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Neurointensive care of patients with severe community-acquired meningitis

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Background: Reports about neurointensive care of severe community-acquired meningitis are few. The aims of this retrospective study were to review the acute clinical course, management and outcome in a series of bacterial meningitis patients receiving neurointensive care.
Methods: Thirty patients (median age 51, range 1–81) admitted from a population of 2 million people during 7 years were studied. The neurointensive care protocol included escalated stepwise treatment with mild hyperventilation, cerebrospinal fluid (CSF) drainage, continuous thiopentotal infusion and decompressive craniectomy. Clinical outcome was assessed using the Glasgow outcome scale.
Results: Twenty-eight patients did not respond to commands on arrival, five were non-reacting and five had dilated pupils. Twenty-two patients had positive CSF cultures: *Streptococcus pneumoniae* ($n = 18$), *Neisseria meningitidis* ($n = 2$), β-streptococcus group A ($n = 1$) and *Staphylococcus aureus* ($n = 1$). Thirty-five patients were mechanically ventilated. Intracranial pressure (ICP) was monitored in 28 patients (intraventricular catheter = 26, intracerebral transducers = 2). CSF was drained in 15 patients. Three patients received thiopentothal. Increased ICP (>20 mmHg) was observed in 7/26 patients with available ICP data. Six patients died during neurointensive care: total brain infarction ($n = 4$), cardiac arrest ($n = 1$) and treatment withdrawal ($n = 1$). Seven patients died after discharge, three due to meningitis complications. At follow-up, 14 patients showed good recovery, six moderate disability, two severe disability and 13 were dead.
Conclusion: Patients judged to have severe meningitis should be admitted to neurointensive care units without delay for ICP monitoring and management according to modern neurointensive care principles.

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COMMUNITY-ACQUIRED bacterial meningitis is a severe disease that is still associated with substantial morbidity and mortality.[1–4] The most frequent cause of death is high intracranial pressure (ICP).[2] The mechanisms behind the development of increased ICP are increased cerebral blood volume due to vasodilatation, vasogenic, cytotoxic and interstitial brain edema, respectively, and communicating hydrocephalus.[5,6] Modern neurointensive care provides a possibility to challenge those disturbances in a physiological way and reduce secondary brain injury.[7] Although neurointensive care may play an important role in severe cases to reduce morbidity and mortality, the reported clinical studies on neurointensive care of bacterial meningitis are few.[8–13] The aims of this study were to retrospectively review the acute clinical course, management and outcome in a series of patients with severe community-acquired meningitis receiving neurointensive care.

Materials and methods

Patients

Consecutive patients with community-acquired meningitis receiving neurointensive care at the University hospital in Uppsala, Sweden, between January 2000 and July 2007 were included in the study. The patients were admitted from a background population of 2×10^6 inhabitants. The indication for neurointensive care was predominantly judged on clinical grounds. Neurointensive care was considered in patients with Reaction Level Scale[14] grade $\geq$ 3B (warding of pain) and also in more alert patients with sudden deterioration. Table 1 shows the Reaction Level Scale grades in relation to the Glasgow Coma Scale Motor scores[15] (Table 1). Other indications of neurointensive care were repeated seizures, severe agitation, cranial nerve palsy, hypertonia and bradycardia,

papill edema or computed tomography indications of increased ICP. The diagnosis was based on the clinical picture suggesting meningitis and the results of cerebrospinal fluid (CSF) analysis (microscopy, chemistry and culturing). Clinical data were obtained from the patient records and surveillance charts.

Antibiotics and corticosteroids

Patients with community-acquired meningitis at Uppsala university hospital were treated according to the Swedish national recommendations.* The antibiotic recommendations are either cefotaxime $3\,g \times 4$ intravenous (i.v.) in combination with ampicillin $3\,g \times 4$ i.v. or meropenem $2\,g \times 3$ i.v. until the results of cultures are presented. The patients receive betamethasone $0.12\,mg/kg \times 4$ i.v. for 4 days, the first dose given before the first dose of antibiotics. The recommended duration of antibiotic therapy was 10–14 days in *Streptococcus pneumoniae*, 7 days in *Neisseria meningitides*, 10 days in *Haemophilus influenzae* and 14–21 days in the remaining usual organisms.

Neurointensive care

Neurological grade was assessed regularly according to the Reaction Level Scale.[14] The physiological monitoring included arterial blood pressure, heart rate, electro cardiogram and temperature. The indication for ICP monitoring was mainly based on the level of consciousness. ICP was monitored in patients with Reaction Level Scale grade $\geq$ 3B-4 (warding of pain-localizes pain) using a ventricular catheter drainage system (Liqvor-Drainage-Katheter-Set®, Smiths Medical, Kirchseeon, Germany) as the first choice and intracerebral pressure transducers (Codman ICP transducer sensor®, Codman, Johnson and Johnson, Chicago, IL) in cases with compressed ventricles. Patients were intubated and mechanically ventilated at the same Reaction Level Scale grade as when ICP monitoring was thought indicated. In cases with sudden impairment, intubation and ICP monitoring were initiated earlier. The neurointensive care management goals are listed in Table 2. Computed tomography scanning of the head was performed on clinical indications and before the ICP monitoring and mechanical ventilation were finished. The neurointensive care followed an escalated ICP-cerebral perfusion pressure-directed protocol as follows: the basal management included mild hyperventilation, normovolemia obtained by generous infusions of albumin 20% to maintain an adequate colloid osmotic pressure, normotension according to age and medical history. Central venous pressure was maintained between 0 and 5 mmHg to avoid hypervolemia with a risk of aggravating brain edema. Hypotension was first managed by infusions of albumin 20% and crystalloids. In cases with high ICP and high cerebral perfusion pressure, the blood pressure was lowered by increased sedation and/or β-blockers at moderate doses. In case of high ICP despite this management, CSF was drained against a pressure level of 15–20 mmHg. The next management step was high-dose thiopentotal coma treatment if ICP persisted to be high and more CSF drainage was not possible due to a compressed ventricular system. Pentothal® (thiopental sodium, 25 mg/ml, Hospira Enterprises B.V., Hoofddorp, the Netherlands) was given as a bolus i.v. injection of 4–8 mg/kg, followed by an infusion, 5–10 mg/kg h the first 6 h and 2–5 mg/kg h thereafter. The dosage and duration of treatment are mainly guided by ICP response and the occurrence of complications. Decompressive craniectomy was considered as the last management

Table 1

Summery of the Reaction Level Scale grades (RLS 85)[14] in relation to the Glasgow Coma Scale Motor scores (GCS M).[15]

RLS 85	GCS M
1 Alert response	6 – obeys
2 Delayed response	6 – obeys
3a Very delayed response*	6 – obeys
3b Wards of pain*	5 – localizing
4 Localizes pain	5 – localizing
5 Withdrawing movements	4 – normal flexion
6 Stereotype flexion	3 – abnormal flexion
7 Stereotype extension	2 – extending
8 No response	1 – no response

*Modification of original RLS 85 in order to differentiate between very delayed response and warding of pain in RLS 3.

Table 2

Neurointensive care management goals.

Variable	Goal
Intracranial pressure	<20 mmHg
Cerebral perfusion pressure	$\sim$60 mmHg
$PaCO_2$	4.0–4.5 kPa
Temperature	$<38^{\circ}C$
B-Glucose	5.0–10 mmol/l
Hemoglobin	120 g/l
S-Albumin	$\sim$40 g/l

*http://www.infektion.net/klinik/cns/vardprogram_CNS-inf_2004-3.pdf (accessed 6 February 2011).

step if the thiopentotal coma treatment was insufficient or associated with severe complications.

Evaluation of outcome

The extended Glasgow Outcome Scale was used.[16,17] Structured telephone interviews were conducted >6 months (mean = 41, median = 36, range = 6–93) after neurointensive care, using the structured questionnaire presented by Wilson et al.[17] The current outcome at the time of follow-up as well as the estimated outcome at 6 months were assessed.

Results

Patient characteristics

Thirty-six patients were identified and included in the series, 10 admitted primarily to the university hospital and 26 referred from local hospitals. The patient characteristics are presented in Table 3. Twenty-three were men and 13 women, with a mean age of 46 years (median 51, range 1-81). Twenty patients had one ($n = 16$) or two ($n = 4$) concurrent diseases, alcohol abuse ($n = 5$), diabetes mellitus ($n = 4$), acute lymphatic leukemia ($n = 1$), lymphoma ($n = 1$), myeloma ($n = 1$), idiopathic thrombocytopenia ($n = 1$), rheumatoid arthritis ($n = 1$), hypothyroidism ($n = 1$) and cardiovascular diseases ($n = 9$). In addition, three patients had previously been treated for pneumococcal meningitis. One of those patients had received pneumococcal vaccination because of recurrent meningitis ($n = 1$) and one had been operated with splenectomy ($n = 1$). Two patients had previously been treated for traumatic brain injury.

Neurological symptoms and signs

Initially, 15 patients had headache, three patients showed neck stiffness and 23 patients had fever. Thirty-three of the 36 patients presented with impaired consciousness. Seven patients had symptoms/signs from their ears (pain, otitis, mastoiditis). Ten patients presented with seizures. The neurological reaction levels at arrival to the intensive care unit are presented in Fig. 1. Five patients were in Reaction Level Scale grade 8 at arrival, four with wide non-reacting pupils bilaterally and one with reacting pupils. One patient in Reaction Level Scale grade 3B (warding of pain) showed a wide non-reacting pupil on the left side.

Diagnosis

The results of the initial CSF studies are summarized in Table 3. In two patients, the results of the CSF and blood cultures were not available, one of those initially managed abroad. Twenty-two of the remaining 34 patients had positive CSF cultures, *S. pneumoniae* ($n = 18$), *N. meningitidis* ($n = 2$), β-streptococcus group A ($n = 1$) and *Staphylococcus aureus* ($n = 1$). Fourteen of the 18 patients with CSF verified *S. pneumoniae* had positive blood cultures. The patients with β-streptococcus group A and *S. aureus* in their CSF cultures also demonstrated positive blood cultures. Among the 12 patients with negative CSF cultures, 11 demonstrated growth in the blood:*S. Pneumoniae* ($n = 6$), *N. meningitidis* ($n = 2$), *Listeria monocytogenes* ($n = 1$), gram positive coccus (no further typing possible) ($n = 1$) and *S. aureus* ($n = 1$). In one patient, in whom the samples were taken during antibiotic therapy, both the CSF and the blood cultures were negative.

Neurointensive care

Thirty-five of the 36 patients stayed a mean of 8 days at the neurointensive care unit (median = 6 days, range = 1–35 days, Table 3). The remaining patient stayed 106 days because of ventriculitis and cystic formations (Table 3). Thirty-five of the 36 patients were mechanically ventilated (mean = 5 days, median = 3 days, range = 1–27 days; long-staying patient excluded; Table 3). ICP was monitored in 28 patients (data available in 26) using an intraventricular drainage catheter system in 26 and intracerebral transducers in 2. ICP was not monitored in six patients who were awake at arrival and in two patients in Reaction Level Scale grade 8 (one with wide non-reacting pupils). The two patients who had intracerebral tranducers were two children in Reaction Level Scale grade 8 with fixed non-reacting pupils at arrival. CSF was drained in 15 of 26 patients with vetriculostomy. Three patients received thiopentotal coma treatment. An increased ICP (>20 mmHg) was observed in seven out of 26 patients with ICP monitoring and available data (Table 4).

Outcome

Six patients died during neurointensive care because of total brain infarction due to refractory ICP ($n = 4$), cardiac arrest ($n = 1$) and treatment withdrawal, followed by cardiac arrest ($n = 1$). Seven patients died after discharge (Table 5) due to

Table 3

Summary of patient characteristics.

Patient (no.)	Age/ sex	RLS score (on admission)	CSF leukocyte count (10^3 cells/ml)	CSF albumin (g/l)	Glucose ratio (serum/ CSF)	Etiology	Mechanical ventilation (days)	NIC (days)	GOS*
1	52/M	6	4450 poly/ 740mono	2.3	9.9/0.5	Negative cultures	>30	106	LSD
2	29/M	3B	4560poly/ 880mono	3.96	11.1/1.2	*S. pneumoniae*	2	5	UGR
3	66/M	7	666poly/190mono	2.5	12.1/3.8	*S. pneumoniae*	27	35	D<6
4	21/F	2	1800poly/ 1090mono	2.7	6.8/1.8	*N. meningitidis*	8	9	UGR
5	81/M	5	420poly/145mono	5.7	17.6/4.6	*S. aureus*	2	2	D>6
6	59/F	3B	326poly/176mono	0.23	10.3/1.9	*S. pneumoniae*	6	6	D<6
7	61/M	6	492poly/220mono	1.43	10.3/0.2	*S. pneumoniae*	8	10	D>6
8	81/M	4	1865poly/75mono	0.85	8.8/3.9	*S. pneumoniae*	3	3	LGR
9	11/M	3B	1020poly/ 680mono	0.10	8.1/1.4	ß-streptococcus group A	14	24	LGR
10	47/M	3B	1530poly/30mono	0.86	9.9/6.4	*S. pneumoniae*	3	6	UGR
11	67/F	5	27900poly/ 7000mono	1.34	10.1/0.1	*S. aureus*	9	12	LSD
12	51/M	6	4poly/14mono	3.38	6.3/<0.1	*S. pneumoniae*	12	12	D<6
13	74/M	2	11000poly/480 mono	6.10	7.8/<0.1	*S. pneumoniae*	1	2	UGR
14	46/M	2	76poly/44mono	0.69	6.7/2.7	*S. pneumoniae*	3	32	LMD
15	45/M	3B	3440poly/ 660mono	0.25	16.5/5.6	*S. pneumoniae*	2	5	Declined
16	20/F	3B	6800poly/ 2100mono	0.12	11.7/6.5	*Neisseria meningitidis*	4	6	LGR
17	66/M	5	260poly120mono		/<0.5	*S. pneumoniae*	4	8	UGR
18	39/M	8	3000poly/ 4700mono	3.9	9.2/<0.3	Results missing	1	1	D<6
19	11/F	2	960poly/40mono	7.1	6.4/3.0	Gram positive coccus	1	8	LGR
20	53/F	3B	80leukocytes†		/<0.3	*S. pneumoniae*	10	10	D<6
21	71/F	3B	2930poly/ 120mono	0.64	9.2/<0.5	*S. pneumoniae*	3	3	UMD
22	52/M	5	3000poly/ 900mono	5	17.1/7.7	*N. meningitidis*	3	13	LMD
23	40/F	3B	9506poly/ 962mono	3.44	8.9/3.4	*S. pneumoniae*	4	6	LGR
24	71/F	2	6000poly/ 140mono	4.4	15.8/4.4	*S. pneumoniae*		1	LMD
25	76/F	8	17poly/16mono	4.48	/<0.1	*S. pneumoniae*	1	2	LMD
26	60/M	3A	347poly/187mono			Results missing	2	5	UGR
27	33/M	4	614poly/114mono	4.2	16/0.7	*S. pneumoniae*	3	6	UGR
28	17/M	4	101poly/46mono	0.27	11.7/7.4	*N. meningitidis*	3	5	UGR
29	66/M	2	8poly/10mono	2.0	7.0/<0.1	*S. pneumoniae*	6	22	LGR
30	31/M	8	108poly/98mono	1.0	8.7/1.5	*S. pneumoniae*	3	3	D<6
31	2/F	8	620poly/ 1230mono		8.7/<0.1	*S. pneumoniae*	4	4	D<6
32	55/F	3B	0poly/2364mono	5.17	/<0.1	*S. pneumoniae*	2	7	D>6
33	1/M	8	106poly/44mono	2.32	5.1/0.1	*S. pneumoniae*	3	3	D<6
34	31/K	4	6200poly/ 4300mono	1.51	9.7/0.8	*S. pneumoniae*	2	2	UMD
35	54/M	3B	33200poly/ 2600mono	9.65	22.7/14.1	*S. pneumoniae*	6	8	D<6
36	37/M	3A	26poly/61mono	6.75	/7.8	*L. monocytogenes*	3	6	D<6

*Glasgow Outcome Scale (UGR, = upper good recovery; LGR, = lower good recovery; UMD, = upper moderate disability; LMD, = lower moderate disability; USD, = upper severe disability; LSD, = lower severe disability; D<6, = dead within 6 months; D>6, = dead later than 6 months).

†Differentiation impossible.

RLS, Reaction Level Scale; NIC, neurointensive care; GOS, Glasgow Outcome Scale; CSF, cerebrospinal fluid; *S. pneumoniae*, *Streptococcus pneumoniae*; *N. meningitides*, *Neisseria meningitides*; *L. monocytogenes*; *Listeria monocytogenes*; *S. aureus*, *Staphylococcus aureus*.

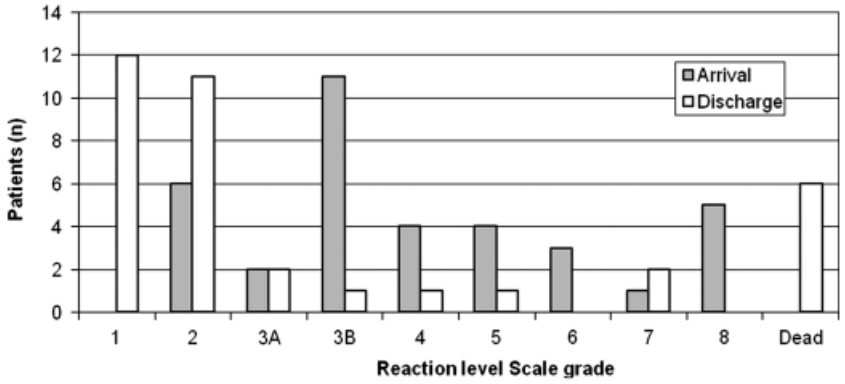

Fig. 1. Level of consciousness in patients with severe acute bacterial meningitis. Reaction Level Scale (RLS) grade[14] on arrival to the neurointensive care unit and at discharge. RLS grade 1 = alert response, RLS grade 2 = delayed response, RLS grade 3A = very delayed response, RLS grade 3B = wards of pain, RLS grade 4 = localizes pain, RLS grade 5 = withdrawing movements, RLS grade 6 = stereotype flexion, RLS grade 7 = stereotype extension and RLS grade 8 = no response.

complications and sequelae of the meningitis ($n = 3$), pneumonia and respiratory insufficiency ($n = 1$), rectal carcinoma ($n = 1$), myeloma and renal failure ($n = 1$) and cardiac insufficiency ($n = 1$).

More patients were awake (Reaction Level Scale grade ≥ 3) at discharge from neurointensive care than at arrival (Fig. 1). At later follow-up, one patient declined follow-up. Current outcome at follow-up showed 14 patients with good recovery, six patients with moderate disability, two patients with severe disability and 13 were dead (Fig. 2). The estimated outcome at 6 months showed eight patients with good recovery, three with moderate disability, 11 with severe disability and three were either dead or not possible to estimate (Fig. 2). Described symptoms contributing to a poor outcome are listed in Table 6. Outcomes in relation to patient characteristics are presented in Table 7.

Table 4

Number of hours (h) spent at different levels of increased intracranial pressure (ICP) for seven patients with increased ICP ($\geq$ 20 mmHg).

Patient	ICP 20–24 mmHg	ICP 25–29 mmHg	ICP $\geq$ 30 mmHg
2	2 h	0 h	1 h
4	3 h	2 h	1 h
9	23 h	10 h	2 h
28	2 h	0 h	1 h
30	0 h	0 h	67 h
31	0 h	0 h	94 h
33	0 h	0 h	72 h

Discussion

The Swedish national guidelines for bacterial infections in the nervous system from 2004* recommend that neurointensive care should be considered under one or more of the following conditions: Reaction Level Scale grade $\geq$ 3B, Reaction Level Scale grade <3A but gradually worsening, repeated seizures, severe agitation, cranial nerve palsy, hypertonia and bradycardia, papill edema or Computed tomography indications of increased ICP. These recommendations have been implemented in our health care region already during 2000 and 2001. From the background population of around 2×10^6 inhabitants, 36 patients were admitted for neurointensive care, showing that neurointensive care may be motivated in a significant number of patients, although only in a minor proportion of all patients with community-acquired meningitis. The present results showed that patients in all ages were admitted to neurointensive care (range 1–81 years, 13% <18 years, 31.5% $\geq$ 45 years, 26.3% >65 years). Another characteristic of the patients was that a large proportion (58%) had one or several concurrent conditions, many of which were associated with immunodeficiency. Impaired consciousness was the onset symptom in 33 of the 36 patients, and at admission to neurointensive care, 17 patients (47%) were unconscious with Reaction Level Scale grade 4–8. The level of consciousness at the time of ICU admission has been reported as a major predictor of the outcome.[18] Schutte and van der Meyden[19] reported a mortality rate of 62.5% among patients with meningitis and GCS score <8 at admission. Therefore, it is of utmost importance that the admittance to neurointensive care not is delayed when required as there is a significant risk of rapid impairment in patients with severe bacterial meningitis.

S. pneumoniae was the predominant bacteria (24 of 36 cases), which is in agreement with international results concerning community-acquired bacterial meningitis in adults.[18,20] *S. pneumoniae* is associated with a high rate of morbidity, mortality

**http://www.infektion.net/klinik/cns/vardprogram_CNS-inf_2004-3.pdf (accessed 6 February 2011).*

Table 5

Mortality after discharge from neurointensive care.

Patient	Cause of death	Age	Time of death after discharge from neurointensive care	Reaction Level Scale grade at discharge
3	Complications/sequelae meningitis	66	3.5 months	7
6	Complications/sequelae meningitis	59	7 days	5
12	Complications/sequelae meningitis	51	2 days	7
20	Pneumonia, respiratory insufficiency	53	7 days	2
5	Rectal cancer	81	6 months	2
7	Cardiac insufficiency	61	4 years and 10 months	3A
32	Myeloma, renal failure	55	2 years och 5 months	2

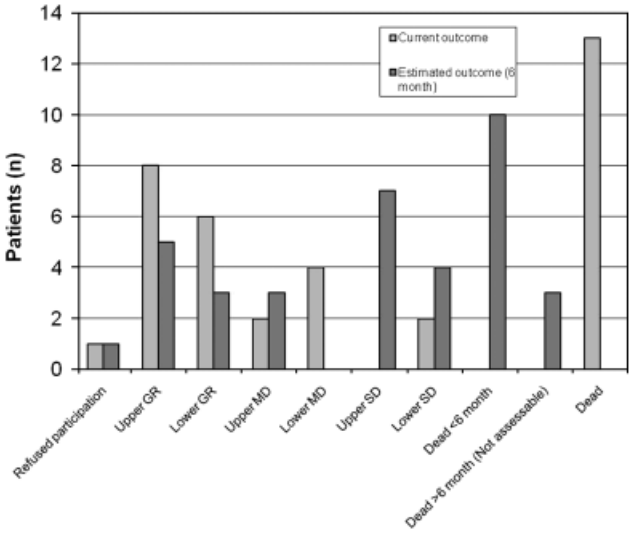

Fig. 2. Clinical outcome after neurointensive care of severe acute bacterial meningitis. Current outcome at follow-up (median = 36 month, range 6–93) according to the extended Glasgow Outcome Scale[17] and estimated outcome at 6 months. GR, good recovery; MD, moderate disability; SD, severe disability.

Table 6

Typical symptoms expressed by the patients contributing to poor outcome.

Described symptoms
Difficult to count. Difficulties with concentration. Poor short-term memory. Problems with memory in school. Failed in the main subjects. Mood swings. Aggressive
Poor eyesight. Used wheelchair temporarily
Impaired hearing. Disturbed by noises. Difficulties viewing the television
Cannot listen to music or be in large crowds
Deaf. Difficulties with balance, staggering walk
Fatigue. Problems socializing with friends. Never argues. Mood swings
Does not dare to drive. Fatigue. Dizziness. Constant rhinitis
Needs a lot of help from relatives with laundry and shopping.
Can use the subway but gets extremely tired and needs to rest.
Can manage physical activity but gets mentally tired very quickly. Mood swings. Depression. Headache
Could not walk after the illness. Learned to walk with support. Week legs
Blind. Home care two times per day. Needs help with personal hygiene. Loss of memory. Mood swings. Anxiety. Difficulties with concentration
Headache. Difficulties with concentration
Depression. Must rest to perform activities in the afternoon and evening. Works 2 h/day 2 days/week. Sews and knits
Works 4 h/day
Poor short-term memory. Many headaches. Overemotional
Constant headache. Home care four times per day. Walks with support. Recreational activity only when her children accompany her
Visually impaired. Fatigue. Initial difficulties in school, all mathematics knowledge was gone. Now reads mathematics at high school level and leads a hockey team
Dyscalculia. Dysphasia. Bad temper
Depressed. Argues and cries
Difficulties making decisions
Lost all friends. Aggressive. Changed personality. Dysphasia. Can only perform simple things such as buttoning a shirt or putting on a cap

and sequel, which was confirmed in the present study with a mortality of 8/18 (44%).[21] The mortality rate is even higher than previous reports of 19–37%,[20,21] which was expected as presented patients were admitted to the neurointensive care unit because of severe disease. *N. meningitidis* and *L. monocytogenes* occurred in a few cases. Three of four patients diagnosed with *N. meningitidis* were between 17 and 21 years and previously healthy. The patient with *L. monocytogenes* had undergone bone marrow transplantation because of leukemia.

The neurointensive care protocol applied followed ordinary management principles including ICP monitoring, CSF drainage and thiopentotal coma treatment in an escalated manner. Reviewing the neurointensive care management given, 35 patients were mechanically ventilated, ICP was monitored in 30 patients, CSF was drained in 15 out of 26 patients with ventriculostomy and three patients received treatment with thiopentothal to reduce ICP <20 mmHg. It is apparent that substantial neurointensive care treatment was required. Despite the intensive treatment given,

Table 7

Estimated 6-month outcome in relation to patient characteristics.

	Dead* (meningitis)	Poor outcome (severe disability)	Favorable outcome (good recovery or moderate disability)
Patients (*n*)	10	2	20
Age (year) mean	39.3	59.5	46.1
Median	45	59.5	46.5
Range	1–66	52–67	11–81
Concurrent diseases	8	1	9
S. pneumoniae	8		13
N. meningitides			4
RLS grade† < 3B (arrival)	4		13
RLS grade† 4–6 (arrival)	1	2	6
RLS grade† 7–8 (arrival)	5		1

*Death caused directly or indirectly by the meningitis (six patients died during neurointensive care and four patients died within 2 days to 3.5 months after neurointensive care).
†Reaction Level Scale (RLS).[14]
S. pneumoniae, *Streptococcus pneumoniae*; *N. meningitides*, *Neisseria meningitides*.

ICP > 20 mmHg was measured in seven cases and four patients developed total brain infarction. It is obvious from our results that ICP monitoring should be mandatory in patients with severe bacterial meningitis and that a ventricular catheter drainage system should be the first choice for ICP monitoring as it also provides the possibility to drain CSF and treat increased ICP, which was necessary in 15 out of 26 patients with ventriculostomy in this patient series.

Compared with the results presented by Lindvall et al.,[8] Thiopenthal was used much more frequently in their series, which may be explained by the fact that parenchymatous ICP transducers were their first method of choice for ICP monitoring and by the fact that thiopenthal came before CSF drainage via a ventriculostomy in their escalated protocol. We argue that CSF drainage should be initiated early if possible in order to reduce high ICP as disturbed CSF circulation contributes to the development of intracranial hypertension in bacterial meningitis. Furthermore, deep Tiopental coma treatment is associated with a high risk of side effects in multiple organs.[22]

The follow-up showed that 20 of 36 patients had a favorable outcome (good recovery and moderate disability). Neurointensive care has probably affected the outcome in a positive way, considering that ICP needed to be treated and was successfully decreased in a substantial proportion of the cases. However, we still have to face that many patients have an unfavorable outcome and not all survive. It is important to identify the factors that predict the prognosis in order to guide the specific neurointensive care management in the individual cases. In our series, the mortality rate appeared to be higher among patients with pneumococcal meningitis and patients with a low level of consciousness on admission.

In patients who survived, we observed an improvement between the estimated 6 months outcome score and the late follow-up. It was obvious that if the final results are to be evaluated after bacterial meningitis, the evaluation period should be longer than 6 months.

We attempted to gain a rough impression of which symptoms also persisted after long term follow-up and contributed to the unfavorable outcome. Several patients complained about a variety of lasting neurological symptoms such as hearing loss, dysphasia, dyscalculia, fatigue, headache and problems with short-term memory. Several patients reported vertigo and that difficulty with balance limited their mobility and restricted their possibility to a normal and active life. Several patients complained about psychological difficulties such as mood swings, bad temper and depression. The ultimate goal of neurointensive care is not only to ensure patient survival but also to ensure that the survivors survive in the best possible condition. Early and intensive neuro-rehabilitation after neurointensive care is probably of utmost importance to obtain as good quality of life as possible.

In conclusion, the results of the present study show that severe community-acquired meningitis is still associated with substantial mortality and morbidity, which probably would have been even higher without the neurointensive care. It is therefore important that patients judged to have severe community-acquired meningitis are admitted to critical care units specialist in neurointensive care without delay for ICP monitoring and management according to modern neurointensive care principles.

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