Case report

Pericarditis due to *Neisseria meningitidis* serogroup W135

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**ABSTRACT**

*Neisseria meningitidis* is a well-recognized cause of bacterial meningitis. Although less common, *N. meningitidis* can also involve extra-meningeal sites, including the pericardium. The frequency of such extra-meningeal clinical manifestations differs depending on *N. meningitidis* serogroup. *N. meningitidis* serogroups C and W135 have been reportedly associated with extra-meningeal meningococcal disease more frequently including pericarditis. In general, meningococcal pericarditis is categorized into three etiologies; primary meningococcal disease, secondary disease due to disseminated meningococceemia, and reactive form as an immunologic complication. Importantly, meningococcal pericarditis can cause massive pericardial effusion with cardiac tamponade that can lead to cardiogenic shock. We report a case of pericarditis due to *N. meningitidis* serogroup W135 secondary to disseminated meningococcal disease.

**Introduction**

*Neisseria meningitidis* is the leading cause of bacterial meningitis with substantial morbidity and mortality occurring particularly in children and young adults. Although the main clinical manifestations of *N. meningitidis* infection are meningitis and meningococcemia, extra-meningeal involvement, such as pericarditis and arthritis, has also been described in the literature. Meningococcal pericarditis can occur as a primary meningococcal infection or a secondary form resulting from disseminated meningococceemia or a reactive form occurring later as an immunologic complication. Various *N. meningitidis* serogroups, particularly serogroups C and W135, have been reportedly associated with extra-meningeal meningococcal disease more frequently. We report a case of meningococcal pericarditis due to serogroup W135 complicated by massive pericardial effusion and cardiac tamponade in a patient presenting with meningococcal meningitis and meningococceemia.

**Case report**

A 32-year-old man with no significant past medical history was in his usual state of health until 3 days before presentation when he suddenly developed severe headaches and fever. The patient was found to have altered mental status and taken to a local emergency room. No sick contact, exposure history or recent travel was reported by his family. The patient was born in the United States and his immunization status was up-to-date, including a quadrivalent meningococcal vaccine. In the emergency room, the patient was minimally responsive to painful stimuli and appeared ill. His vital signs were blood pressure, 89/60 mmHg, pulse rate, 110/min, respiratory rate, 23/min, temperature, 102.1 °F and oxygen saturation 92% on room air. His neck was stiff with nuchal rigidity. The right jugular vein was distended. His heart sounds were weakly audible without apparent murmurs. His lungs were clear to auscultation. His abdomen was soft and not tender. Scattered petechial rash was observed on his palms and soles.

Laboratory studies disclosed a white blood cell count of 18,500 cells/mm\(^3\) with 90% neutrophils, hemoglobin of 13.7 g/dL, and platelets of 202,000 cells/mm\(^3\). The level of sodium was 136 mmol/L, potassium 5.1 mmol/L, bicarbonate 23 mEq/L, urea nitrogen 18.0 mg/dL, creatinine 0.6 mg/dL, erythrocyte sedimentation rate 55 mm/h and C-reactive protein 40.2 mg/L. The patient was empirically started on ceftriaxone, vancomycin, and ampicillin for possible bacterial meningitis. CSF analysis showed the following values: glucose, 23 mg/dL; protein, > 300 mg/dL; and WBC count, 5040 cells/mm\(^3\) with 88% neutrophils. Gram stain of the spinal fluid showed gram-negative diplococci which was later identified as *N. meningitidis*. Electrocardiography revealed ST elevation with prolonged PR interval in multiple leads (Fig. 1). Transthoracic echocardiography (TTE) demonstrated massive pericardial effusion with global left ventricular systolic dysfunction (ejection fraction 35%). A chest radiograph showed enlarged cardiac silhouette and pulmonary congestion.

Urgent pericardiocentesis drained approximately 700 mL of turbid fluid. Gram-negative diplococci were seen on gram stain of the pericardial fluid. Cultures of the pericardial fluid and blood yielded *N. meningitidis*. The isolate was further identified as serogroup W135 *N.

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meningitidis. Vancomycin and ampicillin were discontinued, and the patient completed a 2-week course of ceftriaxone. The patient’s altered mental status improved, and repeat blood cultures were sterile. The follow-up outpatient TTE demonstrated normalized cardiac function with resolution of pericardial effusion.

Discussion

We report a case of meningococcal pericarditis due to serogroup W135 N. meningitidis complicated by cardiac tamponade in an otherwise healthy young individual. Although N. meningitidis is a well-known pathogen for bacterial meningitis, N. meningitidis can also involve extra-meningeal sites, such as pericardium.

Meningococcal pericarditis was originally reported in 1918 by Herrick [1]. In this report, 12 cases in 280 cases of meningococcal meningitis were noted to have pericarditis. In the case series reported in 1971 [2], 17 cases of pericarditis were found in 334 cases of meningococcal meningitis. In addition, there have been many case reports of meningococcal meningitis, including the primary form, in the literature [3–6]. Generally, meningococcal pericarditis is categorized into 3 groups; primary meningococcal infection, secondary meningococcal disease resulting from disseminated meningococcemia, and a reactive form occurring later after the initiation of meningococcal treatment as an immunologic reaction [7].

N. meningitidis is a gram-negative diplococcus possessing a polysaccharide capsule. N. meningitidis are distinguished by serogroup typing. At least 13 serogroups have been identified: A, B, C, D; X, Y, Z; E, W-135; H, I, K; and L [8–13]. Group specificity is determined by chemical composition of the capsular polysaccharide; for instance, the capsular polymer of serogroup W135 is composed of alternating sequence of D-galactose and N-acetyleneuraminic acid [14].

According to the available literature, there may be a high incidence of extra-meningeal manifestations associated with W135 N. meningitidis infection. The analysis of 2019 strains of N. meningitidis by Vienne et al. from 1999 to 2001 suggest that the extra-meningeal complications, including pericarditis, arthritis, and pneumonia, occur at higher rates with W135 serogroup than with serogroup B or C [15]. In this study, meningococcal pericarditis was 4 times more common among patients with W135 serogroup than those with serogroup C.

Meningococcal pericarditis can cause massive pericardial effusion with life threatening cardiac tamponade leading to cardiogenic shock [16]. Our patient initially presented with characteristic signs and symptoms of meningitis. The electrocardiography findings related to hemodynamic instability was highly concerning for pericarditis complicated by cardiac tamponade. Prompt and timely cardiac intervention led to a good clinical outcome. Gram stain of the pericardial fluid showing gram-negative diplococci was highly indicative of secondary meningococcal pericarditis due to meningococcemia and meningococcal meningitis. In this case, we concluded that pericarditis was due to hematogenous seeding of N. meningitidis into the pericardium after cultures of the spinal fluid, blood, and pericardial fluid all yielded N. meningitidis.

Conclusion

N. meningitidis can present with extra-meningeal involvement, including pericarditis. The frequency of such extra-meningeal meningococcal disease differs depending on N. meningitidis serogroups determined by chemical component of polysaccharide capsule. Serogroup W135 may involve extra-meningeal sites more frequently than other N. meningitidis serogroups. Recognition of meningococcal pericarditis is of great clinical importance since it can lead to massive pericardial effusion with cardiac tamponade that can lead to high morbidity and mortality.

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