Unoperated subdural hematomas

Long-term follow-up study by brain scan and electroencephalography

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The authors report nine patients selected from over 100 patients with subdural hematomas successfully treated without surgery. These patients were followed for as long as 5 years. All had angiographically demonstrated subdural hematomas. Electroencephalograms (EEG) documented well the clinical improvement of the patient, but were poor guides to the true size of the hematoma, since EEG returns to normal early in the patient's course. Static scans are a better guide to the presence of a subdural hematoma, but they lag behind clinical improvement and usually remain abnormal for considerable periods of time after a major portion of the hematoma has been reabsorbed, and the patient is asymptomatic.

KEY WORDS • subdural hematomas • unoperated subdural hematomas • brain scan • electroencephalography • follow-up studies

It has been demonstrated that many cases of chronic and subacute subdural hematoma can be successfully treated nonsurgically. With increasing recognition of this fact, there has been a corresponding increase in the need for nonsurgical methods for the diagnosis and evaluation of this condition. Clinical-laboratory correlations must be studied in each case so that prognosis and management can be rationally determined. Radionuclide scanning and electroencephalography (EEG) are methods which can provide valuable information without risk to the patient. To assess their value, we studied the data obtained by these methods in correlation with clinical information and radiographic examinations performed in a series of cases observed over a prolonged period without surgery.

Clinical Material

Since 1958 over 100 patients with subdural hematoma have been successfully treated without surgery at The Mount Sinai Hospital and affiliated institutions. A previous report analyzed the significance of serial EEG studies in data derived from 21 of these cases. Increasing use of radionuclide scanning, using both "static" and "dynamic" techniques, has enabled us to add information
TABLE 1

Summary of the clinical course in nine subdural hematoma patients*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>History, Findings</th>
<th>Course</th>
<th>EEG Findings</th>
<th>Brain Scan Findings</th>
<th>Follow-Up Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>79</td>
<td>trauma-memory defect, headache, gait disturbance, 6 weeks in hospital; gradual improvement; mild OMS on discharge</td>
<td>4/3/70 depressed alpha, lt parieto-occipital, lt temp bursts 1.5-3 cps</td>
<td>4/3/70 lt-sided crescent</td>
<td>Oct '72 no OMS no focal findings EEG normal scan normal</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>no trauma; gradual onset rt hemiparesis; memory disturbance 3 weeks in hospital; rapid improvement; no weakness at discharge</td>
<td>3/24/69 shifting temporal 2.5-4 cps bursts, much more on lt side; diffuse irregular theta, lt greater than rt</td>
<td>3/23/69 lt-sided crescent</td>
<td>March '73 working; exam negative EEG normal scan normal</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>51</td>
<td>trauma; rapid onset lt-sided weakness and lethargy, confusion 7/21-7/25/61; improvement by admission on 8/4/61; papilledema 4 weeks in hospital; no deficit on discharge</td>
<td>8/5/71 normal</td>
<td>8/6/71 rt-sided crescent</td>
<td>August '73 working; exam negative EEG normal scan normal</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>alcoholic ? trauma dragging rt leg 5 weeks in hospital; rapid progression to coma; treated with steroids; improved in 1 wk; no findings on discharge</td>
<td>6/22/69 severe diffuse irregular slowing, strongly accentuated on lt side 10/30/69 normal</td>
<td>6/25/69 lt-sided crescent 7/17/69 no change</td>
<td>August '72 alcoholic; some falling; EEG normal; scan minimally positive on lt; angiogram normal</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>47</td>
<td>recent assault with head injury; rt-sided headaches 4 weeks in hospital; improved; negative status on discharge</td>
<td>11/19/68 normal 11/27/68 diffuse irregular slowing, strongly accentuated, rt temporal 12/4/68 rt temporal theta bursts 12/16/68 minimal irregular theta on lt 12/31/68 no change</td>
<td>11/26/74 rt-sided crescent 12/18/74 no change</td>
<td>Oct. '72 working exam negative EEG normal scan normal</td>
<td></td>
</tr>
</tbody>
</table>

obtained by this method in patients with unoperated subdural hematoma followed over periods up to 5 years.

The 11 patients in this study all had angiographically proved subdural hematoma. The patients were selected from the much larger group because they had multiple scans and electroencephalograms during their hospital stay, and because they were accessible to follow-up scanning and electroencephalography. All patients had repeat angiography before discharge and four had angiography at later dates. Nine patients were not operated on; two patients underwent surgery and are
TABLE 1 (Continued)

<table>
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<tr>
<th>Case No.</th>
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<th>Brain Scan Findings</th>
<th>Follow-Up Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>75</td>
<td>fell, possible head injury; headaches; dragging left foot, rapid progression to left hemiplegia</td>
<td>5 weeks in hospital; gradual improvement; no focal weakness; mild organic mental syndrome on discharge</td>
<td>1/12/71 bursts and runs of paroxysmal 1.5-4 cps waves, right temporal; diffuse irregular theta, right greater than left</td>
<td>1/13/70 right-sided crescent</td>
<td>March '73, no complaints or findings EEG normal scan normal</td>
</tr>
<tr>
<td>7</td>
<td>60</td>
<td>trauma; headache, right-sided weakness, aphasia</td>
<td>5 weeks in hospital; gradual improvement, negative status on discharge</td>
<td>6/20/72 mildly abnormal, diffuse irregular theta, accentuated on left</td>
<td>7/5/72 marked left-sided crescent</td>
<td>Feb. '73, working exam negative EEG normal scan normal</td>
</tr>
<tr>
<td>8</td>
<td>71</td>
<td>trauma; gradual onset left leg weakness and lethargy</td>
<td>10 weeks in hospital; fluctuating course with gradual improvement; no focal signs, mild organic mental syndrome on discharge</td>
<td>11/1/67 normal</td>
<td>11/6/67 very pronounced right-sided crescent</td>
<td>Nov '69, convulsive seizure; exam negative EEG, right-sided 6-7 cps bursts, scan normal angiogram negative</td>
</tr>
<tr>
<td>9a</td>
<td>70</td>
<td>recent head injury; headaches, weakness; right hand progressing to right hemiparesis</td>
<td>6 weeks in hospital; worse at first, then slowly clearing; no focal signs, minimal organic mental syndrome on discharge</td>
<td>2/27/67 marked left-sided delta</td>
<td>3/6/67 left-sided crescent</td>
<td>Dec '72, no complaints; status negative EEG normal scan normal</td>
</tr>
<tr>
<td>9b</td>
<td>no trauma; headache, organic mental syndrome, left-sided weakness</td>
<td>5 weeks in hospital; gradual improvement; no deficit on discharge</td>
<td>5/5/70 right temporal 1.5-3 cps bursts</td>
<td>5/4/70 right-sided crescent</td>
<td>9/20/70 no change</td>
<td></td>
</tr>
</tbody>
</table>

*OMS = organic mental syndrome.

The history, prominent signs, course, and outcome in the patients studied are summarized in Table 1. Nine of the patients had a history of head injury from 2 to 4 weeks prior to admission. Nine had significant lateralizing signs, and seven had evidence of depressed level of consciousness. The duration of hospital stay ranged from 3 to 10 weeks, and all patients improved. Six of the non-surgically treated patients received parenteral and oral steroids. The others did not.
Fig. 1. Case 6. Sequential $^{99m}$Tc brain scans performed over a 3-year period. Left: Initial scan performed at the time of the angiographic diagnosis of subdural hematoma, shows a large right-sided crescent-shaped area of increased activity on the side of the subdural. Center: Follow-up scan performed 3 months later shows a right-sided crescent of lesser intensity. Right: Repeat scan 3 years later shows that the two sides are now normal.

Fig. 2. Case 4. Left: Arterial phase of the initial left carotid angiogram shows a significant left-to-right shift of the anterior cerebral artery (large arrows), and a large concave defect on the left outlining a subdural hematoma (small arrows). Center and Right: Repeat angiogram 3 years later performed through an aortic arch injection shows in the arterial phase (center) that the midline shift noted in the first arteriogram is no longer present (large arrows). Also branches of the middle cerebral artery can be traced to the inner table of the skull indicating that the subdural collection is no longer present. The venous phase (right) of the same study confirms the resolution of the hematoma.

Periodic reevaluations were carried out, with an average follow-up period of 4 years. Eight patients had uneventful courses. The five who had been employed returned to their prior occupations. One patient (Case 9) was readmitted 3 years after his first hospital stay, with a subdural hematoma on the opposite side. This was treated nonsurgically and he has done well. Another patient (Case 4) has had a course complicated by chronic alcoholism and a moderate organic mental syndrome. One patient (Case 8) developed a convulsive disorder.

Brain Scan and EEG Results

At the time of admission, all patients had brain scans showing abnormal uptake on the same side as the subdural hematoma. The abnormality was in the form of a "crescent sign" on the anterior view. All except one had abnormal EEG's that showed dysfunction on the side of the hematoma, as manifested by marked unilateral slowing, with some evidence of underlying diffuse dysfunction. Repeat studies performed 3 to 6 weeks later showed that, although the brain scan was
FIG. 3. Case 9. Serial scans showing the initial subdural hematoma on the left, followed by a subdural hematoma on the right 3 years later. A) Anterior view of a 99mTc-scan showing a left-sided area of increased activity in the form of a crescent. This is on the same side as the angiographically proved subdural collection. B) Follow-up scan 4 months later shows that the abnormality is still present. C) Scan performed 3 years later shows the increased activity now on the right side. Angiogram performed at this time showed that the subdural on the left had completely resolved and that there was a new subdural collection on the right. D) Repeat study 4 months later shows decrease in the intensity of the abnormality. E) Repeat scan 2 years later shows that the scan has returned to normal.

positive in all cases, the EEG was normal in seven cases and showed distinct improvement in two. All patients were markedly improved clinically, most having only minimal dysfunction. The repeat angiograms showed that the subdural collections were definitely smaller, but none had resolved completely at that time.

Late follow-up studies were done after an interval of 3 to 4 years (Fig. 1). The EEG's were normal in all patients. The brain scans had returned to normal in all except one (Case 4), who had a small amount of increased activity on the side that previously had shown a large area of increased uptake. Repeat angiogram in this case showed that the subdural collection had completely resolved (Fig. 2).

One patient (Case 9) developed another subdural hematoma on the opposite side after an interval of 3 years. The scan at this time was negative on the side of the earlier lesion, and positive on the side of the fresh one. Electroencephalography also showed that the abnormality was now on the side of the new lesion. Angiography indicated that the first
Fig. 4. Anterior 99mTc scan of patient with operated subdural hematoma. Scan is positive on the same side as that from which the hematoma was removed 5 years earlier.

collection was completely gone and revealed a large subdural hematoma on the opposite side. He was treated conservatively and 3 years later a brain scan and an EEG were both negative (Fig. 3).

The patient who developed a seizure disorder (Case 8) also had repeat angiography. No hematoma was found. One patient operated on for a left subdural hematoma did not have a preoperative scan or EEG. The other operated patient had a preoperative scan only. One individual who was operated on was rescanned 5 years later, and the scan was distinctly positive over the area of the previous subdural hematoma (Fig. 4). An angiogram, however, showed no mass lesion to be present. The second patient operated on was rescanned at 1 month and again at 8 months and the scans were positive.

Discussion

The use of the brain scan in the diagnosis of subdural hematoma has been well established since Peyton, et al., first demonstrated a subdural hematoma with the use of diiodo-fluorescein. A number of radionuclides have been used; the most common is mercury-197 or 203 chlormerodrin, radioiodinated human serum albumin, and most recently, technetium-99m pertechnetate. A review of a number of series dealing with subdural hematoma shows that there is an overall diagnostic accuracy of about 80% to 85%. It is uncertain why the remaining 15% to 20% of the patients have negative brain scans. A percentage of the scans is negative because the hematoma is bilateral and the resultant symmetry of the isotope uptake leads to an interpretation of normality. Some hematomas may be large enough to be demonstrated angiographically, but are too small to be visualized with certainty by scanning. The most important causative factor in the false negative scan may be related to the duration of the lesion. Data in the literature indicate that the chance for a positive scan increases as the age of the subdural hematoma increases. For example, Cowan, et al., report 91% accuracy in lesions over 10 days' duration and only 50% accuracy in lesions of lesser duration.

No prior reports in the literature deal with the outcome of the positive brain scan if the patient does not undergo surgery. In operated patients, the scan may remain positive for months or years, as in our two operated patients. The positivity of the scan may be attributed to residual membranes or to scar tissue. We are not aware of a study where patients operated on for subdural have been compared postoperatively to determine if those who had residual membranes differed in terms of scan results from those who did not. In one of our operated patients, the follow-up scan at 9 months was still positive even though membranes had been removed at surgery.

As our study shows, the scan in the non-operated patients gradually returns to normal. Since there has been no surgery, we are not sure what becomes of the subdural membrane after the fluid is reabsorbed. However, it is apparent that if the subdural membrane persists, its ability to concentrate radionuclide is minimal once the subdural fluid is absorbed.

The electrical abnormalities in this series were similar to those reported earlier. There was no characteristic finding, but most of the patients showed slow waves in the theta and delta ranges on the side of the lesion, or bilaterally with accentuation on the affected side. Although some of the patients showed asymmetry of the alpha activity, none showed the “suppression” of alpha which was at one time considered diagnostic of subdural hematoma.
Brain scan and EEG in unoperated subdural hematomas

The relatively rapid clinical improvement seen in this group of unoperated cases is consistent with that found in our overall series. The clinical improvement was paralleled by an equally rapid improvement in the EEG. In the early follow-up studies, seven of the nine patients showed completely normal EEG's (Table 1). All of the EEG's were normal at the time of later follow-up.

The very long periods of follow-up observation in this study indicate that the spontaneous resolution of the subdural collection is usually complete and permanent, and that there is usually no residual clinical or EEG evidence of cerebral dysfunction.

Conclusions

We have presented evidence based on serial brain scan and EEG studies performed on nine unoperated subdural hematoma patients followed over a period of 3 to 5 years. The results of this study indicate that the EEG is a more reliable indicator and correlator of clinical dysfunction in the presence of a subdural collection than is the radiographic appearance of the lesion. Our study also shows that radionuclide scanning may be a reliable indicator of the physical presence of the subdural collection as well as a practical method for long-term follow-up of the resolution of the hematoma. The scan, however, is no more reliable than the angiogram in indicating whether the lesion is likely to be causing clinical evidence of cerebral dysfunction. If criteria related to the degree of clinical dysfunction are to be used in determining whether the patient is to be operated on, it is apparent that the EEG is the more significant laboratory method in the management of those cases once the diagnosis has been confirmed.

References


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