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A Bi-centre Study of the Pattern and Evolution of readily detectable Neurological Sequelae of Acute Bacterial Meningitis

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Summary

Akpede GO, Dawodu SO, Iyasere GEA, Olomu SC. A Bi-centre Study of the Pattern and Evolution of readily detectable Neurological Sequelae of Acute Bacterial Meningitis. *Nigerian Journal of Paediatrics* **2003;30:27.** The pattern and evolution of obvious post-meningitic sequelae were determined in 187 post-neonatal children followed up at two tertiary centres. The pattern of sequelae was classified using previously described schemes, as well as by the number of deficits per child. One hundred and eighty-seven children were assessed on discharge, 157 after six weeks of discharge and 134 after three months. The incidence of sequelae was 40/187 (21.4 percent) on discharge versus 23/157 (14.7 percent) after six weeks ($p = 0.106$) and 18/134 (13.4 percent) after three months ($p = 0.069$) of follow up. Two (1.4 percent) of the 147 children who were apparently normal on discharge had sequelae on follow up, while two (7.4 percent) of the 27 children discharged with major sequelae, died. Among 17 children who were followed up for at least six months, three (18 percent) at ≥6 wks to <6 months and ten (59 percent) at ≥6 months ($p = 0.034$) had persistent deficits. Among the 42 children with sequelae, 29 (69 percent) had major deficits alone ($n = 15$) or with minor deficits ($n = 14$). Fifteen (36 percent) had one, 14 (33 percent) two and 13 (31 percent) ≥3 deficits. There was full or partial resolution of deficits in 15/19 (79 percent) children with sequelae who were treated with and in 6/15 (40 percent) ($p < 0.05$) who were not treated with dexamethasone on admission. There was no significant relationship between the pattern and evolution of sequelae and selected characteristics of the acute illness including its severity, pattern of aetiologic agents and response to initial antibiotic therapy. However, characteristics of the acute illness were significantly ($p < 0.01$) associated with a high incidence of sequelae.

Introduction

MOST reports on acute bacterial meningitis in eveloping countries have focussed on the incidence, resentation, the pattern and susceptibility of aetiologic agents to antimicrobial drugs, and their influence on the incidence of an adverse outcome. This focus is relevant; first, the severity of illness at diagnosis is the principal determinant of the incidence of neurological sequelae;[1-3] secondly, the risk of sequelae differs according to the aetiological agents;[4] and thirdly, treatment failure is associated with an increased incidence of adverse outcome.[5,6] However, knowledge of the pattern and evolution of deficits, and their relationship to aspects of the acute illness is also important, but has received only limited attention. This can determine the need for preferential management practices and rehabilitation. Some data on the pattern of sequelae are available in the form of studies of the general pattern of neurologic diseases.[7] This may not be representative of children who have had meningitis

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More specific reports[8-10] on post-meningitic sequelae have methodological limitations which might limit the interpretation of the data and the conclusions. First, the pattern of sequalae is not described beyond providing a glossary of the individual deficits. Secondly, only the resolution of individual deficits, which might not be representative of the resolution of deficits in children with multiple deficits, nor indicative of the evolution of the pattern of deficits, is described. Thirdly, some of the studies are based on referrals of children with post-meningitic sequelae and not on a cohort followed up from discharge. There is also a paucity of data from tropical Africa on the benefits and constraints to the use of dexamethasone in meningitis.

The aims of the present study were (a) to define the pattern and evolution of neurological deficits in a cohort of children followed up from discharge, (b) to determine the effect of selected indices of the acute illness, such as severity, on the pattern and evolution of deficits, and (iii) to determine the impact of dexamethasone therapy on the incidence, pattern and evolution of sequelae.

Patients and Methods

The study involved children aged one month to 15 years who were treated for acute bacterial meningitis at the University of Maiduguri Teaching Hospital, Maiduguri, from January 1, 1993 to June 18, 1996 and at Irrua Specialist Teaching Hospital, Irrua, from May 17, 1993 to June 30, 2000. Minimum criteria for the diagnosis of acute bacterial meningitis included cerebrospinal fluid (CSF) white blood cell count $\geq 10/mm^3$ with neutrophils $\geq 50\%$, CSF/blood glucose ratio $\leq 50\%$ and CSF protein ≥ 80 mg/dl. Children with an identified pathogen [from CSF culture, Gram stain or latex particle agglutination (only three cases were diagnosed using latex test) or blood culture] were taken as having confirmed bacterial meningitis (CBM), and children without an identified pathogen as having apparent bacterial meningitis (ABM). A standard dose regimen of dexamethasone[13] was used. Other aspects of the management and outcome of children in the study have been described elsewhere.[3,6,11,12] The patients were followed up at the Neurology Clinic fortnightly to bi- or tri- monthly. Assessment was by history and physical examination, but the clinical impression of deafness was confirmed audiometrically.

Classification: Severity of the acute illness was classified using a previously described method.[3] A score of one point each was given for age ≤ 2 years, illness >7 days, convulsions, pre-treatment with antibiotics, presence of shock, coma, abnorm posturing, abnormal muscle tone, abnormal respiratio and focal neurological deficit. Children with a tot score of at least 3/10 were classified as having seve illness, while those with lower scores had illnesses moderate and mild severity.[3] Dexamethasone therap was classified as appropriate if commenced at diagnosi preferably before the first dose of antibiotics, children who had not received antibiotics befo diagnosis.[13] It was inappropriate if given to childre with a history of antibiotic therapy before diagnosi Delayed sterilization of the CSF was defined persistent CSF turbidity and depression of CSF/bloo glucose ratio with or without repeat isolation of th causative organism after 96 hours of treatment Neurological deficits were classified into 'major' an 'minor' as described by Herson and Todd:[1] majo sequelae included quadriparesis, blindness, seizure among others, while minor sequelae included ataxi deafness, hyperkinesis, hemiplegia, etc. Deficits we also classified according to their number per child.

Statistical analysis: Frequencies were compare between dichotomous events using Yates' correcte $\div^2$ test, or Fisher's exact test as appropriate, and betwee groups in an n x n table using group $\div^2$ test. Two-taile p values <0.05 were taken as significant. Epi In Version 6[14] was used for statistical analysis.

Results

Two hundred and forty three children were treate for pyogenic meningitis at the two centres. Differenc in clinical profile, aetiological agents, initial therapy an outcome between children in Maiduguri and Irrua, an between children with CBM and ABM are shown Table I. The incidence, pattern, and resolution sequelae are shown in Table II in relation to stu centre and diagnosis. A significantly higher number children in Maiduguri attended follow-up for ≥ 6 wee ($p < 0.05$). Other differences between the two centr or between CBM and ABM were not significant. A the children with sequelae were therefore poole together for further analysis of the pattern an evolution of deficits to allow for the small numbe involved from each centre and diagnostic group.

Incidence of neurologic sequelae

Overall, 187 children were assessed on discharge, 1 after six weeks of discharge and 134 after three month The incidence of sequelae was 40/187 (21.4 percen on discharge versus 23/157 (14.7 percent) after s weeks ($p = 0.106$) and 18/134 (13.4 percent) after thr months ($p = 0.069$) of follow up.

Table I

Clinical Profile, Aetiological Agents, Initial Treatment and Outcome in Children with Meningitis

	Maiduguri		*Irrua*		*p*		
Clinical profile	*CBM*[a] No. (%) (*n* = 69)	*ABM*[b] No. (%) (*n* = 54)	*CBM*[c] No. (%) (*n* = 51)	*ABM*[d] No. (%) (*n* = 69)	*a vs b*	*c vs d*	*a+b vs* c+d
Age ≤2 years	29 (42)	33 (61)	11 (22)	19 (28)	0.055	0.594	<0.01
Illness >5 days	8 (12)	21 (39)	27 (53)	43 (62)	<0.01	0.399	<0.01
Partial treatment	18 (26)	22 (41)	18 (35)	30 (44)	0.127	0.474	0.28
Convulsions	42 (61)	30 (56)	18 (35)	22 (32)	0.682	0.845	<0.01
Shock	12 (17)	9 (17)	3 (6)	1 (2)	0.892	0.311	<0.01
Unrousable coma	19 (28)	6 (11)	11 (22)	6 (9)	0.043	0.083	0.291
Severely ill	32 (46)	24 (44)	20 (39)	21 (30)	0.975	0.419	0.094
Aetiologic agents:							
N. meningitidis/ GNDC	45 (65)	NA	8 (16)	NA	NA	NA	<0.01
S. pneumoniae/GPDC	12 (17)	NA	20 (39)	NA	NA	NA	<0.05
H. influenzae/GNCB	3 (4)	NA	2 (4)	NA	NA	NA	1.00
Miscellaneous	9 (13)	NA	21 (30)	NA	NA	NA	<0.01
Initial therapy:							
Monotherapy with Pen, Amp, or Chl	14 (20)	10 (19)	23 (45)	39 (57)	0.987	0.292	<0.01
Therapy with Pen or Amp + Chl	47 (68)	36 (67)	21 (41)	21 (30)	0.981	0.305	<0.01
Other regimens	7 (10)	9 (17)	7 (14)	9 (13)	0.426	0.871	0.909
Dexamethasone	42 (61)	24 (44)	19 (37)	23 (33)	0.103	0.801	0.005
Outcome:@							
Died	16/63 (25)	9/46 (20)	10/50 (20)	4/67 (6)	0.628	0.043	0.045

GNDC = Gram negative diplococci, GPDC = Gram positive diplococci, GNCB = Gram negative coccobacilli, NA = not applicable, Pen = penicillin, Amp = ampicillin, Chl = chloramphenicol.
@: Excludes children discharged against advice.

Table II

Incidence, Manifestations, Pattern and Resolution of Sequelae in Survivors

	Maiduguri		*Irrua*		*p*		
	CBM[a] (%)	*ABM*[b] (%)	*CBM*[c] (%)	*ABM*[d] (%)	*a vs b*	*c vs d*	*a+b vs c+d*
Incidence of sequelae:							
No. with sequelae/no. survived	11/47 (23)	11/37 (30)	10/40 (25)	10/63 (16)	0.686	0.376	0.354
Manifestations of sequelae[*,@]							
Motor deficits	7 (64)	10 (91)	6 (60)	8 (80)	0.311	0.629	0.854
Visual or hearing loss	6 (55)	4 (36)	2 (20)	3 (30)	0.669	1.00	0.29
Deficit of other cranial nerves	1 (9)	1 (9)	2 (20)	2 (20)	1.00	1.00	0.40
Afebrile seizures	2 (18)	1 (9)	3 (30)	2 (20)	1.00	1.00	0.445
Hydro- or micro-cephaly	1 (9)	1 (9)	2 (20)	3 (30)	1.00	1.00	0.229
Ataxia	2 (18)	1 (9)	2 (20)	2 (20)	1.00	1.00	0.40
Pattern of sequale:							
No. with major ± minor deficits/no. with sequelae	6/11 (55)	8/11 (73)	6/10 (60)	9/10 (90)	0.659	0.303	0.645
No. with ≥3 deficits/no. with sequelae	4/11 (36)	2/11 (18)	2/10 (20)	5/10 (50)	0.635	0.35	0.836
Resolution of sequelae:							
No. followed up ≥6 wks/no. with sequelae	11/11 (100)	10/11 (91)	6/10 (60)	7/10 (70)	1.00	1.00	0.018
No. with resolved or resolving deficits/no. followed up ≥6 weeks	6/11 (55)	7/10 (70)	3/6 (50)	2/7 (29)	0.659	0.592	0.328

* Summation of the frequencies is greater than the total number with sequelae because the deficits were multiple (>1 type) in some subjects (n = 21): motor deficits + visual or hearing loss, delayed speech or other cranial nerve deficits 6, hydrocephaly or microcephaly + motor deficits +/- seizures, visual loss or other cranial nerve deficit 5, ataxia + hearing loss 3, motor deficits + seizures 3, miscellaneous combinations 4.

@ Motor deficits = quadriparesis 20, hemiparesis 10, monoparesis 2, delay or regression of motor milestones 3, loss of neck control 3, hyperactivity 1, choreiform movements 1, rolling movements of the head 1, coarse tremors 1, delayed speech development 1; hearing loss = 8, visual loss = 9; other cranial nerve palsies = 6th 2, 7th 4; hydrocephalus = 6, microcephalus = 2.

Table III

linical and Laboratory Profile and the Incidence, Pattern and Resolution of Deficits in Children treated with Dexamethasone

linical and boratory	*Children with previously untreated meningitis*			*Children with partially treated meningitis*		
	DEX +VE No. (%) (n = 54)	*DEX –VE No. (%) (n = 65)*	*p*	*DEX +VE No. (%) (n = 26)*	*DEX –VE No. (%) (n = 42)*	*p*
ge ≤2 years	17 (32)	20 (31)	0.908	11 (42)	16 (38)	0.928
everely ill	15 (28)	12 (19)	0.323	13 (50)	20 (48)	0.953
onfirmed bacterial eningitis	33 (61)	29 (45)	0.107	10 (39)	15 (36)	0.976
o. with "usual" rganisms/total no. ith +ve culture or erology	18/20 (90)	14/16 (88)	1.00	5/8 (63)	4/8 (50)	1.00
elayed sterilization of CSF	5 (9)	8 (12)	0.813	8 (31)	14 (33)	0.962
cidence, pattern and resolution of deficits:						
o. with deficits	12 (22)	12 (19)	0.78	9 (35)	9 (21)	0.36
o. with major deficits/no. with deficits	8/12 (67)	9/12 (75)	1.00	5/9 (56)	7/9 (78)	0.62
o. with ≥3 deficits/ no. with deficits	3/12 (25)	2/12 (17)	1.00	3/9 (33)	5/9 (56)	0.637
o. with full or partial resolution/no. followed up	9/11 (82)	4/8 (50)	0.319	6/8 (75)	2/7 (29)	0.132

EX +VE = children treated with dexamethasone, DEX –VE = children not treated with dexamethasone.

ote: The number of DEX +VE children is <108 as in Table I because of the exclusion of children who died r were discharged against advice (*n* = 28).

Follow up status of children who were apparently normal on discharge

Of the 147 children who were apparently normal on discharge, 33 did not attend for follow-up or kept only the first appointment, 49 attended for <3 months, and 65 for three to ten months. Seventeen of those who attended for ≤2 weeks and 25 of those who attended for <3 months were seen later for other reasons after defaulting; they remained normal. Thus, overall, 107 children had a known status at ≥3 months after discharge. Two (1.9 percent) had neurological sequelae that were not evident at discharge. The first, a 14-year old boy with pneumococcal meningitis, presented seven months after discharge with generalised tonic clonic afebrile seizures. This child earlier had five episodes of 'febrile seizures' as an under-5. The second, a five-week old male infant with ABM, had focal afebrile seizures precipitated by non-compliance with phenobarbitone at the age of four months and generalised status epilepticus associated with bronchopneumonia at the age of 11 months. The dose of phenobarbitone had been overdue for review to allow for growth but the mother had defaulted from follow-up. Compared to the twin brother, this child also had delayed motor milestones.

Pattern of neurologic sequelae

There was an average of 2.1 deficits per child, and a total of 89 individual deficits: motor 49 (55 percent), special senses 17 (19 percent; visual loss 9, hearing loss 8), other cranial nerve palsies, six (7 percent; 6th cranial nerve-2, 7th cranial nerve - 4), afebrile seizures, eight (9 percent), abnormal growth in head circumference, eight (9 percent; hydrocephalus 6, microcephaly 2) and delayed speech development one (1 percent). Details of these deficits are contained in Table II. The motor deficits included quadriparesis 20, hemiparesis 10, monoparesis 2, ataxia 7, delay or regression of motor milestones 3, loss of neck control 3, and one each of hyperactivity, choreiform movements, rolling movements of the head, and coarse tremors. Fifteen (36 percent) children had major and 13 (31 percent) minor deficits only, while 14 (33 percent) had major plus minor deficits.

Evolution of deficits

The status of deficits in eight children (two, five and one with major, major+minor, and minor deficits, respectively, at or within two weeks of discharge) was uncertain because they did not attend for follow-up, attended for <6 weeks or died before the 6th week follow-up. Overall, the deficits in nine (27 percent) the 34 children with sequelae followed up for at le six weeks had fully resolved. These included 2/13, 9 and 5/12 children with major, major + minor minor deficits, respectively. The deficits evolved fr major or major plus minor to minor in a further two percent) children and from multiple to single in six percent). Seven of 16 children with single vs 2/18 w multiple deficits (p = 0.052) and 5/12 with minor 4/22 with major +/- minor deficits (p = 0.22) fully resolved deficits.

Satisfactory control of seizures was achieved in th of five children, one of whom was seizure-free fo period of one year. Hydrocephalus became arres within a few months in four of the five children wi one was referred for neurosurgery because of press symptoms/signs. Full resolution was observed in children with ataxia, 2/8 with visual loss, 3/8 w hearing loss, and 7/22 with hemi- or quadripare Seventeen (40 percent) children were followed up at least six months, ten (24 percent) for three to months, and seven (17 percent) for at least six wee while eight (19 percent) attended for shorter peri or not at all, or died before six weeks (one child on Five of the eight children followed up for <6 we showed partial resolution of their deficits bef defaulting. Three (18 percent) of 17 children w attended for ≥6 weeks to <6 months and 10/17 percent) who attended for ≥6 months (p = 0.034) non-resolving deficits.

Two of the 40 children who had sequelae discharge are known to have died. The first had *colif* meningitis, was discharged with spastic quadripare 7th nerve palsy and afebrile seizures, and died a traditional healer's home following prolonged seizu five weeks after discharge. The second had ABM, discharged with quadriparesis and afebrile seizures, died of bronchopneumonia 16 months after discha

Relationship between selected characteristics the acute illness, including dexamethaso therapy, and the incidence, pattern and resolut of deficits

Among the 187 children who recovered, 64 percent) were ≤2 years of age and 60 (32 percent) w severely ill on admission. Eighty seven (47 perce had CBM. The aetiological agent was identified in children; 13 had "unusual agents" (Gram negative ba 10, and *Staphylococcal aureus* 3) and 51 usual age (*Neisseriae meningitidis, Streptococcus pneumoniae, Haemophilus influenzae*). Thirty-five (19 percent) h delayed sterilization of the CSF.

The incidences of sequelae were 28/64 (44 percent) the young and 14/123 (11 percent) in the older ildren (p <0.01), and 28/60 (47 percent) in severely vs 14/127 (11 percent) in non-severely ill children <0.01). It was 8/13 (62 percent) in children with nusual agents" vs 12/51 (24 percent) in those with ual agents (p = 0.016), and 14/35 (40 percent) in ildren with vs 28/152 (18 percent) in those without layed sterilization of the CSF (p = 0.011). The cidence did not differ significantly between CBM and BM (21/87 vs 21/100, p = 0.736). There was no gnificant relationship between the pattern or solution of sequelae and characteristics of the acute ness.

A total of 119 (64 percent) children had no history treatment with antibiotics before diagnosis. Thirty-o of 61 such children in Maiduguri and 22/58 in rua (p = 0.159) were treated with dexamethasone. xty-eight children (36 percent) had partially treated eningitis; 12 of 23 such children in Maiduguri and 1/45 in Irrua (p = 0.154) received dexamethasone. haracteristics of the acute illness, and the incidence, ttern and resolution of sequelae in children treated ith dexamethasone are shown in Table III in mparison with those of untreated children. There ere no significant differences between children who d dexamethasone therapy and those who did not, hether they had partially treated or previously ntreated meningitis. However, in the pooled opulation, that is, all children treated with examethasone vs those not treated, the incidence of ll or partial resolution of sequelae was significantly gher in those given dexamethasone [15/19 vs 6/15, lative risk (95% confidence interval) = 1.97 (1.02, 83), p = 0.049].

Discussion

There is a wide variation (10-47.4 percent) in the cidence of post-meningitis sequelae in developing untries.[10,15-17] The incidences of 15.9 to 29.7 percent this study, depending on the study location Maiduguri vs Irrua) and the nature of the diagnosis CBM vs ABM), are within this range. The overall cidence of permanent neurological sequelae (sequelae ersisting >3 months[18]) of 14.9 percent is also within is range, and compares with the 13.4 percent reported ecently in Libyan children.[18] The pattern of sequelae as not, to our knowledge, been reported from eveloping countries as major and minor with respect individual children, although it is possible to deduce e information with respect to individual deficits from revious reports. Working in a developed country, lerson and Todd[1] reported that in children with *H. influenzae* meningitis, four of 15 with sequelae at discharge and six of 16 on subsequent follow-up had major deficits. Compared to this, the corresponding rates of 29/42 and 25/34 in the present study were high. Differences in the pattern of organisms and severity of illness may account for the difference in rates.[3]

Previous reports from African countries do not contain enough data to help in determining the pattern of sequelae at discharge, but enough data is available to determine the proportion of major sequelae among the individual deficits. In earlier reports, 68 percent deficits reported from Lusaka, Zambia,[19] 67 percent from Ibadan, Nigeria[8] and 35 percent in Enugu, Nigeria[20] were major. In more recent reports, six percent deficits in Accra, Ghana,[17] 47 percent in Benghazi, Libya[18] and 37 percent in Enugu, Nigeria[10] were major. Thus, as with the incidence of sequelae, the proportion with major deficits is also variable, although mostly high. The 50 percent deficit rate obtained in the present study falls within this range.

Reduction of the risk of sequelae in survivors is an important objective in the management of meningitis.[21] Dexamethasone has been recommended for this purpose, but this is controversial.[2,22-25] However, dexamethasone might also have beneficial effects on the evolution of sequelae.[21] This is supported by the results of this study. Although a reduction in the incidence of sequelae could not be demonstrated in this study, this has been shown in other studies from developing countries.[26] This, plus the beneficial effect on the evolution of sequelae,[21] which is also supported by results of this study, suggest that dexamethasone therapy may also be worthwhile in developing countries. Another issue is the selection of patients for preferential treatment with dexamethasone or other agents. Although there are reports which suggest that dexamethasone may be more beneficial in mildly ill patients,[22] it has also been shown to be beneficial in severely ill patients.[27] One conclusion from the results of this study is that unlike the risk of sequelae,[1,3] the pattern and evolution might not be predictable from the characteristics of the acute illness. Therefore, illness severity may not be a sufficient basis for the selection of patients. An important limitation to the use of dexamethasone in developing countries may be the problem of partial treatment with antibiotics. Misuse of antibiotics is common in developing countries[28] and dexamethasone therapy might not be beneficial in partially treated meningitis.[13] Other limitations to its use have been discussed by Tefuarani and Vince.[16]

The evolution of sequelae in this study is encouraging, even allowing for the limited follow-up attendance. First, although 67 percent of the sequelae were major

or mixed initially, those in 30 percent of evaluable patients evolved from major/mixed to minor or none. Second, 62 percent of evaluable patients had fully (29 percent) or partially (32 percent) resolved deficits. This is similar to reports of other series,[29,30] including those on individual deficits.[9,10]

Two findings in this study were of a low incidence, but their occurrence is, nonetheless, a cause for concern: First, 6.9 percent of the 29 children with major sequelae died from morbidities arguably related to the sequelae. The problem of delayed mortality associated with major sequelae may, therefore, be important. Second, two children who were initially assessed as normal, subsequently developed afebrile seizures. One had a preceding history of recurrent febrile seizures, a risk factor for afebrile seizures.[31] The second child subsequently manifested with other deficits. Afebrile seizures as a complication of meningitis are usually associated with other deficits,[29] but this is not invariable.[29] Thus, the absence of clinical abnormalities on discharge, an assessment which may not be infallible, might not preclude the subsequent development of seizures. Whatever the explanation, the phenomenon of delayed appearance of sequelae is rare but not unknown,[29] and underlies the need for follow-up of even apparently normal patients.

Other than the overall pattern of sequelae, the incidence of individual sequela may also be of interest. Hearing loss has in particular been emphasized.[21,32] The 3.7 percent incidence among 187 survivors in this study is within the 1 to 4 percent range of profound or total impairment reported from developed countries.[32] Afebrile seizures have also been reported in 5 to 44 percent of survivors.[21] The incidence of 4.3 percent in the present study may represent an underestimation for two reasons: First, its apparent rarity contrasts with the high incidence of seizures at diagnosis and during treatment.[3,15] Seizures during the illness are a strong predictor of the risk of major sequelae.[1,3,29] Second, many families still believe that traditional or spiritual therapy is better for seizure disorders and might not have taken their children to formal health facilities. In this study, one of those with sequelae who subsequently died was taken to the traditional healer on the insistence of the grandmother.

Certain aspects of the methodology in this study merits further discussion. First, only "readily detectable sequelae" were reported because full assessment of all the children could not be done. For example, whereas all children should have a hearing assessment post-meningitis,[33] only those who were deaf on clinical assessment had formal audiometric assessment for confirmation. This was due to lack of facilities. Formal psychometric assessment for cognitive deficits was also not performed, but non-formal assessment of mo of those who recovered fully, during presentations f other reasons, indicated that they were doing well. addition, we adopted the classification of Herson a Todd[1] because the degree of functional impairme expected from the deficits seems to have been the ba for the classification. However, other studies[29,34] ha shown that the pathological basis for some "mino deficits such as hemiparesis might be no less gross th that of major deficits. Also, some "minor" deficits m be no less important. Thus, hearing loss has importa effects on speech development and education.[33] added the number of deficits as an additional meth of classification because a child with multiple defic would be expected to suffer more function impairment, as well as require more resources f rehabilitation, than one with fewer deficits. Anoth aspect is the poor compliance with follow-u appointments which was a major problem as only percent of the patients with sequelae attended f upwards of six months. The speed of resolution sequelae may be a factor, and children who attend for ≥6 months were less likely to have fully or partia resolved sequelae. Neuroimaging other than plain sk radiography was not done, mainly because of lack facilities. Nonetheless, none of the children had course of illness suggestive of mass lesions as complication. These are also quite rare as complicatio of meningitis.[2] Finally, children with ABM we included because they fulfilled other criteria for t diagnosis of bacterial meningitis. There were only few significant differences between CBM and AB as discussed in previous reports.[6,12]

We conclude that most patients with post-meningi sequelae in developing countries have major or maj plus minor deficits and deficits may resolve partia or fully in about two-thirds of affected children; t pattern and evolution of sequelae are not clea predictable from the characteristics of the acute illn on admission, but dexamethasone therapy may associated with a more favourable evolution of defici

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