5-1990

Save a life- Diagnose subarachnoid haemorrhage!

Rashid Jooma

Iqtidar H. Bhatti

SAVE A LIFE - DIAGNOSE SUBARACHNOID HAEMORRHAGE!

Pages with reference to book, From 101 To 102

Rashid Jooma, Iqtidar H. Bhatti (Department of Neurosurgery, Jinnah Postgraduate Medical Centre, Karachi.)

Though our title may sound like a rhetorical statement, when viewed in the context of the 60 percent mortality at two years attending untreated aneurysmal subarachnoid haemorrhage (SAH) it should be considered a challenge to all practicing physicians. Spontaneous SAH arises from a ruptured berry aneurysm in the majority of cases while arteriovenous malformations (AVM) account for about 15 percent. The triad of headache, neck stiffness and blood stained CSF is certainly well known in practice but difficulties in diagnosis arise by the variations of severity in the presentation. SAH is one of the few causes of sudden death while on the other hand it may cause no more than a transient, albeit severe headache. The diagnostic considerations range from an obvious intracranial catastrophe to an attack of migraine. The important thing is that the outcome of treatment is directly related to the condition of the patient at the time of the diagnosis and a good result will only be achieved if the lesion is suspected before the patient has been devastated by an extensive haemorrhage[1]. It is usually thought that SAH is uncommon in Asia and Africa compared to the western world, in keeping with the lower incidence of deg2enarative vascular disease. Indeed, a report from Africa[2] put the annual incidence at 3.5 cases per 100,000 populations whereas the figure for the USA is 11 per 100,000[3] . Indian workers have regularly remarked on the lower than expected number of SAH patients in their experience[4]. However, this may not be entirely true and a large number of cases are probably missed. In Japan, where the diagnosed incidence of SAN is now close to being the highest in the world, the rise in the number of cases diagnosed over the past two decades has closely paralleled the expansion of health services and the number of neurologists and neurosurgeons[5]. A recent report has estimated that there are about one thousand cases of SAH occurring in Karachi annually while only four or five of these are referred for definitive specialist care[6]. This maybe overstating the case but it does make the point that SAH is a grossly neglected form of stroke in the city and viewed in the context of the mortality of untreated SAH, the magnitude of the disservice this neglect does to our patient population is apparent. As mentioned earlier there is a wide spectrum of clinical manifestations in SAH with the essence being the acute onset of signs of meningeal irritation associated with blood in the CSF. Because of the prognostic importance of the patient's condition at the time of presentation, the Hunt and Hess grading of patients is widely used with grade 1 patients having only headache while grade 5 patients are moribund with decerebrate posturing at best[7]. About 20 percent of patients present to hospital in grade 5 and these cases have a mortality of 95 percent. Grade land 2 cases account for about 50 percent of hospitalised SAN patients and have mild to severe headache but without neurological deficits apart from cranial nerve palsies. The outcome of patients in better grades can be modified by appropriate surgical therapy by reducing the risk of re-bleeding and it is to this effect that our efforts are most directed. In patients surviving an aneurysmal SAH, rebleeding results in significant mortality and morbidity if untreated. The risk of rebleeding within the first month is 33 percent, and the mortality from a rebleed approaches 75 percent. The risk of rebleeding is greatest in the second week and thereafter diminishes but does not disappear and long term survivors rebleed at an annual rate of 3.5 percent[8]. It has been stressed that in patients admitted to hospital with SAH a careful history reveals evidence of a "warning leak" in almost a third and this usually precedes the major catastrophic bleed by about two weeks[9]. This premonitory, 'sentinel" headache is abrupt in onset and even in patients who may suffer regular headaches is recognised as being unlike any previous event. Neck stiffness may be absent following a minor leak

and therefore this important event is often overlooked. A CT scan would probably be normal and a lumbar puncture (L.P.) is the only way to make the diagnosis. A high index of suspicion and an L.P. in this context will alert the physician and save the patient from what may be a major haemorrhage. The overt SAH is usually obvious with excruciating. headache, nuchal rigidity, nausea, vomiting and in upto half of the patients a transient loss of consciousness. In 10 percent of cases the bleeding may be severe enough to cause loss of consciousness for upto several days. In these instances CT scan will show free blood in the CSF space particularly at the base of brain. Blood may also be visible by fundoscopy with scattered retinal haemorrhages or more characteristically subhyaloid haemorrhage extending outward from the disc margin. If there is no evibdence of a focal haematoma and if information from CT and fundoscopy is negative or not available, an L.P. is indicated. Often the question of a traumatic tap is raised when blood stained CSF is obtained. Examination of three specimens for uniformity of the blood staining and looking for xanthochromia in the centrifuged supernatant will resolve the question. Subarachnoid haemorrhage is a serious disorder with a significant mortality and morbidity However it is also the one form A of “stroke” where surgical treatment in the selected patient will improve the outcome over the natural history of the disease. The surgery of SAH is mainly directed to obliterating aneurysms and excising AVMs so as to reduce the incidence of fatal rebleeds. The selection of patient calls for clinical examination and cerebral angiography and information from these will determine the risks and expected results of treatment. Central to this process is early diagnosis and this is where saving the life of the patient begins.

REFERENCES

1. Rapper, A.H. and Zetvsa, N. T. Outcome 1 year after SAH from cerebral aneurysm: Management morbidity, mortality, and functional status in 112 conaecutive good-risk patienta. 3. Neuroaurg., 1964; 60: 909.
2. Levy, L.E., Racbman, L and Castle, W.M. Spontaneous primary subarachnoid haemorSage in Rhodesian Africana. Mr. 3. Med. Sci., 1973; 4: 77.
3. Kassell, N.F. and Toner, J.C. Epidemiology of intracranial aneurysms. Int. AneatheaioL Qin., 1982; 20: 13.
4. Ramamurthi, B. Are intracranial aneuryama rare in some pars of the World? Surg. NeuroL, 1985; 23: 325.
5. Suzuki, J., Hod, S. and Sakurai, Y. Intracranial aneurysms in the neurological clinics in Japan. J. Neurosurg., 1971; 35 : 34.
6. Hashmi, S.S. and Bhatti, L H. Spontaneoua intracranial haemorrhage a neglected form of stroke. Oneyearatudy a Uaqat National Hospital (LNH). Amn, Jinnah Postgraduate Med. Centre, 1987; 4:9.
7. Hunt, W.E. and Hess, R.M. Surgical risk as related ro time of intervention in the repair of intracranial aneurysms. 3. Neurosurg., 19* 28: 14.
8. Winn, H. it, Richardson, A.E., O'Brien, W.M. and Jane, J.A. The long-term prognosis in untreated cerebral aneurysms IL Late morbidity and mortality. Ann. Neurol., 1978; 4:418.
9. Ball, M.J. Pathogenesia of the sentinal headache" preceding berry aneurysms rupture. Can. Med. Assoc.J., 1975; 112:78.