoid haemorrhage detected on CT and the mortality rate, it was seen that the mortality rate was higher in patients who suffered diffuse subarachnoid haemorrhage with intracerebral or intraventricular clots.

Keywords: Traumatic subarachnoid haemorrhage; computed tomography; head trauma.

Introduction

Although traumatic subarchnoid haemorrhage (TSAH) is not a rare clinical condition following a head injury, clinical studies reported on this subject are very few[7, 10, 11]. It is known that they are not fatal unless they are associated with other intracranial pathology. The only exception of this is subarachnoid haemorrhage (SAH) orginating from a rupture of the vertebral artery and there are many reports of such cases in the medicolegal literature [1, 3, 5, 6, 12, 13].

In this study we aimed to establis the ratio of TSAH in all head trauma cases and to determine its relationship with associated intracranial lesions. We also tried to find out if any relationship exists between the mortality rate and the severity of subarachnoid haemorrhage (TSAH) detected by computerized tomography (CT).

Clinical Methods and Material

2056 cases with head injuries were treated in the Department of Neurosurgery at the Izmir State Hospital between 1985 and 1990. After clinical and/or radiological investigations, 89 of these patients were diagnosed as TSAH. 12 of the patients were female whereas 77 were male. The average age was 39.8 and ranged between 13 and 98 (Fig. 1).

The causative factor was predominantly traffic accidents (59 cases). The other causes were falls in 23 patients and assaults in 9 patients. Alcohol intake was noted in 20 of the cases.

Our criteria for diagnosis was evidence of meningeal irritation (neck stiffness) and detecting SAH by spinal tap and/or CT. The patients who had positive signs of meningeal irritation without positive spinal tap or CT results were excluded from this study.

The Glasgow Coma Score of each patient was evaluated after admission and CT scanning was performed immediately on a GE 640 scanner with a 640 × 640 matrix along the orbitomeatal line. Contrast medium was not used.

Patients with intracranial mass lesions underwent emergency surgery. All other cases were treated conservatively. In addition, multiple spinal taps were performed on patients who had pure TSAH, with the intention of facilitating cerebrospinal fluid circulation. The outcome was scored according to the Glasgow Outcome Score upon discharge four months later.

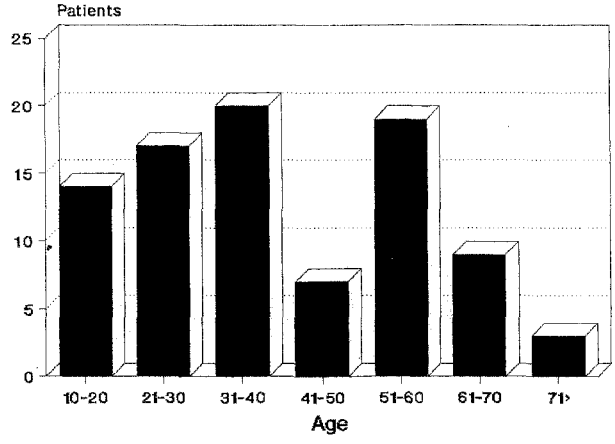

Fig. 1. Age distribution in TSAH cases

Results

Neck stiffness and other signs of SAH were present in only 29 patients. Spinal taps were performed on 11 of them and various degrees subarachnoid bleeding was demonstrated in 11 of these. SAH was revealed by CT examination in 60 patients. In three patients who suffered neck stiffness, spinal tap was negative in contrast to positive CT results.

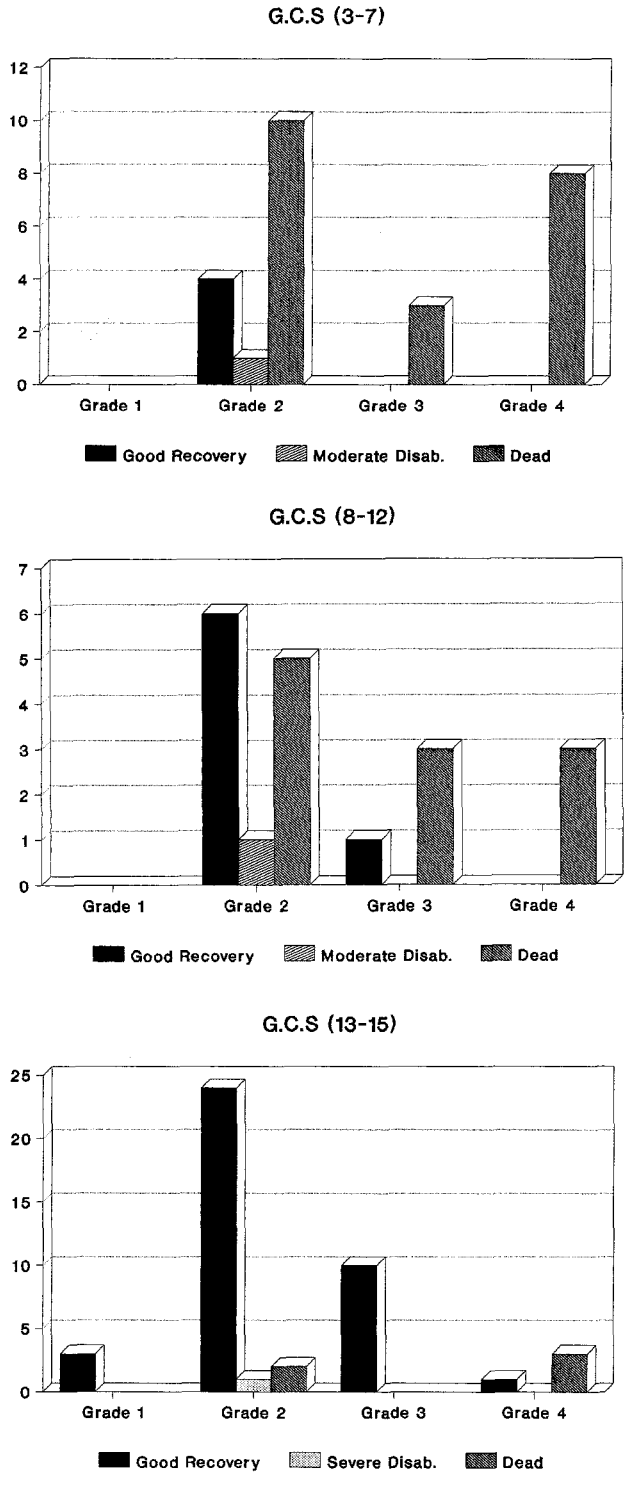

Fig. 2. The amount of subarachnoid blood (by Fisher's grading system) and prognosis in three G.C.S. groups

Visualisation of haemorrhage on CT for 89 patients was classified according to Fisher's grading system. There were three patients in group 1 (no subarachnoid blood was detected), 54 patients in group 2 (diffuse deposition or thin layer with all vertical layers of blood less than 1 mm thick), 17 patients in group 3 (localized clots and/or vertical layers of blood 1 mm or greater in thickness), 15 patients in group 4 (diffuse or no subarachnoid blood with intracerebral or intraventricular clots).

On the Glasgow Coma Score of patients in admission, the amount of subarachnoid blood which was detected by CT and the outcome are summarized in Fig. 2. It is notable that 14 of 15 patients in group 4 died ($p < 0.01$).

The other intracranial lesions accompanied by TSAH are shown in Table 1. The most frequent types of associated lesions were focal or global contusion and diffuse brain injury in accordance with previous knowledge. In 13 patients, CT imaging did not show any other lesion in the brain paranchyma and prognosis of this type of TSAH (pure TSAH) was excellent except for one patient who died ($p < 0.01$) (Fig. 3).

16 patients underwent operations because of associated intracranial lesions such as epidural, subdural, or intracerebral haematomas. All other cases were treated conservatively. In two cases who developed early hydrocephalus, external ventricular drainage was carried out, but neither of them survived. In total, 36 of 89 cases with TSAH died (mortality group).

Considering these cases, it was seen that a poor Glasgow Coma Score (less than eight), severity of SAH on CT, and evidence of accompanying lesions such as

Table 1. *Traumatic Intracranial Pathology with TSAH in 89 cases*

Pathology on CT	No. of patients	Per cent
Contusion (focal or global)	42	42%
Diffuse brain injury	15	17%
Epidural haematoma	4	5%
Contusion and epidural haematoma	3	3%
Subdural haematoma	4	5%
Intracerebral haematoma	1	1%
No other lesion	13	15%

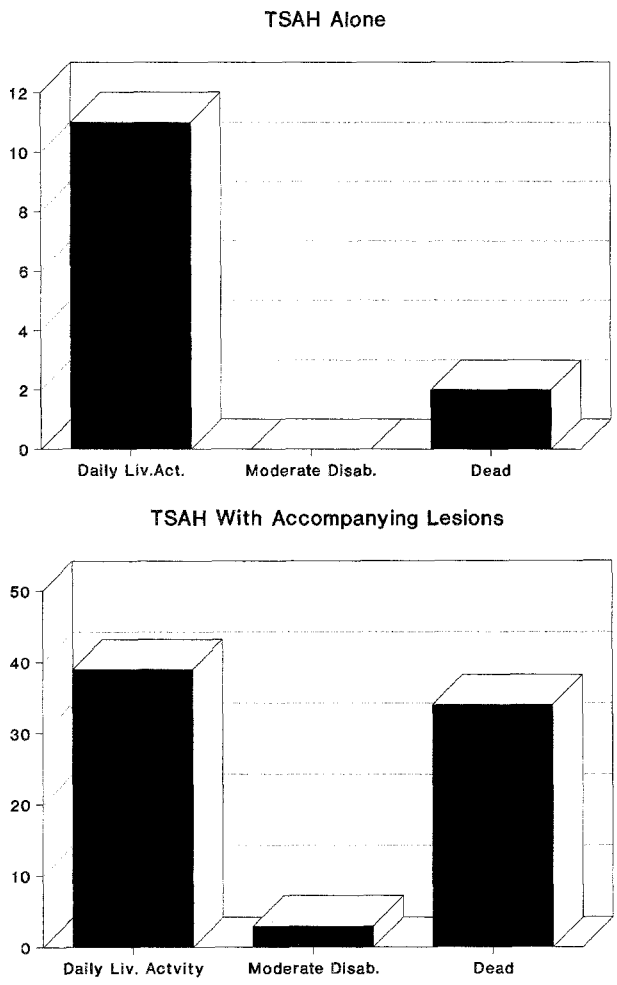

Fig. 3. Prognosis in TSAH alone and TSAH with accompanying intracranial lesions

contusion, diffuse oedema, subdural haematoma were associated with a poor prognosis. While only one of 15 patients in group 4 survived with a good outcome, all the other patients died. On the other hand, there was no mortality in group 1. There was only one death in pure TSAH cases in contrast to those with severe brain lesions. Moreover, the mortality group included three patients with secondary complications due to SAH such as hydrocephalus and vasospasm. Those three patients were in group 4. Hydrocephalus, which developed acutely, was observed in two patients. They had not responded to treatment despite external ventricular drainage. None of the surviving cases developed any clinical or radiological signs of hydrocephalus. Two patients with angiospasm were not diagnosed as vasospasm clinically, but in another patient vasospasm was obvious since a large hemispheric hypodense ischaemic area was observed on CT.

All living patients with pure TSAH returned to their routine daily living activities in the late follow-up period.

Discussion

Spontaneous SAH is one of the basic subjects which has been studied in detail in neurosurgery and many researches on its management have been carried out. However, TSAH has not been considered very interesting since it has a relatively benign nature and is frequently associated with other primary gross intracranial pathology in the majority of cases. There are only a few articles on this subject which include clinical data. It is more often seen in Journals of Forensic Medicine as case reports dealing with the origin of haemorrhage. The interest of Forensic Medicine is especially directed at finding out whether the haemorrhage is spontaneous or traumatic in fatal cases with a history of minor head injury. Frequently, the site of bleeding is from the intracranial portion of the vertebral artery [1, 3, 5, 6, 12, 13]. Momentary oscillation of the brain as a result of rotational acceleration of the head and stretching of the vertebro-basilar system in hyperextension of the head have also been implicated as possible pathogenic mechanisms of vascular tears[13]. In Simonsen's series, which is the largest on this subject[12], the origin of haemorrhage could not be demonstrated in the great majority of traumatic cases (67% on 75 cases). Simonsen has also noted the role of alcoholic intoxication in TSAH. Alcohol intake was reported in 87% of his cases. Cameron and Mant pointed out that alcohol causes dilatation of the cerebral arteries[3]. Alcohol intake was present in 20 patients (23%) in our clinical series.

Considering clinical series, Kobayashi *et al.* found 23% TSAH in acute severe head injury cases[7]. All of the their patients had an eight or less on their Glasgow Coma Score. They pointed out that TSAH was seen more frequently in patients with a lower Glasgow Coma Score. Another similar result was given by Levi *et al.*[8]. TSAH was observed in 20% of patients with diffuse axonal injury in their cases. In our series, we did not have a limit for the Glasgow Coma Score, so the frequency of TSAH was found to be as low as 4%, and this was in accordance with Levi's observations. In another study, TSAH was reported in 2% of head injury cases examined by CT[2].

Brain contusion is the most frequent associated lesion with TSAH. In our series, it occurred in 47% of the patients. It was followed by diffuse brain injury

(22%). 13 patients in which any other lesion was not shown by CT were so called pure TSAH cases and this type of haemorrhage had an excellent prognosis. However, mortality rate is significantly higher in cases with associated intracranial lesions and severe SAH visualized on CT. It is clear that SAH which fills all of the basal cisterns contributes to increasing intracranial pressure.

Otsuka *et al.* has classified their TSAH cases into two groups according to evidence of associated lesions detected by CT: Group A includes patients with SAH alone, and Group B, TSAH with concurrent intracranial lesions[10]. In the latter contusion was seen in 43% of the patients and subdural haematoma in 24%. Comparing Group A and B, they have found significantly more cases classified dead in Group B and good recovery in Group A. The same observations were found in the present study. Patients with very extensive SAH stretching from basal cisterns to cortical sulci and associated lesions had a lower Glasgow Coma Score and had a poor prognosis resulting in death. Lesions secondary to TSAH were rare in our study. While the incidence of hydrocephalus was apparently high in spontaneous SAH (20%)[9, 14], it was as low as 2% on our traumatic patients. We have not encountered hydrocephalus in the late period and this can be explained by the high mortality rate of our patients with extensive SAH.

We had only one patient with vasospasm, but we could not comment on this subject because we have not performed cerebral angiography routinely. Shigemori et al. has reported a patient with delayed vasospasm and communicating hydrocephalus[11]. This patient had massive TSAH, but a high Glasgow Coma Score on admission. Except for this case, as far as we know, hydrocephalus following TSAH has not been reported before.

The results of this study suggest that final outcome was mainly influenced by the severity of the co-existing intracranial lesions and the degree of SAH detected by CT. Generally, patients with TSAH alone will have a chance for a good prognosis. On the other hand, patients with massive SAH accompanied by other intracranial lesions will have a high mortality risk.

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