SPONTANEOUS SUBARACHNOID HEMORRHAGE

A Brief Review of Fifty Consecutive Cases

JOHN TUCKER, M.D.

As a rule, an attack of spontaneous subarachnoid hemorrhage is characterized by the sudden onset of severe suboccipital or parietal headache, dizziness, nausea, vomiting and somnolence or even coma. If a spinal puncture is done within the first week, one finds an even distribution of blood throughout the fluid and a definite elevation in intraspinal pressure. Unfortunately, this disaster is prone to occur in young adults, rather than in those individuals who have reached the degenerative period of life. Great importance should be attached to the proper diagnosis of the milder cases, particularly in its differentiation from migraine headaches. Recurrences of subarachnoid hemorrhage are the rule, particularly if the patient is not educated to live a quiet life, free from any physical or emotional strain. Even under the best of conditions, the patient usually dies of a second or third massive hemorrhage. While the exciting factor is mainly the increase of intravascular pressure brought about by lifting, straining at stool or emotional excitement, still a few cases in our series experienced the hemorrhage when lying quietly in bed.

Autopsy studies have shown that the vascular weakness is usually congenital in origin. At one or more points where the arteries of the circle of Willis give off branches, there may be an inadequate amount of muscle fibre in the media. As a result of this lack of strength, a small aneurysm forms which often, without any prodromal symptom, ruptures into the subarachnoid space. Unfortunately, these weak spots have no support from any surrounding tissue, so that one can never be certain that bleeding may not recur. It is true that other pathologic states are responsible for subarachnoid hemorrhage such as traumatic ruptures of thecal arteries secondary to skull fracture, infected emboli, blood dyscrasias with hemorrhagic diathiasis or intracerebral hemorrhages which communicate with a ventricle or which rupture into the subarachnoid space. As a rule, the intracerebral bleeding occurs in the later decades of life, and when it is accompanied by permanent paralysis or paresis, it constitutes the usual "stroke," while in spontaneous subarachnoid hemorrhage, any impairment in muscle power or in nerve function is usually transient.

Inasmuch as sudden hemorrhage into the subarachnoid space may occur without the dramatic picture of coma or convulsions, especially when the leakage is slight, we thought it would be worth while to review a small series of cases, especially from the standpoint of diagnosis. In this study we have considered the age of the patient, type of

onset, previous known attacks of subarachnoid hemorrhage, the spinal fluid findings, special operative procedures and, as far as possible, the end results.

In an analysis of fifty consecutive cases of spontaneous subarachnoid hemorrhage, we have found, as have most other authors, that the preponderance of instances occur in the younger age groups. While the range was from thirteen to seventy years, the average age was 41.7 years. When taken by decades, there were three in the second, five in the third, thirteen in the fourth, twelve in the fifth, twelve in the sixth, four in the seventh, and one in the eighth. In the series, thirty-three, or 66 per cent, were below fifty years of age. Studies from other sources show approximately the same average age at onset[1,2].

The sex distribution was fairly even, there being twenty-nine men and twenty-one women. We must emphasize the fact that it is a disease of young adults and early middle life.

Prodromal symptoms occurred in eighteen of our cases, existing from one month to as long as ten years. In the main, these consisted of attacks of suboccipital and frontal headaches, often associated with disturbances of vision such as blurring, diplopia or sharp ocular pains. While many of these complaints had been diagnosed "migraine," yet in some of the more severe attacks there is reason to suspect that small subarachnoid hemorrhages had occurred, with only short periods of disability. In five patients, one previous attack had been verified by the discovery of bloody spinal fluid. In one of these cases, two hemorrhages had occurred six months and eighteen months prior to the massive bleeding which had brought him to the hospital. The other four had experienced one small hemorrhage six weeks, eight weeks, eighteen months and twenty-two months previously.

The symptoms that announced the hemorrhage were quite consistently those of a sudden rise in intracranial pressure associated with meningeal irritation. In 90 per cent of the subjects, the major complaint was a sudden, severe headache, usually suboccipital and either limited to this area or associated with retrobulbar or bi-temporal pain. At times the whole skull felt as if it would burst or the sensation was that of a violent blow on the head. Associated with the headache was severe vomiting in 72 per cent of the cases and in the others there was more or less severe nausea. The onset of coma appeared suddenly or gradually in 44 per cent. All cases considered here survived this first period of unconsciousness, but as will be seen later, many had recurrences of coma at a later date, without recovery of the conscious state.

There were many other symptoms of diagnostic importance among this group of fifty cases. Paralysis or paresis of an arm, a leg, one side of the face or one side of the body appeared in 58 per cent. These

DEATHS UNDER OBSERVATION

	Age	Operation	Circumstances of Death	Post Mortem
1.	35	Encephalogram. L.F. Craniotomy for hematoma.	1 day P.O.	Hematoma L. Frontal Lobe. S.A.H. L. ant. comm. art.
2.	38	0	17th day.	0
3.	57	0	Found dead in bed.	0
4.	16	Sub. occip. craniotomy. Tumor 4th ventricle.	Post-op.	S.A.H. Medulloblastoma.
5.	53	0	6th day—during spinal puncture.	S.A.H. Aneur. R. ant. comm. art.
6.	48	0	5th day—2nd hemorrhage.	0
7.	60	0	2nd day.	0
8.	50	0	4th day—2nd hemorrhage.	S.A.H. Aneur. L. int. carotid.
9.	29	0	8th day—2nd hemorrhage.	0
10.	50	0	2nd day—2nd hemorrhage.	0
11.	31	0	10 minutes following spinal puncture.	0
12.	50	Encephalogram. Laminectomy T. 3-8. Comm. Hydroceph.	8 days P.O.	0
13.	41	Enceph. R. frontopar. hematoma. Craniotomy, muscle applied to aneurysm.	9 days P.O.	S.A.H. R. midcerebral hemorrhage.
14.	7	0	2 months—2nd hemorrhage.	0
15.	64	0	Pneumonia 20 days after hemorrhage.	S.A.H. Aneurysm R. int. carotid. Broncho-pneumonia.
16.	43	0	3rd hemorrhage, 2 days.	0
17.	48	0	4th day.	0

S.A.H. = subarachnoid hemorrhage.
L. ant. comm. art = left anterior communicating artery.

TABLE 1

findings were transient except in one case in which an intracerebral hemorrhage, which produced hemiplegia, had ruptured through the cortex into the subarachnoid space. This condition was verified by autopsy. While generalized convulsions appeared in only 18 per cent of cases, in one of these the patient continued to have recurrent grand mal attacks.

Ocular disturbances are a fairly common finding in aneurysms of the circle of Willis. In the anterior communicating artery there may be pressure on the optic chiasm while in the posterior communicating artery, pressure may be exerted on the oculomotor or abducens nerve with paralysis and diplopia. These symptoms may be intensified at the time of the hemorrhage and at times even the sheath of the optic nerve is infiltrated with blood. In 44 per cent of our cases there appeared such abnormalities as homonomous hemianopsia, quadrant defects, retinal hemorrhages, retrobulbar neuritis and extra-ocular palsies. The ophthalmic examination was often unsatisfactory due to poor cooperation by the patient.

Elevated blood pressure was not an outstanding finding. It was raised in only 12 per cent and in these the range was between 160 systolic, 70 diastolic and 170 systolic, 120 diastolic. One-half of these hypertensive cases died while under observation. Inasmuch as spontaneous subarachnoid hemorrhage is due principally to the simple rupture of a weak spot in an artery of the circle of Willis, the degree of intravascular pressure may determine the time of hemorrhage. While persistent arterial hypertension is relatively infrequent in this disease, yet the effects of exertion are important. In twenty-nine cases, an accurate story was obtained of the precise activities at the time of the vascular accident. In thirteen, or 26 per cent, the patient was under physical strain such as heavy lifting, while in sixteen, or 32 per cent, the patient was at rest, either standing quietly, sitting or lying down. Obviously, the factor of effort only hastened the hemorrhage and yet, if life can be prolonged by mental and physical quietude, a rigid limitation of activity is well worth while.

The essential diagnostic procedure in spontaneous subarachnoid hemorrhage is the lumbar puncture. One or more spinal taps were made in all fifty cases. In 76 per cent the fluid was grossly bloody, and in these the pressure ranged between 150 and 700 mm. of water. The average pressure in this group was 364 mm. of water. In 18 per cent the fluid was xanthochromic and in these the pressure varied between 80 and 350 mm. water. In the three remaining cases the fluid was clear and colorless with pressures of 110, 120, and 205, respectively. If we accept the upper limit of normal pressure at 150 mm. to 200 mm. water, we find that, with few exceptions, the intraspinal pressure is raised. The hemorrhagic fluids were taken within the first three days of the illness, while

those of yellow color were secured five to eight days after bleeding had occurred. The colorless fluids were obtained ten days to three weeks after the onset.

Careful investigation has shown that xanthochromia begins three to four hours after a subarachnoid hemorrhage occurs and that the fluid is clear and colorless in twenty days, even in cases of large hemorrhage[3]. According to Brain[1], xanthochromia begins in a few hours, the yellow color reaches its greatest intensity in about a week and becomes colorless in fourteen to twenty-one days. He believes that the red blood cells disappear from the subarachnoid fluid in two to three days. In our experience, the time interval was somewhat longer. However, these observations emphasize the importance of an early diagnostic lumbar puncture.

If at this point we summarize the symptoms and physical signs characteristic of spontaneous subarachnoid hemorrhage, the picture is about as follows: If a man or woman, under forty-five years of age, who was previously in good health suddenly becomes dizzy, develops a severe suboccipital or frontal headache, vomits, collapses, is carried to bed in a stupor, one should suspect the advent of an acute subarachnoid hemorrhage. A bloody spinal fluid under high pressure, as recorded by a manometer, completes the diagnosis. Unfortunately, however, the syndrome is not always so clear cut. For instance, the patient may not have been in good health prior to the accident. Perhaps headaches, visual disturbances or neurasthenic symptoms have been troublesome. In such instances the bloody spinal fluid may be due to a bleeding intracranial tumor or a rare case of hemorrhagic encephalitis. Likewise, if the amount of blood is small, if the neck is quite stiff, or a positive Kernig is present and high fever develops rapidly we may be dealing with a case of true meningitis. These possible diagnostic pitfalls make it necessary for us to have adequate laboratory facilities. Likewise various other diagnostic procedures may be required such as plain roentgenograms of the skull for possible intracerebral calcification, erosion of the clinoid processes of the sella or other evidences of vascular or neoplastic lesions.

In four of our series of cases an encephalogram was done even in the presence of bloody spinal fluid and the patients survived the ordeal. In one case, a tumor of the fourth ventricle was present in addition to the subarachnoid hemorrhage.

The treatment of this group of fifty cases consisted largely of symptomatic care with the maintenance of good comfort, nutrition and adequate fluids, together with repetition of the spinal punctures as often as necessary to keep the intracranial pressure below 150 mm. of water. The spinal taps were made with a full realization of the danger of the procedure. In only two cases we are reasonably certain that death resulted

from the spinal puncture. (See table 1.) One patient ceased respirations during the withdrawal of spinal fluid and the other died about ten minutes after this procedure. Therefore, in view of this hazard, it is necessary to reduce the pressure slowly and as often as twice a day in cases of rapid, massive bleeding. The risk is much greater if this procedure is neglected.

While the ultimate prognosis in cases of spontaneous subarachnoid hemorrhage is most discouraging, the only hope for the prolongation of life lies in early diagnosis, attempts to arrest bleeding by rest and sedatives and the control of intracerebral pressure by repeated spinal punctures. Dr. W. James Gardner, of the neurosurgical service, has in two cases attempted to strengthen the weakened spot in the artery at the base of the brain by encircling it with a bit of temporal muscle. However, in both instances the patient expired later during a second hemorrhage. Venesection of several hundred cc. of blood has also failed to correct continued subarachnoid bleeding. In general, simple medical measures have been more satisfactory than the use of the more complicated procedures.

References

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