Transient complete heart block as a presentation of acute (bacterial) meningitis: a case report

Astrid Declercq, MD[1]; Arnout Bruggeman, MD[2]; Hans Vandekerckhove, MD[1]

Institutions:

1. Department of Cardiology, General Hospital Sint-Lucas, Groenebriel 1, 9000 Ghent, Belgium
2. Department of Neurology, General Hospital Sint-Lucas, Groenebriel 1, 9000 Ghent, Belgium

Corresponding author
Astrid Declercq
Cardiology Resident
Department of Cardiology, General Hospital Sint Lucas
Groenbriel 1, 9000 Ghent, Belgium
astrid1.declercq@ugent.be

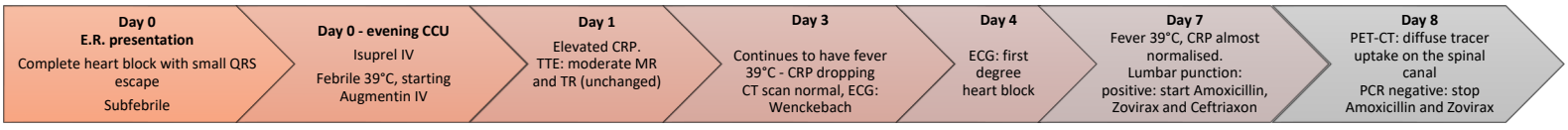
Abbreviations:
CSF = cerebrospinal fluid
ECG = electrocardiogram
PMR = polymyalgia rheumatica
TTE = transthoracic echocardiography

Introduction

Acute meningitis is defined as an inflammation of the meninges covering the brain. It is most commonly caused by a viral or bacterial infection, although rarely a fungal infection can also be the culprit. Common symptoms of meningitis include headache, nuchal rigidity, high fever, and potential alterations in consciousness. A definitive diagnosis requires a lumbar puncture (also known as a spinal tap) to collect cerebrospinal fluid (CSF). The CSF typically exhibits an elevated number of leucocytes (pleocytosis). In adults, >5 leucocytes/µL is defined as elevated. The presence of a pathogen in the CSF confirms bacterial or viral meningitis. Though in many cases of adult meningitis, the specific pathogen responsible for the infection remains undetected. Symptoms of meningism and the identification of relevant bacteria in the blood may suggest bacterial meningitis[1].

Although rare, the cardiac conduction system may be involved in systemic infections, resulting in life-threatening arrhythmias[2].

Summary figure



ER = emergency room
CCU = cardiac care unit
IV = intravenously

Case presentation

We present the case of an 87-year-old women, with a medical history of multivalvular disease (moderate mitral-and tricuspid regurgitation) as well as polymyalgia rheumatica (PMR) for which she had been on a slow tapering regimen of corticosteroids for over 1,5 years.

She presented at the emergency department with symptoms of exercise intolerance and headache. Upon assessment, her blood pressure was 107/59 mmHg, heart rate was 44 beats/min, oxygen saturation was 97% on room air, and she had a subfebrile temperature of 37.3 °C. Clinical examination revealed no cardiac murmurs. Electrocardiogram (ECG) showed a complete heart block with small QRS escape ***(figure 1a).*** The patient was on a chronic daily low dose of beta-blocking therapy (bisoprolol 2,5 mg). Laboratory results indicated a slightly elevated C-reactive protein (CRP 33 mg/l), chronic kidney disease with an estimated glomerular filtration rate (eGFR) of 40 ml/min, and diffuse mildly elevated liver function tests. Troponin levels were slightly elevated (32,4 ng/l), while CK-levels were within normal range (85 U/l). Radiographic imaging did not show any signs of pneumonia, but showed mild signs of congestion. Transthoracic echocardiography (TTE) revealed a normotrophic left ventricle with normal function, along with known moderate mitral and tricuspid valve regurgitation. The aortic valve was normal, without added structures or new onset regurgitation.

The patient was admitted to the cardiac care unit (CCU) and beta-blocking therapy was discontinued while Isuprel (isoprenaline hydrochloride) was initiated at a dose of 0,05 gamma due to symptomatic bradycardia. Subsequently, the patient developed a high fever of 39°C in the evening. Various cultures including blood, urine, Covid/Influenza/RSV PCR were obtained, and empirical antibiotic therapy with amoxicillin-clavulanic acid was started. The following day, laboratory results revealed an elevated CRP level of 153 mg/l. Despite a decrease in CRP levels under antibiotic treatment and negative culture results, the patient continued to experience persistent high fever (38°C – 39°C) while headache remained unchanged, without signs of nuchal rigidity or temporal artery swelling. The patient also reported back pain with varying locations. Neurological assessments remained normal.

CT imaging of the thoracic and abdominal regions revealed no signs of acute infectious pathology. Antibiotic therapy (amoxicillin-clavulanic acid) remained unchanged. On day 3 of hospitalization and antibiotic therapy, a change in heart rhythm to second degree heart block type 1 (Wenckebach) was seen ***(figure 1b)***, and Isuprel was discontinued. From the fourth day of antibiotic therapy onwards, the patient maintained sinus rhythm with first-degree heart block ***(figure 1c).*** Lyme disease was ruled out as a cause of the infectious syndrome and conduction abnormalities, as indicated by negative Borrelia IgM and IgG tests.

Despite ongoing fever on the seventh day of antibiotic therapy, the patient's CRP levels in the blood were decreasing. A lumbar puncture was performed by a neurologist due to persistent headache as the only clinical symptom, revealing a diagnosis of meningitis with elevated white blood cell count (predominantly lymphocytes and monocytes), increased protein levels, and slightly decreased glucose levels in the cerebrospinal fluid (CSF) **(table 1)**. Empirical therapy was initiated with high-dose Ceftriaxone, Amoxicillin 4x 2 gr and Zovirax (3 x10 mg/kg/day). As soon as cultures and PCR results from the CSF turned out negative, Amoxicillin and Zovirax were discontinued, while high dose Ceftriaxone was continued for 2 weeks, due to suspicion of bacterial meningitis (with negative cultures after already receiving one week of intravenously amoxicillin – clavulanic acid treatment). Cerebral MRI could exclude brain abscess.

A PET-CT scan was already scheduled to rule out a potential exacerbation of polymyalgia rheumatica or associated vasculitis. The scan was performed the day after the lumbar punction and indicated widespread tracer uptake in the spinal canal, predominantly in the cervical region, with no tracer uptake observed on the heart valves ***(figure 2).***

The patient was subsequently transferred to the neurological department, where intravenous Ceftriaxone (2 x 2 grams) was administered for a duration of 2 weeks. Throughout the hospitalization period, the patient

maintained sinus rhythm, with no occurrences of complete or second-degree heart block observed during monitoring on the neurology ward. Following a few weeks of care on the revalidation unit, the patient was discharged home. Two months post initial presentation with complete heart block, ambulatory 24-hour heart rhythm monitoring was conducted, revealing a nocturnal first-degree atrioventricular block, with normalization of the PR interval during the day. No recurrence of second or high-degree heart block were detected during this monitoring period. The patient also fully recovered clinically.

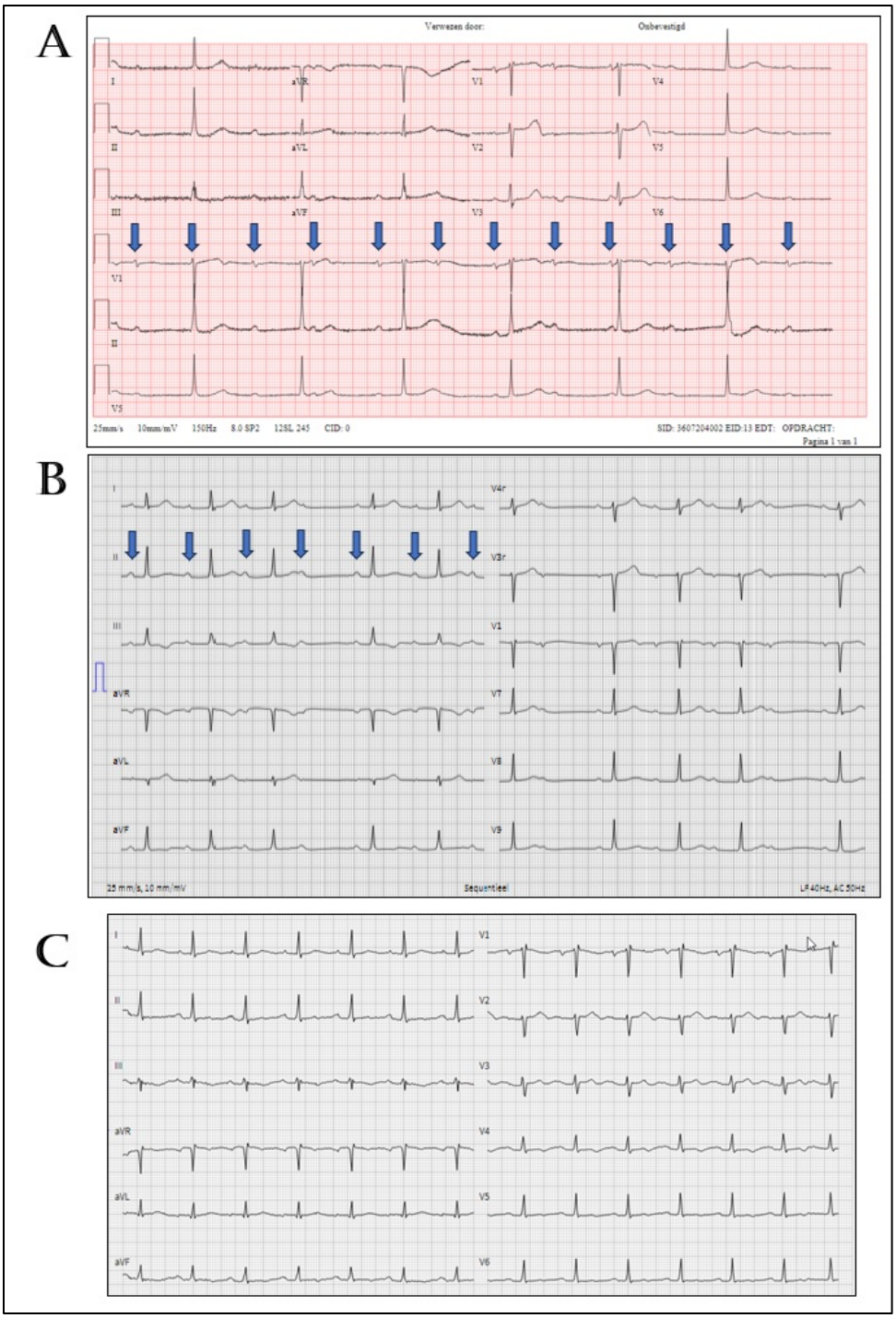


Figure 1. A) Electrocardiogram at presentation, revealing complete heart block with small QRS escape; **B)** ECG on day 3 of hospitalisation, showing second degree heart block type I (Wenckebach); **C)** ECG on day 4 of hospitalisation, showing sinus rhythm with first degree heart block

Results of lumbar punction – CSF analyses		
	Reference range	**Results CSF**
WBC	0 – 5 /µl	32/µl
Cytology	/	mostly lymphocytes and monocytes
RBC	0-5/µl	100/µl
Protein CSF	15 – 40 mg/dl	125 mg/dl
Protein serum	*65 – 80 g/dl*	*50 g/l*
Albumin	10 – 30 mg/dl	75,5 mg/dl
Albumin serum	*35 – 52 g/l*	*30 g/l*
IgG/albumin ratio	0,3 – 0,7	0,5
		No oligoclonal IgG bands
Glucose	40-70 mg/dl	38 mg/dl
Bacterial cultures	Negative	Negative
PCR	Negative	Negative *(Escherichia coli, Haemophilus influenzae, Listeria monocytogenes, Neisseria meningitidis, Streptococcus agalactiae, Streptococcus pneumoniae, Cytomegalovirus, Enterovirus, Herpes simplex virus type 1 and 2, human Herpesvirus type 6, Parechovirus, Varicella zoster virus and Cryptococcus gattii / Cryptococcus neoformans)*

Table 1. Results from the lumbar puncture with reference range

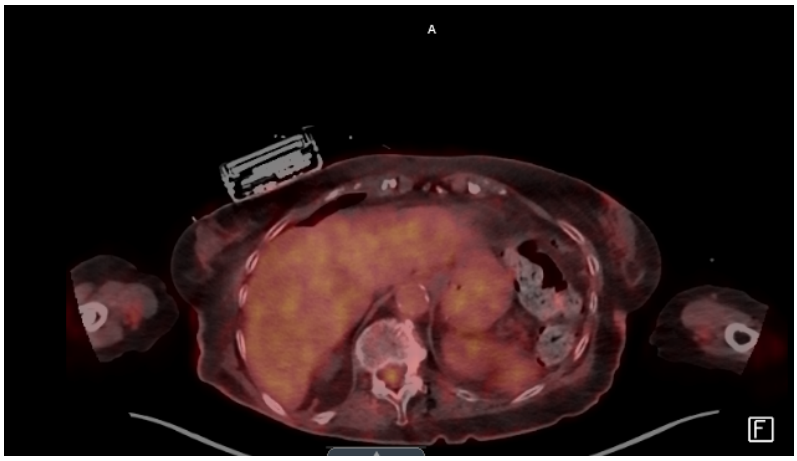


Figure 2: PET-CT image of diffuse tracer uptake on the spinal canal.

Discussion

We describe a case of transient complete heart block, as an initial manifestation of acute meningitis. Prior literature has reported an association between complete heart block and acute (bacterial) meningitis. These patients often require temporary transvenous pacing, but most of the cases show spontaneous recuperation

without the need of permanent pacemaker implantation. Most described involved pathogens include Neisseria meningitidis and Haemophilus influenzae[2-4].

Besides complete heart block, other ECG-changes that have been described in bacterial meningitis include ST elevation, ventricular tachycardia, prolongation of QT-segment and sick sinus syndrome. Though most described ECG changes and arrhythmias resolve with improvement of symptoms of meningitis and consequent reduction of intra-cranial pressure[4].

The precise mechanism of cardiac involvement remains uncertain, although several potential mechanisms have been proposed. In cases with bacteraemia, direct infiltration of heart tissue by the organism can cause myocarditis or endocarditis, with consequent destruction of the AV junction or the bundle of His. Moreover, severe sepsis can cause ischemic alterations that may lead to ST-T changes and predispose to both brady- and tachyarrhythmias. Delayed cardiac arrhythmias while recovering from bacterial meningitis are very rare and may be linked to the deposition of immune complexes[4].

The pathophysiology of cardiac involvement in meningitis cases without bacteraemia is less clear. Cardiac arrhythmias may be mediated by abnormalities in sympathetic tone due to elevated intracranial pressure. Autonomic neural stimulation from the hypothalamus or increased circulating catecholamines may contribute to myocardial damage. Meningococcal endotoxemia might trigger coagulation and platelet thrombosis, resulting in myocardial vessel ischemia, necrosis, and impaired cardiac function[4].

Conclusion

Though no causing pathogen could be detected, our case demonstrates the possibility of cardiac involvement in acute meningitis in adult patients. It is important to notice that most cardiac arrhythmias in meningitis are self-limiting, and require close monitoring without intervention.

References

1. Griffiths MJ, McGill F, Solomon T. Management of acute meningitis. Clin Med (Lond). 2018 Mar;18(2):164-169. doi: 10.7861/clinmedicine.18-2-164. PMID: 29626023; PMCID: PMC6303447.

2. Morriss JH, Gillette PC, Barrett FF. Atrioventricular block complicating meningitis: treatment with emergency cardiac pacing. Pediatrics. 1976 Dec;58(6):866-8. PMID: 995515.

3. Allan S. Detsky, Irving E. Salit. Complete heart block in meningococcemia, Annals of Emergency Medicine,Volume 12, Issue 6,1983,Pages 391-393,ISSN 0196-0644, https://doi.org/10.1016/S0196-0644(83)80473-9.

4. Abu Omar R, Fink D, Megged O. Cardiac arrhythmias in meningococcal meningitis - case report and review of the literature. Acta Paediatr. 2013 Jun;102(6):e279-80. doi: 10.1111/apa.12205. Epub 2013 Mar 6. PMID: 23414146.

Data availability

The data underlying this article will be shared on reasonable request to the corresponding author

Disclosures

The authors have nothing to disclose.

Funding:

None

Word count: 1023 words