

Meningococemia Complicated by Purulent Pericarditis

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Introduction

Meningococemia is an infectious syndrome caused by Gram-negative diplococci *Neisseria meningitidis*, a germ that has man as its natural reservoir, inhabiting and colonizing the nasopharynx.¹ After nasal infection, the germ can reach the bloodstream and spread throughout the body.¹ The most frequent clinical syndrome triggered by meningococemia is acute purulent meningitis,² which, in our community, represents a major public health problem and is responsible for many cases, especially in autumn and winter, even in large urban centers. Between 1939 and 1962, 600,000 cases of meningococcal disease were estimated worldwide, with more than 100,000 deaths - mostly in children.² As the germ spreads throughout the body, it can lead to other infectious complications, in addition to meningitis, with suppurative arthritis being the most commonly found, followed by pannophthalmus. Cardiac involvement is more often expressed by purulent pericarditis.³ Pericardial involvement is uncommon and usually without clinical manifestations, which makes it difficult to characterize.³

This study aimed to report the case of meningococemia exposed by meningitis associated with pericardial involvement.

Case report

A 38-year-old woman, with no prior comorbidities or chronic use medication, reported a 7-day condition consisting of myalgia, arthralgia, 39 °C fever, erythematous-violet patches on lower and upper limbs, and headache. She was referred from the emergency room of another hospital where she was evaluated, and benzathine penicillin (1,200,000 units) was prescribed. The patient was advised to return after 72 hours for reevaluation of suspected dengue fever. She returned to the same emergency room after 1 week, with persistence of fever and symptoms previously reported, despite continuous use of dipyron. Meningococemia was suspected due to the skin condition (petechial rash on limbs), with volume expansion initiated with 0.9% saline and antibiotic therapy with oxacillin 2 g intravenously every 4 hours, and ceftriaxone 2 g intravenously every 12 hours.

The patient was then referred to the Emergency Room of Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, where she presented, on physical examination,

102 bpm heart rate and 100 x 80 mmHg blood pressure. Cardiovascular auscultation showed regular heart rhythm, with hypophonic sounds and no murmurs. Erythematous-violet lesions on the hands and feet were also observed. Laboratory tests showed hemoglobin 12.5 g/dL; hematocrit 40%; 95,000 platelets; 23,880,000 leukocytes; C-reactive protein of 286 mg/dL; sodium 144 mEq/L; potassium 3.4 mEq/L; and magnesium 2.8 mEq/L. After initial care, cerebrospinal fluid (CSF) was collected, revealing growth of Gram-negative diplococci (Table 1).

Chest X-ray showed increased cardiac area (Figure 1). Transthoracic echocardiography (Figure 2, Videos 1 and 2) revealed normal biventricular function, severe pericardial effusion with hyperechogenic areas, suggesting effusion with increased density and signs of ventricular filling restriction (right atrial systolic collapse, right ventricular diastolic collapse, inferior vena cava without respiratory variation, marked respiratory variation of atrioventricular flows). Computed tomography of the chest (Figure 3) confirmed severe pericardial effusion. Cranial tomography did not show any abnormalities, but magnetic resonance imaging of the skull showed high T1 signal and foci of moderate enhancement in the cortical sulci, suggesting leptomeningeal inflammatory/infectious process. The patient was evaluated by the chest surgery team, and pericardial drainage was performed at a surgical center, with 500 mL of purulent secretion. Pericardial biopsy and culture collection were performed. The patient remained for more 5 days under intensive care, evolving clinically stable, without the need for vasoactive drugs; control echocardiogram showed marked decrease in pericardial effusion and no signs of restriction to ventricular filling (Figure 4). The patient was discharged asymptomatic after 57 days of treatment.

Discussion

Acute pericarditis is an uncommon manifestation of meningococcal infection. The incidence of acute pericarditis

Table 1 - Laboratory parameters for cerebrospinal fluid (CSF) and pericardial fluid.

Liquid	CSF	Pericardial
Aspect	Cloudy	Purulent
Cellularity	10,000 cells (neutrophils: 74%; lymphocytes: 18%; monocytes: 8%)	Numerous polymorphonucleates
Lactic dehydrogenase, U/L	-	5.310
Glucose mg/dL	-	38
Search for acid-alcohol-resistant bacilli	Negative	Negative
Gram	Gram-negative diplococcus	Gram-negative diplococcus
Culture	Negative	Negative

Keywords

Pericarditis; Meningitis; Echocardiography.

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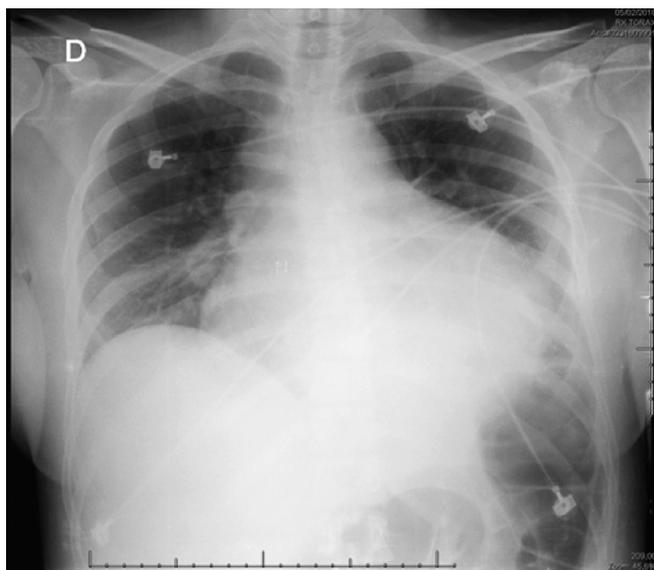


Figure 1 – Anteroposterior chest X-ray showing increased cardiac area.

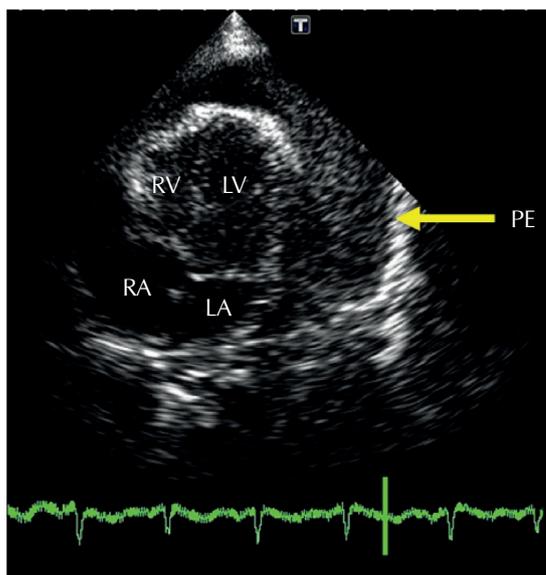
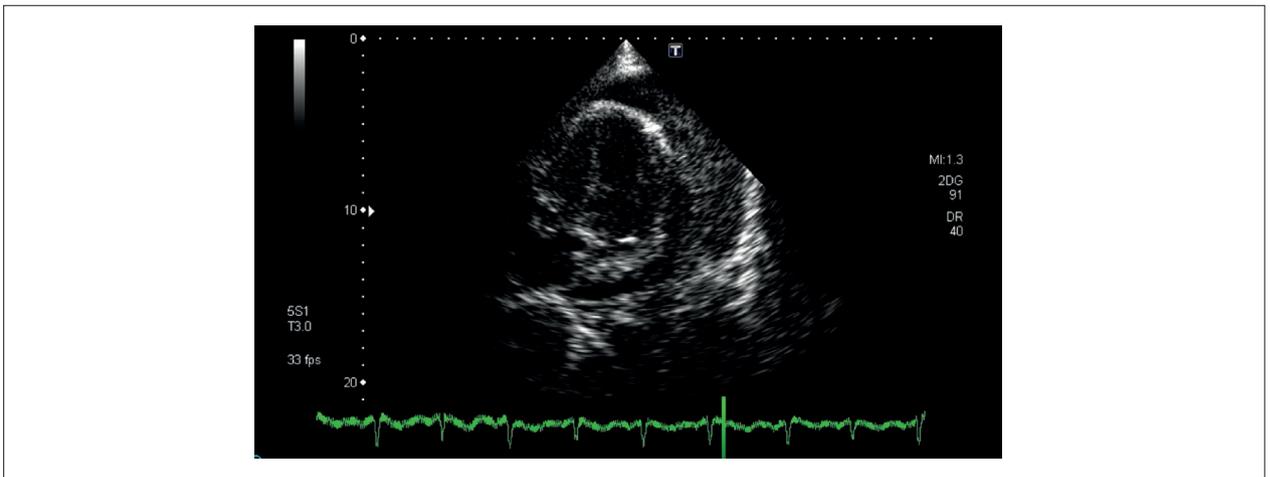


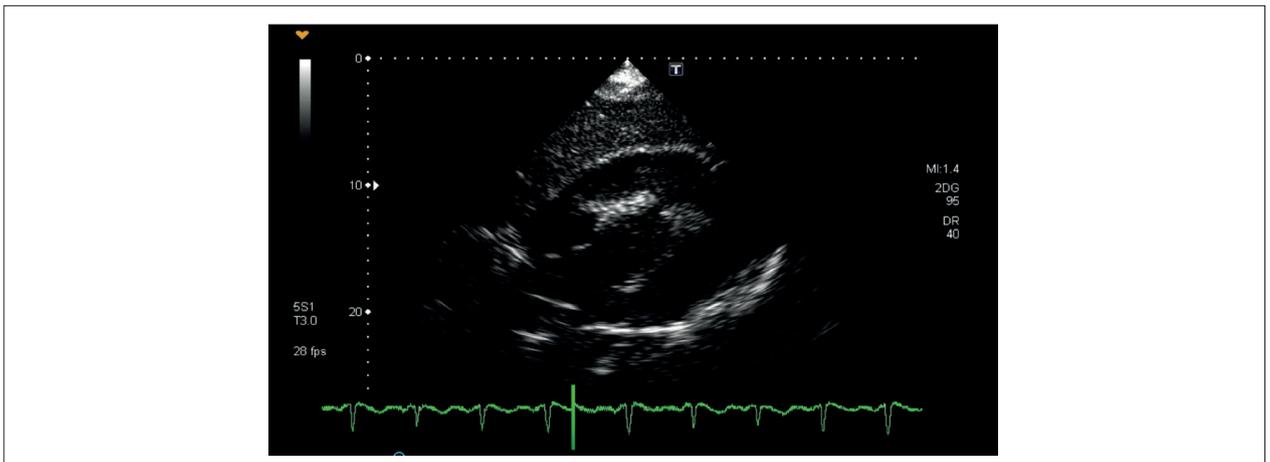
Figure 2 – Transsthoracic echocardiogram (4-chamber apical view) showing massive pericardial effusion with increased density, associated with the lateral wall of the left ventricle. Right ventricle; LV: left ventricle; AO: aorta; PE: pericardial effusion; RA: right atrium; LA: left atrium.

complicating meningococcal infection varies from zero to 19%⁴ and implies worse prognosis. Its most common symptoms are chest pain (80%), fever (70%) and dyspnea (50%).⁴ Meningococcal pericarditis can be classified into three types, based on the pathophysiological process: disseminated meningococcal disease with pericarditis, isolated meningococcal pericarditis and meningococcal reactive pericarditis. In disseminated meningococcal disease, pericarditis occurs as a consequence of the direct invasion of

the pericardium by the pathogen during episodes of bacteremia and, clinically, there is manifest meningococcal disease. Isolated meningococcal pericarditis (also called primary meningococcal pericarditis) is defined as purulent pericarditis with culture of pericardial fluid or positive blood culture for *N. meningitidis*, and there is no evidence of meningitis or other manifestation of meningococemia. On the other hand, reactive meningococcal pericarditis is a late complication of meningococcal disease, occurring 6 to 16 days after commencement of treatment;



Video 1 – Transsthoracic echocardiogram (4-chamber apical plane), showing large pericardial effusion with increased density, adjacent to the lateral wall of the left ventricle, with signs of ventricular filling restriction. Watch the video here: http://departamentos.cardiol.br/dic/publicacoes/revistadic/2019/v32_3/video_v32_3_meningococemia_complicada_ingles.asp



Video 2 – Transsthoracic echocardiogram (4-chamber subcostal plane), showing severe pericardial effusion with hyperechogenic areas, suggesting effusion with increased density and signs of ventricular filling restriction (right ventricular systolic and right ventricular diastolic collapse). Watch the video here: http://departamentos.cardiol.br/dic/publicacoes/revistadic/2019/v32_3/video_v32_3_meningococemia_complicada_ingles.asp

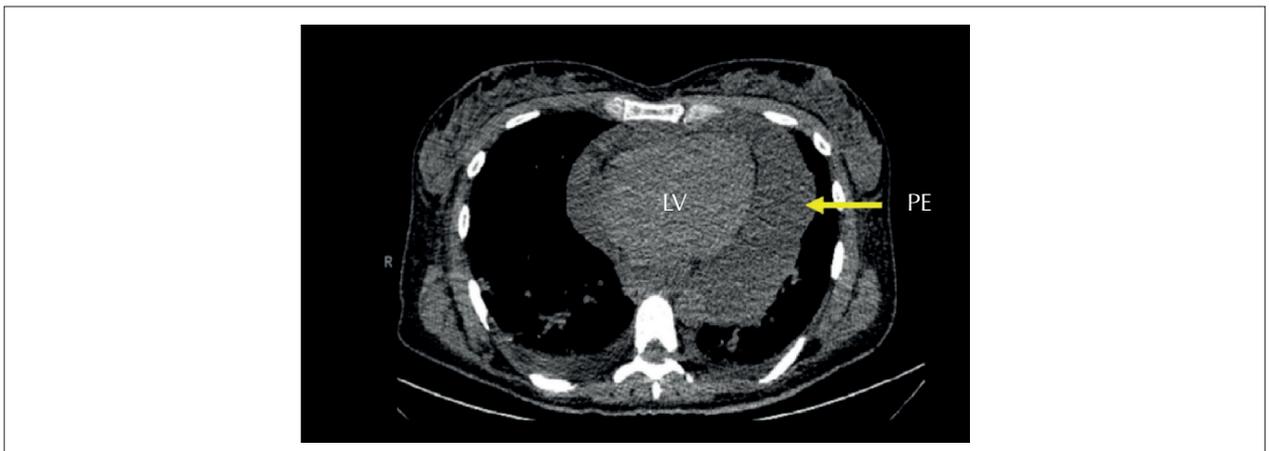


Figure 3 – Computed tomography of the chest, showing massive pericardial effusion on the left, more related to the lateral wall of the left ventricle. LV: left ventricle; PE: pericardial effusion.

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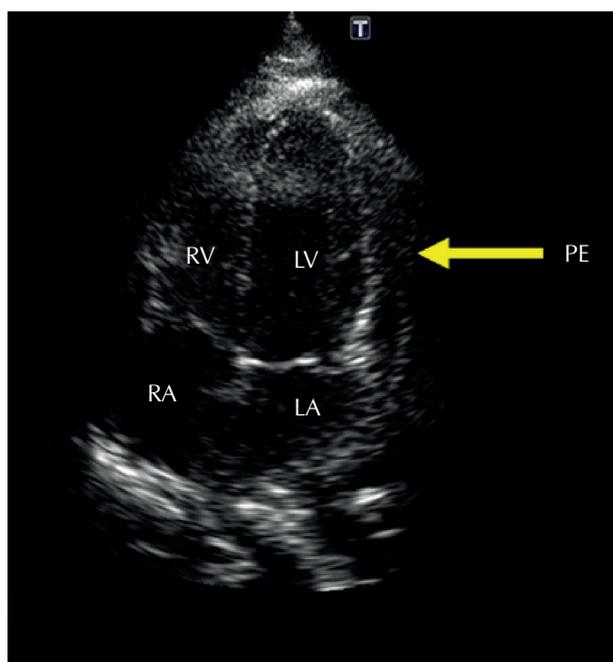


Figure 4 – Transthoracic echocardiogram (4-chamber apical view) showing reduction in the amount and echogenicity of the pericardial effusion 5 days after drainage. RV: right ventricle; LV: left ventricle; PE: pericardial effusion; RA: right atrium; LA: left atrium.

its etiology is attributed to an immunological process, and the infectious agent is not present in the pericardial fluid.⁴

In this case, we found purulent pericarditis with cardiac tamponade associated with meningococcal meningitis. Diagnosis of meningococemia was made from the neurological picture along with skin conditions, and the CSF result was compatible with bacterial infection. Additionally, the infectious nature of the pericardial fluid (purulent appearance, increased lactic dehydrogenase, low glucose, numerous polymorphonuclear leukocytes and Gram evidencing Gram-negative diplococci) confirmed the diagnosis of meningococcal pericarditis as a complication of the clinically disseminated disease. This form of presentation usually occurs in children and, more commonly, it appears within 1 week after the onset of infection, being uncommon in a young adult patient (38 years), as in our case.⁴

Regarding the diagnostic exams, smear or culture of pericardial liquid aspirated before the administration of antibiotics often evidences *N. meningitidis*; however, our results showed only the presence of Gram-negative diplococci, possibly due to the previous use of antibiotics. Meningococcal pericarditis is responsive to antibiotics and is usually not severe when treated early and appropriately. However, its prognosis is variable and largely dependent on the response to disseminated meningococcal infection.

Pericardial effusion with tamponade is uncommon.⁴ What makes this case interesting is the fact that the patient is an adult and evolves with pericardial effusion with echocardiographic signs of ventricular filling restriction — not expected in this type of condition. Echocardiography is a highly useful test in cases of pericarditis, allowing not only the diagnosis, but also suggesting

the etiology due to the pericardial fluid characteristics.⁴ Besides, echocardiography makes it possible to help in the conduct, guiding the drainage of pericardial fluid. Monitoring for the presence of early and late pericarditis in the course of meningococcal disease is important and should include electrocardiography and echocardiography. Recognizing the different types of pericarditis associated with meningococcal infection is important, since different therapeutic approaches are required and lead to different prognoses.

In conclusion, purulent pericarditis is a rare potentially severe complication of meningococcal meningitis and should be suspected in patients with chest pain, abnormalities on the electrocardiography or enlargement of the mediastinum on chest radiography. It is important to emphasize the role of echocardiography for its diagnosis and therapeutic guide, contributing to the favorable clinical evolution of the disease.

Authors' contributions

Research creation and design: Rodrigues LD, Barros LC, Kowatsch I, Andrade JL, Rodrigues ACT; Data acquisition: Rodrigues LD, Barros LC, Kowatsch I, Andrade JL, Rodrigues ACT; Data analysis and interpretation: Kowatsch I, Andrade JL, Rodrigues ACT; Manuscript writing: Rodrigues LD, Barros LC, Kowatsch I, Andrade JL, Rodrigues ACT; Critical revision of the manuscript for important intellectual content: Kowatsch I, Andrade JL, Rodrigues ACT.

Potential Conflicts of Interest

There are no relevant conflicts of interest.

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