Paediatrica Indonesiana 14 : 43 — 52. March — April 1974.

From the Department of Child Health, Medical School, University of Indonesia, Jakarta

Subdural Effusion Complicating Purulent Meningitis.

by

TASLIM S. SUTOMENGGOLO, SOFJAN ISMAEL, HARRY CH. HARDJATNO and S.M. LUMBANTOBING

Subdural effusions are collections of fluid in the subdural space, characterized by an aspirated volume of more than 2 ml. at any one time, a protein concentration of at least 40 mg. per 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 per ml.

Pathogenesis of subdural effusion

Until recently the cause of subdural collection of fluid in meningitis or in non - meningitic states is still not exactly known. Among many hypotheses or theories which have been suggested, are:

1. Effusion of fluid through irritated or damaged capillary walls in the arachnoid or the duramater (Smith et al. 1951, Gitlin 1952, Guthkelch 1953, and Mc Kay et al. 1953). In bacterial meningitis damage is caused by inflammation, in pneumoencephalography irritation by the air itself might be the possible cause.
2. Thrombophlebitis of the cerebral veins. This theory was suggested by Guthkelch after his autopsy finding in a patient with cerebral venous thrombosis, who displayed subdural effusion during life. Thus it seems that thrombophlebitis of small blood vessels adjacent to the subdural space, probably most often the bridging veins, might be one of the basic mechanisms for the production of subdural effusion in meningitis or in focal infection elsewhere in the body.
3. Direct extension through a necrotic arachnoid membrane, either at the arachnoid villi or over the hemisphere (Spitz et al. 1945).

Presented at the Second National Indonesian Pediatric Congress, Bandung, 21 — 25 April 1971.
Received 4th December 1973.

4. Simultaneous infection of the arachnoid and the dura by a blood borne infection (Spitz et al. 1945).
5. Rupture of the arachnoid membrane due to raised subarachnoid pressure (Turner 1954).
6. Complication of either intra-thecal antibiotic therapy or of repeated lumbar punctures.
7. Williams & Stevens (1954) speculated that excessive withdrawal of cerebrospinal fluid (10-15 ml.) for diagnostic purposes in infants with suspected meningitis might create sagging of the brain and tearing of bridging veins.
8. Smith and Crothers (1950) speculating on the mechanism responsible for the formation of effusion following pneumoencephalography, suggested that as air enters the subdural space and the brain "falls away", the bridging veins with their arachnoid sheaths are stretched and torn, so that the fluid escapes into the subdural space.
9. Abnormally fragile or stretched bridging veins and dural reaction which is caused by an irritating substance in the subdural space. This theory was suggested by Rabe (1967). According to him, the introduction of purulent fluid or blood into the subdural space could occur in a patient with acute meningitis as the result of (a) rupture of an infected thinwall arachnoid villus with discharge of the subarachnoid fluid into the subdural space, (b) phlebitis of the bridging veins secondary to meningitis with rupture of the venous wall as it crosses the subdural space or (c) primary infection of the dura by blood-borne bacteria, which presumably also produce the meningitis.

The purpose of this paper is to evaluate the incidence, sex, age, nutritional condition, finding the effusion, mortality and, clinical signs and symptoms which can support the diagnosis of subdural effusion.

Material and methods

The material consists of 242 patients of purulent meningitis which were hospitalized in the Department of Child Health, Dr. Tjipto Mangunkusumo General Hospital, since January 1968 until December 1969. Transillumination was done in all the patients. Subdural tap was performed in the patients with positive transillumination finding. Transilluminati-on was performed in a dark room using an instrument as seen in figure 1.

Transillumination was considered positive when:

1. The area of translucence was more than 3 cm. in infants under the age of 6 months and more than 2 cm. in infants above the age of 6 months.
2. There were asymmetric areas of translucence.

Subdural tap was done with a lumbar puncture needle no. 19 or 20. The needle was inserted with a depth of 3-5 mm. in the lateral area of the anterior fontanelle. If no fluid was available, the needle could be manipulated forward, lateral or backward horizontally. For reasons of safety the amount of fluid evacuated did not exceed 15 ml. Taps were repeated every two days for a period of about 2 weeks. If fluid remained present there after, performance of temporal burr hole was the consequence. Shunting was performed when in spite of burr hole the fluid reaccumulated considerably. All these meningitic patients were primarily treated with antibiotics, i.e. procaine penicillin and chloromycetin or ampicillin. Electroencephalography was performed using Grass instrument with 8 channels.

Result

Forty seven of 242 meningitic patients showed positive transillumination. After tapping and further examination, 45 out of 47 revealed subdural effusion, whereas the remaining had porencephaly. Of the 45 patients 37 (82%) suffered from bilateral effusion, the remaining 8 had either left or right unilateral localization (table 1).

TABLE 1 : *Localization of subdural effusion.*

Localization	No. of patients	%
Right	4	9
Left	4	9
Bilateral	37	82
Total	45	100

Among the 45 patients 22 were males and 23 were females. Most were below the age of one year and 34 patients (75.5%) below the age of 6 months (table 2). The youngest patient was 1 month and the oldest 15 months old.

Of the 45 patients 36 (80%) were in a good nutritional condition.

Most of the subdural effusions could be detected during the first week of hospitalization. Figure 2 shows that in 29 children (64.5%) the diagnosis could be established

TABLE 2 : *Sex and age group of 45 patients.*

Age group in months	SEX		Total	%
	male	female		
0 - 6	16	18	34	77.5
6 - 12	5	5	10	22.5
>12	1	—	1	2
Total	22	23	45	100

during the first week and in totally 39 (90%) during the first 2 weeks of hospitalization.

Clinical signs & symptoms can be seen on table 3. Fever which occured more than 2 times 24 hours was found in 45 patients (100%), impaired consciousness in 39 patients (86%), bulging of the anterior fontanelle in 33 patients (73%), seizures in 19 (42%), neurological deficit in 17 (40%), vomiting in 16 (36%), ophthalmological disorders in 4 (9%), whereas opisthotonus and irritability were absent.

TABLE 3 : *Clinical Signs & Symptoms of 45 patients with Subdural Effusion.*

Clinical signs & symptoms	Number	%
Fever more than 2 × 24 hours	45	100
Impaired Consciousness	39	86
Bulging anterior fontanelle	33	73
Seizures	19	42
Neurological deficit	17	40
Vomiting	16	36
Ophthalmological disorders	4	9
Opisthotonus	—	—
Irritability	—	—

Most of the patients suffered from fever for more than one week. Only in 6 fever was less than one week. i.e. 1 patient for 3 days, 2 for 5 days, and 3 for 6 days. The relation between fever and subdural effusion can be seen on figure 3.

Neurological deficit in 17 patients consists of spastic tetraparesis 8, spastic tetraparesis and extrapyrami-

TABLE 4 : *Neurological Deficit in 17 patients.*

Neurological deficit	Number of patients
Spastic tetraparesis	8
Spastic tetraparesis + extrapyramidal disorder	1
Hemiparesis	8
Total	17

dal disorder 1 and hemiparesis 8 (table 4). Thirteen out of 17 patients with neurological deficit died.

The result of serial taps was in 31 patients (69%) good, 10 (22%) died before the 2-week taps ended, 3 forced discharge, in one patient tapping failed, so that this patient was sent directly to the Department of Neuro-Surgery for shunt operation (table 5).

TABLE 5 : *The result of subdural taps.*

Result	Number of patients	%
Good	31	69
Died before serial taps were finished	10	22
Forced discharge	3	7
Shunt operation (failure of tapping)	1	2
Total	45	100

Diseases that could be mentioned as the primary cause can be seen on table 6. Acute otitis media was found in 11, bronchopneumonia in 7, acute otitis media combined with bronchopneumonia in 2, perforated otitis media in 2, pharingitis in 1, and unknown in 22 patients.

Electroencephalography (E.E.G.) was done on 26 patients and the result was normal in 13 (50%), borderline normal in one (4%), and abnormal in 12 patients (46%) (table 7).

Fourteen of the 45 patients (31%) died. Eleven of them suffered from fever for more than two weeks and 13 suffered from neurological deficit. Most of the deaths were not caused by subdural effusion itself, but might be caused by the accompanying diseases as can be seen on table 8. In the 14 dead patients subdural empyema

TABLE 6 : *Diseases that might be the primary cause.*

DISEASE	Number of patients
Acute otitis media	11
Bronchopneumonia	7
Acute otitis media + bronchopneumonia	2
Perforated otitis media	2
Pharingitis	1
Unknown	22
TOTAL	45

TABLE 7 : *EEG in 26 patients.*

RESULT	Number of patients	%
Normal	13	50
Borderline normal	1	4
Borderline abnormal	—	—
Abnormal	12	46
TOTAL	26	100

was found in 4, hydrocephalus in 1, shunt operation in 1, aspirated pneumonia in 2, bronchopneumonia in 2, meningitis itself in 1, and unknown cause of death in 3 patients.

TABLE 8 : *The accompanying diseases of the dead patients.*

ACCOMPANYING DISEASE	Number of patients
Subdural empyema	4
Hydrocephalus	1
Shunt operation	1
Aspirated pneumonia	2
Bronchopneumonia	2
Meningitis	1
Unknown (longstanding fever)	3
TOTAL	14

Discussion

Out of 47 patients with positive transilluminations only 45 suffered from subdural effusion. Positive transilluminations is not always caused by subdural effusion, but also by other conditions, such as hydrocephalus, cerebral atrophy, and porencephaly (Suckling 1959, Sofjan Ismael 1970).

The incidence of subdural effusion in purulent meningitis varies considerably among different authors, but variation as seen in table 9 is not significant, except Smith et al. 1951 who found an incidence of as high as 46%; however, his material consisted of only 43 patients.

TABLE 9 : *Incidence of subdural effusion by various authors.*

Authors	No. of purulent Meningitis patients	No. of subdural effusion	%
SMITH et al. (1951)	43	20	46
PLATOU et al. (1959)	343	55	16
BENSON et al. (1960)	320	67	20
BAMBANG MADIONO et al. (1967)	40	6	15
TASLIM et al. (1971)	242	45	18

There is no difference in sex in this series. However, Benson et al. (1960) and Meacham et al. (1970) found that most of their patients were males.

Most of our patients are below the age of one year, and 75.5% are below the age of 6 months. Sixty percent of Mc Kay's cases were also below the age of one year, and Platou's cases were 91% below the age of one year and 68% below the age of 6 months.

Smith et al. (1951), Platou et al. (1959), Rabe (1967), and Till (1970) stated that the clinical signs and symptoms were longstanding fever, impaired consciousness, vomiting, bulging of the anterior fontanelle, convulsions, neurological deficit, ophthalmological disorders, opisthotonus, and irritability. The most common clinical signs and symptoms in our series are fever of more than two times 24 hours, impaired consciousness, and bulging of the anterior fontanelle. Most of our patients suffered from fever for more than one week. Poey et al. (1967) also found that their cases suffered from longstanding fever.

By mainly tapping 31 (69%) of 45 patients gave good result. It is a pity that 10 died and 3 forced to be discharged before the serial taps were finished. In 1 patient tapping failed and shunt operation was performed. By mainly tapping the result could

be considered good; it was most probably due to the early detection of the subdural effusion. Most observers agree that repeated subdural taps, if it is started early and performed in every one to two days, may dry up the effusion. In more chronic or persistent accumulation however, the fluid usually becomes encapsulated between an outer and inner fibrous membrane. Epstein (1953), Lagos and Siekert (1969) stated that usually the membranes were formed after two to three weeks. So, if after two to three weeks of continued drainage or aspiration, the fluid shows no signs of regression, surgical exploration must be performed to determine the presence of the membranes, and to remove when they are present already (burr hole and craniotomy). If the fluid reaccumulates, shunt operation must be done. In most of our patients subdural effusion was detected during the first and second week of hospitalization. Of the 45, twenty nine (64.5%) were discovered during the first week and 90% during the first two weeks of hospitalization. Repeated subdural taps of our patients were started early.

TABLE 10: *The Mortality of subdural Fluid Collections by various authors.*

Authors	No. of patients	Died	%
Rosenberg (1921)	33	7	21
Ingraham and Matson (1944)	98	9	9
Statten (1948)	28	10	35.5
Elvidge and Jackson (1949)	55	20	36
Ingraham and Matson (1949)	113	—	—
Guthkelch (1953)	24	5	21
Ingraham and Matson (1954)	222	—	—
Scheppe (1954)	13	—	—
Freundlich et al. (1956)	19	6	31.5
Herzberger et al. (1956)	33	7	21
Benson et al. (1960)	59	10	17
Schulman and Ransohoff (1961)	53	7	11
Christensen and Husby (1962)	24	15	62.5
Hendrick et al. (1964)	235	52	22
Rabe et al. (1965)	62	9	14.5
Yashon et al. (1968)	92	21	22.5
Taslim et al. (1971)	45	14	31

E.E.G. was done on 26 patients and 13 (50%) of them were normal. Steifler et al. (1958) suggested the E.E.G. as an important diagnostic

tool, but occasionally normal records have been obtained from patients with subdural collection of fluid. Matson and Berman (1958) pointed out that the only definite diagnosis was positive subdural tap and that the use of E.E.G. in subdural effusion has been of more academic interest. Rabe (1967) said that E.E.G. is not widely accepted as a help in diagnosing or localizing subdural collections of fluid. So, a normal E.E.G. does not mean that there is no subdural effusion.

The mortality of this series is too high (31%), but most of the children suffered from severe diseases. Besides the subdural effusion, they also suffered from purulent meningitis ,and other accompanying diseases (table 8). Eleven of them suffered from fever of more than 2 weeks, and 13 suffered from neurological deficit. About mortality various authors found different figures (table 10).

Summary

Two hundred and forty two patients with purulent meningitis were transilluminated and 47 of them showed positive results. Forty five of these latter suffered from subdural effusion. Most of the patients were below the age of one year and 75.5% below the age of 6 months. By serial taps every two days 69% of them improved; one patient failed. The mortality was high, but it might be caused by the accompanying diseases. A normal E.E.G. does not mean that there is no subdural effusion.

REFERENCES

1. BAMBANG MADIJONO, ASRIL AMINULLAH, MUSLICHAN, OEI GOAN PIEN LIEM and SUTEDJO : Evaluation of a modified treatment of purulent meningitis. Paediatr. Indones. 7 : 165 (1967).
2. BENSON, P.H., NYHAM, W.L., and SHIMIZU, H. : The prognosis of subdural effusions complicating pyogenic meningitis. Pediatrics 57 : 670 (1960).
3. EPSTEIN, J.A., and GOLDZIEHER, S.E. II : Bilateral encapsulated subdural effusion complicating bacterial meningitis in infancy. Arch. Neurol. & Psychiat. 69 : 242 (1953).
4. GUTHKELCH, A.H. : Subdural effusion in infancy: 24 cases. Brit. med. J. 1 : 233 (1953). (Cited by Husein Alatas, 1967).
5. GITLIN, D. : Pathogenesis of subdural collections of fluid. Pediatrics 16 : 345 (1955).
6. HUSEIN ALATAS : Subdural effusion complicating bacterial meningitis. Paediatr. Indones. 7 : 137 (1967).
7. LAGOS, J.C., SIEKERT, R.G.: Intracranial hemorrhage in infancy and childhood. Clin. pediat. 8 : 9 (1969).
8. MATSON, D.D. and BERMAN, B.A. in Gellis Yb. Pediat. 462 - 472 (1958 - 1959). (Cited by Husein Alatas 1967).
9. Mc KAY, R.J., Jr., INGRAHAM, F.D., and MATSON, D.D. : Subdural fluid

complicating bacterial meningitis. J. amer. med. Ass. 152 : 387 (1953).

10. MEACHAM, W.F., Mc PHERSON, W.F. : The diagnosis and treatment of subdural fluid collections in infants. Pediat. Clin. N. Amer. 17 : 363 (1970).
11. PLATOU, R.V., RUNKER, A., and DERRICK, J. : Acute subdural effusions and late sequelae of meningitis. Pediatrics 23 : 963 (1959).
12. POEY, S.H., HALIMUN, E.M., and SULARJO, S. : Subdural effusion in longstanding fever. Paediatr. Indones. 7 : 131 (1967).
13. RABE, E.F. : Subdural effusion in infants. Pediat. Clin. N. Amer. 14 : 831 (1967).
14. SMITH, M.H.D., DORMONT, R.E., and PRATHER, G.W. : Subdural effusion complicating bacterial meningitis. Pediatrics 1 : 34 (1951).
15. SMITH, H.V., and CROTHERS, B. : Subdural fluid as a consequence of pneumoencephalography. Pediatrics 6 : 375 (1950).
16. SPITZ, E., POLLAK, A. and ANGRIST, A. : Subdural suppuration originating in purulent leptomeningitis. Arch. Neurol. Psychiat. 53 : 144 (1945).
17. STEIFLER, M., FREUNLICH, E., and BELLER, A.J. : Electroencephalograpny in subdural hematoma and effusion in infants. Amer. J. Dis. Child. 95 : 25 (1958).
18. SUCKLING, P.V., and WITTMANN, W. : Subdural effusion with respiratory infections in the first year of life. Sth. afr. med. J. 33 : 1057 (1959).
19. SOFJAN ISMAEL, LUMBANTOBING, S.M., KARJOMANGGOLO, W.T., and TAMAELA, L.A. : "Air study" sebagai penolong dalam menegakkan diagnosis efusi subdural. Maj. Kedok. Indon. 8 : 355 (1970).
20. YASHON, D., JANE, J.A., WHITE, R.J., and SUGAR, O. : Traumatic subdural hematoma of infancy. Arch. Neurol. 18 : 374 (1968).
21. TILL, K. : Subdural haematoma and effusions in infancy. Brit. med. J. 3 : 400 (1968).
22. TURNER, L. : Subdural collection of fluid in tuberculous meningitis. Lancet i : 849 (1954).
23. WILLIAMS, J.M., and STEVENS, H. : Post meningitis subdural effusion. J. int. Coll. Surg. 27 : 590 (1957). (Cited by Husein Alatas 1967).