Longterm Follow-up on Patients with Subdural Effusions

by

TASLIM S. SUTOMENGGOLO, SOFIAN ISMAEL, S.K. HENDARTO, and S.M. LUMBANTOBING

(Departments of Child Health and Neurology, Medical School, University of Indonesia, Jakarta)

Abstract

Seventy-two patients with subdural effusions who had been hospitalized in the Department of Child Health, Dr. Cipto Mangunkusumo General Hospital, Jakarta, were followed up for at least one year. Of the 72 patients, 35 were included in this evaluation. Of the 35 patients, 16 were caused by purulent meningitis, and 19 by other diseases. On follow up examination comparison was made between the prognosis of patients with subdural effusions which dried in less than 2 weeks and subdural effusions which dried in more than 2 weeks or the patient who was asked to be discharged before the subdural effusions dried. The conclusion is that the former group has less neurological deficit, epileptic seizures, motoric and mental retardation than the latter. The prognosis is worse in patients with subdural effusions of long duration.

Received 11th March 1977.
Introduction

Subdural effusions are collections of fluid in the subdural space, characterized by an aspirated volume of more than 2 ml. at any time, a protein concentration of at least 40 mg./100 ml. higher than that in the concomitantly sampled lumbar subarachnoid fluid, and a red blood cell count ranging from a few to no more than one million/ml. (Rabe, 1967).

According to Epstein and Goldzier (1953) and Lagos and Siekert (1969), usually a membrane was formed from 2 or 3 weeks after the existence of fluid in the subdural space. This membrane may serve as a constrictive agent and could prevent further growth of the brain, thus accounting for severe and progressive retardation of motoric and intellectual development (Ingraham and Matson, 1954; Benson et al., 1960). Moreover, these thick heavily vascularized neomembranes intimately applied to the brain may at times extend small capillary roots into the cerebral substance, thus performing meningocerebral cicatrices, responsible for the later development of convulsive seizures. The aim of this paper is to evaluate the prognosis of patients with subdural effusions in the Department of Child Health, Dr. Cipto Mangunkusumo General Hospital, Jakarta. Follow up examinations were done since 1968 until 1974. We used the criteria for subdural effusions as started by Rabe (1967). Follow up examinations were performed on neurological deficit, electroencephalography (EEG), motoric and intellectual development, and complication of epileptic seizures. The minimal duration of follow up examination was one year.

On follow up examination we divided the patients into 2 groups. We called patients with subdural effusions who improved during 2 weeks subdural taps as group A, and group B for patients with subdural effusions improved after 2 weeks, and patients who asked to be discharged before the subdural effusions got dry. We also compared follow up examination of patients with subdural effusions due to purulent meningitis and other diseases.

Results

Of the 72 patients, 35 were included in this evaluation. Thirty-seven patients were excluded due to lack of follow up examination. The follow up examinations of these 37 patients were less than one year. Of the 35 patients, 22 were males and 13 females. Of the 35 patients, 16 were caused by purulent meningitis and 19 by other diseases. Of the 35 patients 12 were included in group A and 23 group B. Neurological deficit in group A after discharge consisted of spastic hemiplegia in 4 patients (33.3%), and paresis of the VIIth cranial nerve...
in one patient (Table 1). On follow up examination all the patients became normal. Group B consisted of spastic hemiplegia in 3 patients (13%), spastic tetraplegia in 5 (21.7%), and paresis of the VIIth cranial nerve in one patient. On follow up examination 1 patient with facial nerve paresis became normal, but 1 patient without neurological deficit became spastic tetraplegia (Table 1).

Motoric development of the 35 patients can be seen in Table 2. Five patients (41.7%) in group A suffered from motoric retardation after discharge, and on follow up examination only 1 patient became normal. While in group B 14 patients (60.9%) suffered from motoric retardation, and on follow up examination also only 1 patient became normal.

**TABLE 1: Neurological deficit in groups A and B**

| Neurological deficit | Group A | Group B |
|----------------------|---------|---------|
|                      | After discharge | Follow up | After discharge | Follow up |
| Spastic hemiplegia   | 4 (33.3%) | —        | 3 (13%)    | 3 (15%)   |
| Spastic tetraplegia  | —       | —        | 5 (21.7%) | 6 (26%)   |
| Facial paresis       | 1 (8.3%) | —        | 1 (4.3%)  | —         |
| Normal               | 7 (58.3%) | 12 (100%) | 14 (61%)  | 14 (61%)  |
| Total                | 12 (100%) | 12 (100%) | 23 (100%) | 23 (100%) |

**TABLE 2: Motoric development of the 35 patients in groups A and B**

| Motoric development | Group A | Group B |
|---------------------|---------|---------|
|                     | After discharge | Follow up | After discharge | Follow up |
| Motoric retardation | 5 (41.7%) | 4 (33.3%) | 14 (60.9%) | 13 (56.5%) |
| Normal              | 7 (58.3%) | 8 (66.7%) | 9 (39.1%)  | 10 (43.5%) |
| Total               | 12 (100%) | 12 (100%) | 23 (100%)  | 23 (100%)  |
Table 3 shows the EEG of the patients in groups A and B. It is a pity that not in all patients could the EEG be performed. In group A after discharge only in 10 patients could the EEG be performed, and on follow up examination only in 8 patients. In group B the EEG could be performed in 17 patients after discharge, and on follow up examination in 13 patients. Of the 10 patients in group A after discharge, only one showed EEG with irritative foci, and on follow up examination 2 showed EEG with irritative foci; while in group B of the 17 patients 3 revealed EEG with irritative foci, and on follow up examination the irritative foci in the EEG could be found in 7 patients.

The complications of epileptic seizures and mental retardation are shown in Table 4. In group A only 1 patient suffered from epileptic seizures, while in group B 10 patients. Three patients in group A suffered from mental retardation, while in group B 12 patients.

### TABLE 3: The EEG of patients in groups A and B

| E. E. G.        | **Group A** |  | **Group B** |  |
|-----------------|-------------|---|-------------|---|
|                 | After discharge | Follow up | After discharge | Follow up |
| Normal          | 6 (60%)      | 3 (37.5%)  | 7 (41%)      | 4 (31%)    |
| Borderline normal | 1 (10%)     | 1 (12.5%)  | 1 (6%)      | —          |
| Borderline abnormal | —         | —         | —          | 1 (7.5%)  |
| Disrythmia      | 2 (20%)      | 2 (25%)    | 6 (35%)     | 1 (7.5%)   |
| Irritative foci | 1 (10%)      | 2 (25%)    | 3 (18%)     | 7 (54%)    |
| **Total**       | 10 (100%)    | 8 (100%)   | 17 (100%)   | 13 (100%)  |

### TABLE 4: Epileptic seizures and mental retardation in groups A and B

| Group | Total patients | Epileptic seizures | Mental retardation |
|-------|----------------|---------------------|--------------------|
| A     | 12             | 1 (8.3%)            | 3 (25%)            |
| B     | 23             | 10 (43.5%)          | 12 (52%)           |
Table 5 shows the psychological evaluation of 10 patients in groups A and B. It is a pity that only in 10 patients could the intellectual state be evaluated. Of the 5 patients in group A 2 were mentally retarded, while in group B mental retardation was found in 3 patients.

Neurological deficit in patients with subdural effusions due to purulent meningitis and other diseases is revealed in Table 6. Of the 16 patients due to purulent meningitis, after discharge 5 had spastic hemiplegia, 3 had spastic tetraplegia, and 2 had paresis of the VIIth cranial nerve. On follow up examination 3 patients had spastic hemiplegia and 2 had spastic tetraplegia. In 19 patients due to other diseases 2 had spastic hemiplegia and 2 had spastic tetraplegia. On follow up examination only spastic tetraplegia was found in 4 patients.

TABLE 5: Psychological evaluations of 10 patients

| Group | Mental retardation | Normal | Total |
|-------|--------------------|--------|-------|
| A     | 2                  | 3      | 5     |
| B     | 3                  | 2      | 5     |

TABLE 6: Neurological deficit in patients with subdural effusions due to purulent meningitis and other diseases

| Neurological deficit | Purulent meningitis | Other diseases |
|----------------------|---------------------|----------------|
|                      | After discharge | Follow up | After discharge | Follow up |
| Spastic hemiplegia   | 5 (31.5%) | 3 (18.7%) | 2 (10.5%) | — |
| Spastic tetraplegia  | 3 (18.7%) | 2 (12.5%) | 2 (10.5%) | 4 (21%) |
| Facial paresis       | 2 (12.5%) | — | — | — |
| Normal               | 6 (37.5%) | 11 (68.8%) | 15 (79%) | 15 (79%) |
| Total                | 16 (100%) | 16 (100%) | 19 (100%) | 19 (100%) |
Motoric development of the 35 patients with subdural effusions due to purulent meningitis and other diseases can be seen in Table 7. Ten of the 16 patients with subdural effusions due to purulent meningitis suffered from motoric retardation, and there was no improvement on the follow up examination. While of 19 patients with subdural effusions due to other diseases, 9 patients suffered from motoric retardation and 2 others became normal on follow up examination.

Table 8 shows the EEG of patients with subdural effusions due to purulent meningitis and other diseases. After discharge only one of the 13 patients with subdural effusions due to purulent meningitis showed irritative foci in the EEG, and on follow up examination irritative foci in the EEG could be found in 4 of the 12 patients. While in patients with subdural effusions due to other diseases 3 of the 14 patients showed irritative foci in their EEG, and on the follow up examination irritative foci in the EEG could be found in 5 out of 9 patients.

**TABLE 7: Motoric development of the 35 patients with subdural effusion due to purulent meningitis and other diseases**

| Motoric development | Purulent meningitis | Other diseases |
|---------------------|---------------------|---------------|
|                     | After discharge | Follow up  | After discharge | Follow up |
| Motoric retardation | 10 (62.5%)      | 10 (62.5%)  | 9 (47.4%)      | 7 (36.8%) |
| Normal              | 6 (37.5%)       | 6 (37.5%)   | 10 (52.6%)     | 12 (63.2%)|
| Total               | 16 (100%)       | 16 (100%)   | 19 (100%)      | 19 (100%) |

**TABLE 8: The EEG of patients with subdural effusions due to purulent meningitis and other diseases**

| E. E. G.       | Purulent meningitis | Other diseases |
|----------------|---------------------|---------------|
|                 | After discharge | Follow up  | After discharge | Follow up |
| Normal          | 2 (15.4%)        | 4 (33.3%)  | 11 (78.6%)      | 3 (33.3%) |
| Disrythmia      | 2 (15.4%)        | —          | —                | 1 (11.1%) |
| Borderline normal | —            | 1 (8.3%)   | —                | —        |
| Borderline abnormal | 8 (61.5%)  | 3 (25.1%)  | 3 (21.4%)       | 5 (55.6%)|
| Irritative foci | 1 (7.7%)         | 4 (33.3%)  | 3 (21.4%)       | 5 (55.6%)|
| Total           | 13 (100%)        | 12 (100%)  | 14 (100%)       | 9 (100%) |
The complications of epileptic seizures and mental retardation are shown in Table 9. In 16 patients with subdural effusions due to purulent meningitis 8 had epileptic seizures and 9 were mentally retarded. In 19 patients with subdural effusions due to other diseases only 3 had epileptic seizures, and 6 were mentally retarded.

Table 10 shows the psychological evaluation on 10 patients with subdural effusions due to purulent meningitis and other diseases. Mental retardation was found in 2 of the 3 patients with subdural effusions due to purulent meningitis, and in 3 patients due to other diseases.

### TABLE 9: The complications of epileptic seizures and mental retardation in patients with subdural effusions due to purulent meningitis and other diseases

| Cause                  | Total | Epileptic seizures | Mental retardation |
|------------------------|-------|--------------------|--------------------|
| Purulent meningitis    | 16    | 8 (50 %)           | 9 (56.2%)          |
| Other diseases         | 19    | 3 (15.8%)          | 6 (31.6%)          |

### TABLE 10: Psychological evaluation on 10 patients

| Cause                  | Mental retardation | Normal | Total |
|------------------------|--------------------|--------|-------|
| Purulent meningitis    | 2                  | 1      | 3     |
| Other diseases         | 3                  | 4      | 7     |

**Discussion**

Table 1 shows the difference between groups A and B in neurological deficit. Five patients in group A suffered from neurological deficit after discharge, but on follow up examination all the neurological deficit disappeared. While in group B out of 8 patients with neurological deficit only 1 recovered, and 1 patient without neurological deficit after discharge got spastic tetraplegia on follow up examination.

Thus, the neurological condition was worse in patients with subdural effusions of long duration. Benson et al. (1960) found that 78% of those in whom the duration of effusion was less than 15 days appeared normal at the time of follow up, but of those with duration of effusion of more than 15
days only 58% appeared to have escaped sequelae. The prognosis was also influenced by the size of the effusion (Benson et al., 1960). He also stated that the prognosis of patients with subdural effusion that took less than 15 days to dry was better than those that took more than 15 days to dry.

As a result of this finding we performed subdural taps for 2 weeks. If fluid still remained present, we consulted the patients to the Department of Neurosurgery (Sutomenggolo et al., 1974).

The motoric development, intellectual development, and epileptic seizures appear in Tables 2 and 4. In group A 33.3% of the patients are retarded in their motoric development, while in group B 60.9%. Mental retardation in group A is 25%, while in group B it is 52.1%. Epileptic seizures in group A are 8.3%, while in group B they are 43.5%. From these data we may conclude that the prognosis of patients in group B is worse than in group A. Ingraham and Matson (1949) found 20% of their cases suffering from mental retardation. If we compare our cases with those of Ingraham and Matson (1949), our figure of mental retardation is rather high. It may be that the normal patients did not come on follow up examination.

The high complication of epileptic seizures in group B (43.5%) may be due to a new membrane formation. This membrane contains many new capillaries with roots extending into the cerebral substance, thus performing meningoencephalocerebral cicatrices which are responsible for the development of convulsive seizures.

The EEG can be seen in Table 3. Unfortunately, in only a small part of these patients could their EEG be examined. This was due to a lack of understanding from the parents of the patients. The difference of the EEG of the patients in groups A and B is very clear. On follow up examination we found 25% irritative foci in the EEG of group A, while in group B we found 54%. This figure is very high compared with the findings of Benson et al. (1960). They found abnormal EEG in 30% of their cases, while our overall abnormal EEG were 43% (9 out of 21 patients in groups A and B on follow up examination).

The mortality occurred only in 1 patient of group B. Benson et al. (1960) also found the mortality in 1 patient with subdural effusion of more than 15 days duration. Table 6 to 9 show the difference of sequelae of patients with subdural effusions due to purulent meningitis and other diseases. From these data we can conclude that the prognosis of subdural effusion due to purulent meningitis is worse than to other diseases.

This condition is quite different from the findings of Benson et al. (1960). They stated that the prognosis of subdural effusions due to purulent meningitis was better than due to other diseases. This may be caused by the late hospitalization of our patients with purulent
FOLLOW UP SUBDURAL EFFUSIONS

meningitis. Usually they came to our hospital in a severe condition.

Acknowledgement

The authors are very grateful to the staff of the Child Guidance Clinic, Faculty of Psychology, University of Indonesia, Jakarta, for their psychological evaluation on some of our cases.

REFERENCES

1. BENSON, P.H.; NYHAN, W.I. and SHIMIZU, H.: The prognosis of subdural effusions complicating pyogenic meningitis. Pediatr. 57 : 640 (1960).

2. EPSTEIN, J.A. and GOLDZIER, S.E. II: Bilateral encapsulated subdural effusion complicating bacterial meningitis in infancy. Arch. Neurol. Psychiatr. 69 : 242 (1953).

3. INGRAHAM, F.D. and MATSON, D.D.: — Subdural haematoma in infancy. Adv. Pediatr. 4 : (1949).
— Subdural haematoma in Neurosurgery of infancy and childhood. 3rd ed., p. 186 (Charles C. Thomas, Springfield 1954). Cited by Yashon et al. (1968).

4. LAGOS, J.C. and SIEKERT R. G.: Intracranial hemorrhage in infancy and childhood. Clin. Pediatr. 8 : 9 (1969).

5. PLATOU, R.V.; RUNKER, A. and DERRICK, J: Acute subdural effusions and late sequelae of meningitis. Pediatr. 23 : 962 (1959).

6. RABE, E.E.: Subdural effusion in infancy. Pediatr. Clin. North Am. 14 : 831 (1967).

7. SUTOMENGOLO, T.S.; ISMAEL, S.; HARDJANTO, H. CH. and LUMBANTOIBING, S.M.: Subdural effusion complicating purulent meningitis. Paediatr. Indones. 14 : 43 (1974).

8. YASHON, D.; JANE, J.A.; WHITE, R.J. and SUGAR, O.: Traumatic subdural hematoma of infancy. Arch. Neurol. 18 : 374 (1968).