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Recurrent meningitis in children: etiologies, outcome, and lessons to learn

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Abstract

Purpose Recurrent meningitis in children is a rare condition. However, its early recognition is important in order to prevent serious complications. This study aims to review cases of recurrent meningitis in children.

Methods This is a retrospective study that included children diagnosed with recurrent meningitis and who were followed at child neurology clinic at the Jordan University Hospital from January 2001 to June 2017.

Results Thirteen patients were included (nine males and four females). Age of first episode of meningitis ranged from 2 months to 9.5 years. The delay in diagnosis of the underlying cause after the first episode ranged from 6 months to 2.5 years. Underlying causes included inner ear malformation in one patient, skull fractures in two, and dermal sinuses (thoracic spinal and occipital dermal sinus) in two patients. No identifiable cause was found in eight patients. *Streptococcus pneumoniae* was identified in four (31%) patients, *Staphylococcus aureus* in two (15%), and no organism was isolated in seven (54%). Three patients (23.1%) developed neurological sequel including developmental delay, limb spasticity, and epilepsy. Two patients had sensorineural hearing loss related to meningitis, and two patients had sensorineural hearing loss mostly related to their original disease.

Conclusion A detailed history, examination, and thorough investigations are necessary to determine the underlying cause of recurrent meningitis. In addition, in patients with positive CSF bacterial culture, finding the underlying etiology is very likely.

Keywords Recurrent meningitis · Children · Congenital inner ear malformation · Mondini dysplasia · Dermal sinus · Jordan

Introduction

Recurrent meningitis in children is a rare condition. However, it is an important condition to be recognized since if a cause can be found, further complications can be avoided. Known etiologies of recurrent meningitis include cranial anatomical defects such as skull fractures, chronic parameningeal infections, recurrent benign lymphocytic meningitis, antibody or complement deficiency, and hyposplenism [1].

In a systemic meta-analysis by Tebruegge et al. in 2008 [1], a total of 363 cases of recurrent bacterial meningitis were identified to be reported in 144 publications in the last two

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decades. Most of publications (73%) reported only a single patient with recurrent bacterial meningitis; only 15 reports (including reports on adults and children) described five or more cases. Only very few pediatric series were reported since then [2, 3]. The exact incidence of recurrent bacterial meningitis is not known. Drummond et al. in 1999 [4] reported that only 1.3% of children admitted with meningitis had at least one previous episode of bacterial meningitis. In this study, we describe the etiologies and outcome of recurrent meningitis in 13 Jordanian children who were followed at a child neurology clinic at Jordan University Hospital.

Methods

This is a retrospective study that included children (1 month–18 years) who were treated for more than one episode of meningitis and were followed at the Child Neurology clinic at Jordan University Hospital from January 2001 to June 2017.

Recurrent meningitis was defined as a second episode of meningitis resulting from a different pathogen than the first or if it was due to the same organism but occurred more than 3 weeks after the completion of therapy for the initial episode [5]. A relapse was defined as meningitis due to the same organism within 3 weeks of the completion of therapy for the initial episode [5].

Data from medical records was collected to determine the age at onset of first episode of meningitis, number of episodes, types of organisms, investigations performed, the underlying cause for the recurrence, age at diagnosis of the correct cause, neurological sequel, and the total follow-up period. Patients who presented to the neurosurgical department due to trauma and did not present to the child neurology clinic were not included. The institutional review board committee (IRB) of Jordan university Hospital has approved this study.

Results

Thirteen patients were identified to have recurrent meningitis: nine males and four females. Apart from one patient who had mitochondrial disorder, all the patients were healthy prior to the first episode of meningitis. Age at first episode of meningitis ranged from 2 months to 9.5 years. Following the first episode, patients suffered two to five episodes of recurrent meningitis before investigations to identify a possible etiology. The delay in determining the etiology in patients with an underlying cause ranged from few months to 2.5 years.

Investigations included temporal bone CT which was available for ten patients, brain MRI for 12 patients, and spinal MRI for 7 patients. Serum immunoglobulin, C3, C4, and CH50 levels were performed to all the patients and were normal. CSF values at presentation were variable suggesting either bacterial or aseptic meningitis. Bacterial pathogen was isolated from the CSF or dermoid cyst of six patients: *Streptococcus pneumoniae* from the CSF of four patients and *Staphylococcus aureus* from dermoid cysts in two patients. Seven patients had no identified bacterial pathogen and were mostly due to recurrent viral infections. Viral work up was not done because of unavailability in our hospital at time of presentation.

An underlying cause for the recurrent meningitis was identified in five (39%) patients. Causes included inner ear malformation (Mondini dysplasia) in one patient, skull fracture following trauma in two, and dermal sinuses in two (in the thoracic spinal region and in the occipital region). Eight patients had no identifiable cause.

The total follow-up period ranged from 6 months to 13 years. Neurological sequel related to meningitis included global developmental delay in three (23.1%) patients, spasticity in one (8%), epilepsy in two (15.4%), and hydrocephalous in two (15.4%). Two patients (15.4%) had sensorineural hearing loss related to meningitis, and two patients (15.4%) had sensorineural hearing loss mostly related to their original disease. Death due to meningitis was not reported.

None of the patients received prophylactic antibiotic. Following identification of the final etiology, surgical interventions were carried out to all patients except the patient with Mondini dysplasia due to lack of experience in our hospital at time of diagnosis.

Pitfalls in history and examination in our series include the following cases:

Case 1: This child had her first episode of meningitis at 3.5 years of age. Three episodes of meningitis occurred and revealed *S. pneumoniae* from CSF on two occasions. The child had a dermal sinus in the lumbosacral region for which a spine MRI was done and it revealed diastematomyelia and a tethered cord. The dermal sinus was erroneously thought to be the cause for recurrent meningitis, and surgical repair was done for the sinus. However, a fourth episode of meningitis occurred, and eventually, a temporal bone CT revealed the presence of Mondini dysplasia. The case was previously described in more details [6].

Case 2: This child had her first episode of meningitis at 2 years of age. A total of four episodes of meningitis occurred before investigations including a brain MRI at 4 years of age which revealed a dermoid cyst in the cerebellum. This cyst was connected to an occipital scalp sinus: the portal of entry for the bacteria causing the meningitis. The patient underwent suboccipital craniotomy with excision of the dermoid cyst and evacuation of pus that

yielded *S. aureus*. Failure to listen well to the history and failure of proper examination of the scalp lead to this delay in diagnosis. The mother complained on several occasions about the presence of a papule in the scalp of her child which often drained a yellowish material, but unfortunately, the mother's words were ignored, and the scalp was never examined. The case was previously described in more details [7].

Case 3: This child had his first episode of meningitis at age of 6 months. A second episode of meningitis occurred at the age of 1.5 years. Subsequent investigations including a spine MRI revealed an abscess in the spinal cord with dermoid cyst connected to a thoracic dermal sinus—the portal of entry of the organism. Failure of proper examination of the back at an early stage lead to missing of the dermal sinus with subsequent two episodes of meningitis complicated by hydrocephalous, bilateral sensorineural hearing loss, spasticity, and severe global developmental delay. Figure 2 shows angiopathy/vasculitis in the spinal cord as a complication of infection.

Case 4: This child had her first episode of meningitis at age of 5 years following trauma to the head and a fracture in the right frontal region. The delay in surgical closure for more than a year lead to two episodes of *S. pneumoniae* meningitis, complicated by hydrocephalous with subsequent epilepsy and developmental delay. An earlier surgical intervention could have saved the patient such a complication.

Case 5: The child had his first episode of meningitis at age of 9.5 years. Two episodes of meningitis occurred and both revealed *S. pneumoniae*. The child fell off the bicycle before the first episode. However, the history missed the details of falling down as he had a direct trauma to the face. He developed meningitis few weeks later. An axial CT scan for the brain was done for him, which missed a fracture in the base of skull. It was not until he had his second episode (6 months after the trauma) that a coronal CT was done and revealed a fracture in the cribriform plate: the source of portal of entry for the recurrent meningitis. Surgical repair was done following this result.

Table 1 shows the characteristics of all patients.

Table 2 shows CSF fluid analysis at presentation and other diagnostic work up for the 13 patients

Figure 1 shows CT scan of temporal bone for patient n 2, revealing labyrinthitis ossificans as a complication of meningitis.

Figure 2 shows an MRI cut of the spine of patient n 4 revealing patchy ill-defined lesions of bright T2 signal and possible microhemorrhage that could be related to angiopathy or vasculitis as a complication of infection.

Discussion

In this study, we are reporting 13 children with recurrent meningitis. An underlying cause for the recurrent meningitis was identified in 39% of the patients. However, in patients with positive bacterial cultures from CSF or drained cysts, an underlying cause was identified in 80% of the patients. The most common causes of recurrent meningitis in the literature were related to anatomical problems (59%), immunodeficiency (36%), and parameningeal infections (5%) [1].

In our series, identified causes of recurrent meningitis were all related to an anatomical problem. Causes included congenital inner ear malformation, dermal sinuses, and skull fractures.

Congenital inner ear malformations are becoming more recognized recently due to the development of sophisticated imaging studies [8]; they are considered now to be an important cause for recurrent meningitis [9].The organisms reported in meningitis due to inner ear malformation and CSF leak include *S. pneumoniae* [4, 10], *Haemophilus influenza* [11], and *S. aureus* [12]. It is believed that the organisms reach the CNS through a defect caused by the inner ear malformation [8]**.** One of our patients (case n 1) had Mondini dysplasia, which is a rare congenital inner ear malformation. Mondini dysplasia consists of one and half coils of the cochlea instead of the normal two and half coils, cystic dilatation of the common apical chamber with absence of interscalar septum between the middle and apical coil and a hypoplastic modiolus [13]. This patient had in addition to the mondini dysplasia a dermal sinus in the lumbosacral region, diastematomyelia, and a tethered cord which mislead the diagnosis. The pathogen, *S. pneumoniae*, that was detected from the CSF cultures on several occasions should have oriented towards a basal skull or inner ear portal of entry rather than a dermal sinus. In addition, the presence of one anomaly should not have ruled out the presence of other anomalies. Nevertheless, it is noteworthy to mention that the association of Mondini dysplasia with diastematomyelia is extremely rare [6]**.** Surgical correction of the inner ear malformation or defect is necessary to prevent recurrent pyogenic meningitis in these patients [14]. Unfortunately, no surgical intervention was done to our patient due to lack of surgical experience in our hospital at time of diagnosis. In addition, none of our patients received prophylactic antibiotics. However, the role of prophylactic antibiotics in patients known to have CSF leaks has been controversial; a Cochrane review failed to show benefit of prophylactic antibiotics, and there is no proof that it can prevent the recurrence [15]. Furthermore, once antibiotic prophylaxis is started, it should be continued for many years with the risk of colonization by more resistant flora [16].

Other known causes for recurrent meningitis in our series include dermal sinuses. Dermal sinuses are rare causes of recurrent meningitis and are most commonly located in the

Table 1 Clinical characteristics of the patients

Number	Sex	Age at first episode	No. of episodes	Pathogen	Etiology (age at diagnosis)	Outcome (total follow-up period)
1	F	3.5 years	4	*S. pneumoniae* on 2 episodes	Inner ear malformation (Mondini dysplasia) (5 years)	- Normal development - Unilateral sensorineural hearing loss probably related to inner ear malformation - (2 years)
2	M	5 years	5	*S. pneumoniae* on 3 episodes	No identifiable cause Probably related to anatomical skull defect that was not detected by the neuroimaging facilities available at time of diagnosis (Fig. 1 shows labyrinthitis ossificans as a complication of meningitis) (7 years)	- Normal development - Unilateral sensorineural hearing - (2 years)
3	F	2 years	4	None in CSF *S. aureus* from the dermoid cyst	Occipital sinus–cerebellar dermoid cyst (4 years)	- Normal development - (2 years)
4	M	6 months	2	*S. aureus* from dermoid cyst	Thoracic spinal sinus–spinal dermoid cyst (1 year)	- Spastic, global developmental delay, hydrocephalous, sensorineural hearing loss - (7 years)
5	F	5 years	2	*S. pneumoniae* in one episode	Skull fracture: linear fracture of the right frontal, parietal, and cribriform plate bones (6 years)	- Hydrocephalous, developmental delay, poor school performance and epilepsy - (9 years)
6	M	9.5 years	2	*S. pneumoniae* in one episodes	Skull fracture: fracture in the cribriform plate of the ethmoid bone on the right side (10 years)	- Normal development - (1 year)
7	M	8 years	3	None	No identifiable cause	- Normal development - (3 years)
8	M	3.5 years	4	None	No identifiable cause	- Developmental delay and sensorineural hearing loss on left side mostly related to mitochondrial cytopathy - (2 years)
9	M	4 years	2 (including one episode of meningoencephalitis as seen on brain MRI)	None	No identifiable cause	- Global developmental delay and epilepsy - (13 years)
10	M	7.5 years	2	None	No identifiable cause	- Normal development - (4 years)
11	M	5 years	2	None	No identifiable cause	- Normal development - (5 years)
12	M	2 months	2	None	No identifiable cause	- Normal development - (6 months)
13	F	2.5 months	2	None	No identifiable cause	- Normal development - (4 years)

lumbosacral region; sinuses in other areas are extremely rare [1]. However, because dermal sinuses can be diagnosed by physical examination alone, they should be excluded early in the evaluation of every patient with recurrent meningitis [17]. In our series, two patients had dermal sinuses: one thoracic and one cerebellar. The incomplete history and examination in addition to the rare locations of the cysts in these two patients lead to failure of quick recognition. *S. aureus* was the

Table 2 CSF fluid analysis at presentation and other diagnostic work up for the 13 patients

Patient number	Complete blood count (CBC) Hemoglobin (HB) WBC (neutrophils /lymphocytes) Platelets	CSF analysis WBC: (neutrophils/ lymphocytes) Protein (mg/dl) Sugar in CSF /blood (mg/dl)	Immunoglobulin and subclasses	Complement level (C3, C4, CH50)	Other diagnostic work up
1	NA	WBC:215(40/60) Protein:74 Sugar:50/90	Normal	Normal	CSF culture: *Streptococcus pneumoniae*
2	Hb: 10.5 WBC: 14 (82/5) Platelets: 442	WBC:1000(65/35) Protein: 540 Sugar: 4/70	Normal	Normal	CSF culture: *Streptococcus pneumoniae*
3	Hb: 12.9 WBC: 7.9 (70/18) Platelets: 516	WBC: 1400(88/12) Protein: 55 Sugar: 30/NA	Normal	Normal	CSF culture: negative Acid fast bacilli in CSF: negative CSF cytology: normal Culture from dermoid cyst: *Staphylococcus aureus*
4	Hb: 13.7 WBC: 27.8 (53/36) Platelets: 520	NA	Normal	Normal	Culture form dermoid cyst: *Staphylococcus aureus* CSF culture: negative
5	Hb: 11.3 WBC: 25.6 (89.1/5.7) Platelets: 280	WBC: 400 (84/16) Protein: 17 Sugar: 69/112	Normal	Normal	CSF culture: *Streptococcus pneumoniae*
6	Hb: 10.5 WBC: 30.1 (94/3) Platelets: 360	WBC: 3320 (88/12) Protein: 80 Sugar: 32/132	Normal	Normal	CSF culture: *Streptococcus pneumoniae*
7	Hb: 13.8 WBC: 7.9 (66/27) Platelets: 316	WBC:100 (15/85 Protein: 70 Sugar: 52/103	Normal	Normal	PCR herpes negative CSF culture negative
8	Hb: 8.7 WBC: 10.7 (59/33) Platelets: 642	WBC: 15 (5/95) Protein: 30 Sugar: 48/91	Normal	Normal	CSF culture negative
9	Hb: 10.7 WBC: 10.2 (64/28) Platelets: 275	WBC: 150 (15/85) Protein: 37 Sugar: 52/NA	Normal	Normal	CSF culture negative
10	Hb: 13 WBC: 8.5 (77/15) Platelets: 229	WBC: 300 (15/85) Protein: 60 Sugar: 55/88	Normal	Normal	CSF culture negative
11	Hb: 11.4 WBC: 16.3 (73/13) Platelets: 374	WBC: 60 (55/45) Protein: 28 Sugar: 43/65	Normal	Normal	CSF culture negative
12	Hb: 9/26 WBC: 13.2 (23/68) Platelets:649	WBC: 420 (15/85) Protein: 98 Sugar: 49/NA	Normal	Normal	CSF culture negative
13	Hb: 9.9 WBC: 23.7 (30/61) Platelets: 546	WBC: 105 (52/48) Protein: 72 Sugar: 58/82	Normal	Normal	CSF culture negative

NA data missing in the file

pathogen recovered from both cysts. Of note, *S. aureus* as a cause of spontaneous meningitis is rare outside of the neonatal period and is usually associated with an anatomic or immunologic defect. In a series of 40 pediatric cases of meningitis caused by *S. aureus*, only 20% of patients had no known predisposing central nervous system abnormality [12]. Cutaneous sinuses extending to the subarachnoid space can usually be found with a thorough physical examination that includes shaving the head if the scalp cannot be fully visualized. Infections caused by Gram-positive organisms such as *S. aureus* are frequently associated with this type of anatomic defect. However, other organisms including enteric Gram-negative bacilli and anaerobes have been reported [12, 18].

Other possible reported causes for recurrent meningitis include acquired defects which are seen most commonly in the context of head injury or neurosurgery. Traumatic dural lesions can remain asymptomatic for decades [19]; however, prompt recognition and repair of the defect prevent further episodes of meningitis and ensures a good outcome for neurological development [20].

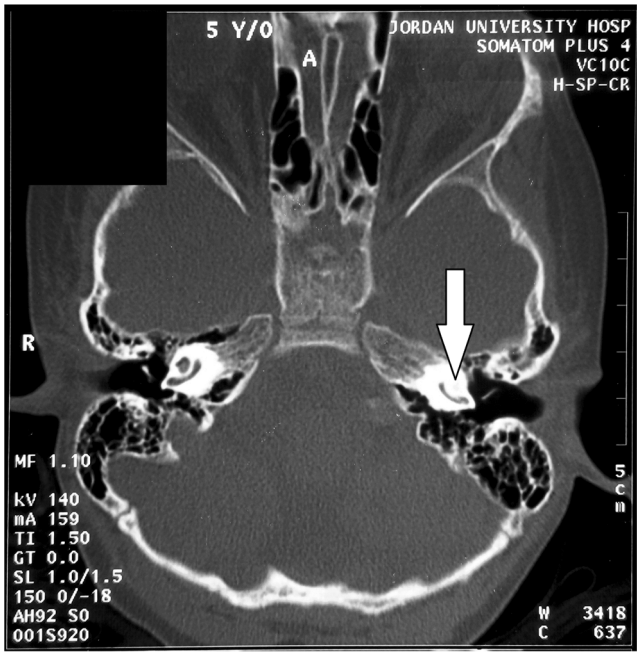

Fig. 1 CT scan of temporal bone for patient no. 2, revealing labyrinthitis ossificans as a complication of meningitis

The classic site for fractures is at the delicate cribriform plate of the ethmoid bone [21]. In our series, two patients had acquired fractures involving the cribriform plates. Both patients suffered from *S pneumoniae* infection. Case no. 6 had delayed diagnosis because of incomplete history about the mechanism of falling and had only an axial CT scan which missed the cribriform fracture. Thin section cranial CT offers a relatively easy, reliable, and non-invasive method of delineating anatomical defects in recurrent meningitis [22] while axial cranial computed tomography may fail to identify defects in the basal ethmoidal area and cribriform plate and so give false reassurances [22].

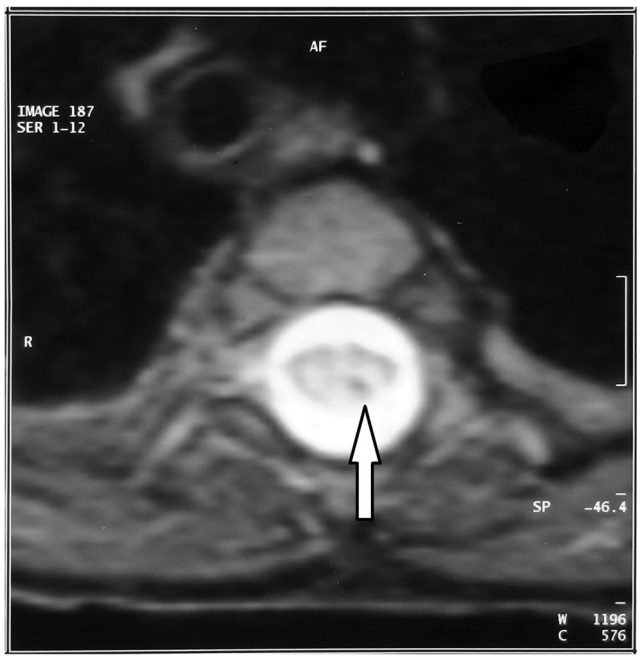

Fig. 2 An MRI cut of the spine of patient no. 4 revealing patchy ill-defined lesions of bright T2 signal and possible microhemorrhage that could be related to angiopathy or vasculitis as a complication of infection

Apart from the identified etiologies described above, no possible cause could be identified in eight (62%) patients. Patient no. 2 had four episodes of *S pneumoniae meningitis*; we think that this patient had mostly an anatomical skull defect that was not identified by our available techniques of neuroimaging at time of diagnosis. The recurrence in the other seven patients could be most probably related to an incidental recurrence caused by viral infections. Various viruses can lead to aseptic meningitis. Mollaret's meningitis is a variant of recurrent aseptic meningitis, and the most common identified agent is herpes simplex virus type 2 [23]. Unfortunately, we did not examine the CSF for viral DNA since the test was not available in our hospital at that time.

In addition to the abovementioned causes, other rare causes of recurrent aseptic meningitis reported in the literature include immune deficiency. However, evaluation for immune deficiency is not indicated in healthy individuals without history of recurrent infections before the episode of meningitis or when risk factors for the human immunodeficiency virus (HIV) infection is not present [24]. Other rare causes include autoimmune diseases such as sarcoidosis, lupus, and Behcet's disease [25] and drug-induced meningitis such as non-steroidal anti-inflammatory drugs, anticonvulsants, antibiotics, immunosuppressive, and chemotherapy agents [25].

As for the outcome, death was not observed in our series, ongoing with previous reports in the literature [10]. However, global developmental delay with or without neurological deficit was observed in one third of our patients. Earlier diagnosis which could have been achieved through a better history taking and examination could have saved these patients the handicap.

Conclusion

Although recurrent meningitis in children is a rare condition, nevertheless, it may lead to severe neurological handicap. Detailed history and examination are the golden clues to early diagnosis. The presence of a positive bacterial pathogen can often orient towards the possible etiology, and one should bear in mind that the presence of one anomaly does not rule out the presence of other associated anomalies.

Compliance with ethical standards

The institutional review board committee (IRB) of Jordan university Hospital has approved this study.

Conflict of interest All authors declare no conflict of interest.

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