

INFECTIVE ENDOCARDITIS CAUSED BY GROUP G STREPTOCOCCUS WITH MULTIPLE CEREBRAL EMBOLI

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Summary: The group G streptococcal endocarditis is a rare form of infective endocarditis. In this form of infective endocarditis, serious neurological complications most commonly develop. We reported this case because of its being an unusual form of infective endocarditis that was caused by Group G Streptococcus. We also reviewed the literature. The patient was admitted to infectious disease service with a presumptive diagnosis of central nervous system infection. Blood cultures were positive for group G streptococcus. There was a mass on the posterior surface of the mitral valve which was 2x 2.5 cm in length on the echocardiography. In the cranial computerized tomography of our patient, slightly increased contrast media uptake was observed in the both parietal lobes, in the both frontal lobes, and in the anterior areas of right occipital lobe. Therefore, this case was assumed as infective endocarditis caused by group G streptococcus with multiple cerebral emboli. Ceftriaxone was given for 4 weeks and gentamicin was given for 2 weeks, and progressive improvement of the patient's condition was seen.

Key words: Infective endocarditis; Cerebral emboli; Group G streptococcus

Introduction

Infective endocarditis is an infection of the endocardial structures, including the cardiac valves and mural endocardium. There are both persistent bacteremia and anatomic lesions of the valves in infective endocarditis. Infective endocarditis may result in life-threatening complications. Therefore, precise clinical and microbiologic diagnosis is important and appropriate antibiotic therapy must be considered. In recent years, advances both in clinical microbiology and in the imaging techniques, especially transthoracic and transesophageal echocardiography, have made the diagnosis of infective endocarditis more accurate. Due to these recent developments Durack et al. proposed a new diagnostic criteria that known as Duke criteria in 1994. Duke criterias were revised in year 2000 (2,5,10,11,13).

Many different microorganisms may cause infective endocarditis, but the majority of cases are caused by streptococci, particularly *Streptococcus viridans*. Infective endocarditis caused by beta hemolytic streptococci is infrequently seen. The frequency of group G beta hemolytic streptococcal endocarditis reported in the literature is quite rare; approximately 1 % in accord with various studies. The view of the authors is that Group G streptococcal endocarditis is often more aggressive, and is more frequently associated with embolic, neurologic and cardiac complications than endocarditis caused by viridans streptococci. We re-

ported this case since Group G streptococci is unusual causative agent of infective endocarditis (1,9,12,16,18).

Case report

A 29 year old female, was admitted to our hospital emergency department with the complaints of fever and loss of consciousness. It has been learned that she had had weakness, fatigue and diffuse myalgias for one week. On admission she was unconscious and was appeared malnourished. Her temperature was 38.5 °C, her pulse was 84 beats/min and her respiratory rate were 18 breaths/min, her blood pressure was 100/70 mmHg. Bilateral subconjunctival petechias were present. A grade 2/6 systolic murmur was audible at the left sternal border. There were left lower extremity paresis and right lower extremity plegia. The remainder of her physical examination yielded nothing remarkable. The patient had no significant medical history and in her family history her father had cardiac disturbances.

The laboratory investigation results were as follows: The leukocytes count was $14.7 \times 10^9/l$; hematocrit level was 22.8 %, mean corpuscular volume was 84.3 fl, and platelet count was $96 \times 10^9/l$. The peripheral blood smear showed 87 % polymorphonuclear leukocytes, 7 % lymphocytes, 6 % monocytes. Erythrocyte sedimentation rate was 50 mm/h. Alanine transferase was 0.73 μ kat/l, aspartate amino-

transferase was 0.78 μ kat/l, alkaline phosphatase was 1.83 μ kat/l, γ -glutamyl transpeptidase was 1.38 μ kat/l, lactate dehydrogenase was 5.02 μ kat/l, total bilirubin was 5.13 μ mol/l, total protein was 51 g/l, albumin was 18 g/l, blood urea nitrogen was 3.99 mmol/l, creatinine was 70.7 μ mol/l, sodium was 143 mmol/l, potassium was 4.8 mmol/l, chloride was 107 mmol/l, prothrombin time was 14.4 second. Urine analysis results were normal. Computed tomography of the brain (without contrast medium) was unremarkable. Chest radiography did not reveal any infiltrates.

A mildly turbid cerebrospinal fluid was yielded with lumbar puncture. When we analyzed it, glucose concentration was 3.55 mmol/l, protein concentration was 290 mg/l, and cell count was 57×10^6 /l (90 % neutrophils, 10 % lymphocytes). Gram stain and cultures of cerebrospinal fluid were negative.

The patient was admitted to infectious disease service with a presumptive diagnosis of central nervous system infection. After blood, urine, pharynx and cerebrospinal fluid specimens were obtained for culture, ceftriaxone (2x2 g/day, i.v.) was initiated empirically due to the leucocytosis and shift to the left.

In the cranial computerized tomography of the patient on the second day of hospitalization, only slight increase of contrast media uptake was observed in both parietal lobes, both frontal lobes and in the anterior areas of right occipital lobe.

Echocardiography had been done to the patient since the patient had cardiac murmur and signs of infection. On the echocardiography, there was a mass on the posterior surface of the mitral valve which was 2x2.5 cm in length that might belong to a vegetation. According to the control echocardiographies, this mass was decreased in size.

Blood cultures became positive for group G streptococcus and the organism was sensitive to penicillin, ceftriaxone, and vancomycin. In addition, the level of the complement 3 and 4 was normal. Thyroid function tests were normal. Brucella agglutination test was negative. VDRL test was negative. Tuberculin test was negative. ANA test was negative.

As a result, this case was accepted as infective endocarditis caused by group G streptococcus with multiple cerebral emboli. Gentamicin (2 x 80 mg/day, i.v.) was added to the treatment at the eighth day of her hospitalization and Ceftriaxone was switched to 2x1 g/day, i.v. from 2x2 g/day, i.v. Ceftriaxone was given for 4 weeks and gentamicin was given for 2 weeks. The fever of the patient was begun to decline after the six days and progressive improvement of the patient's condition was seen. Cardiovascular surgeons did not think to operate the patient. The patient was followed by our clinic for 5 weeks and was sent to a rehabilitation center after antibiotic therapy.

Discussion

Group G streptococci may be found as a member of normal flora of the pharynx, intestine, vagina and skin. It

may cause infections such as endocarditis, septic arthritis, pharyngitis, puerperal sepsis, peritonitis, pleuropulmonary infections, meningitis, cellulitis, otitis media and neonatal sepsis. The frequency of group G streptococcal endocarditis reported in the literature is quite low approximately 1 % accordingly to various studies (2,4,16). Blair et al. (3) reported that G group streptococci caused 8.4 % of all beta hemolytic streptococcal endocarditis cases in accord to the published English language literature between year 1973 and 1987. Baddour et al also reported 3 cases as Group G streptococcal endocarditis (1).

The reported overall incidence of central nervous system complications of infective endocarditis varies greatly. In most series, the incidence of central nervous system involvement during the course of infective endocarditis is about 30 % of all cases. The high rate of neurologic complications have been found in cases of infective endocarditis with left sided involvement. The two thirds of the case presenting with neurologic symptoms are due to major cerebral emboli and most commonly affect the middle cerebral artery and its branches. The remaining one third of cases the presenting signs and symptoms are seizures, meningismus, subarachnoid hemorrhage, intracerebral hemorrhage, subdural haemorrhage, personality change, visual disturbances, or weakness of the extremities. The patient who had neurological symptoms like purulent meningitis on admission had diagnosed as cerebral emboli. Group G streptococcal endocarditis is often associated with embolic, neurologic and cardiac complications (8).

Cranial computed tomography or magnetic resonance imaging are the most useful diagnostic procedures in diagnosis of cerebral emboli. In the management of patients with infective endocarditis and cerebral emboli, optimal therapeutical strategy should be considered to prevent new emboli formation. The role of anticoagulant use is not clear. The role of cardiac surgery in the prevention of embolism in infective endocarditis is not widely accepted. The cardiac surgery is considered if vegetation is greater than one centimeter in diameter. In this case vegetatectomy or valve replacement may be tried (8).

In the treatment of infective endocarditis combination of cell wall active agents with an aminoglycoside is recommended at least during the first two weeks of therapy because non-group A β hemolytic streptococcal strains may be less susceptible to penicillin than group A β hemolytic streptococcal strains. According to the data from a retrospective analysis of Group G streptococcal endocarditis cases, combination therapy may diminish mortality rates. The optimal use of antibiotics and their effects remain controversial. There is little of informations in the literature about the in vitro antimicrobial bactericidal susceptibility patterns of Group G streptococci, especially for nonpenicillin agents. The excellent bactericidal activity of penicillin G and III generation cephalosporins against Group G streptococci was shown in recent literature. Although in vitro antibiotic sensitivity against Group G streptococci is

excellent, the delayed clinical response to antibiotic therapy is an interesting feature of group G streptococci (1,7,12, 15,16,17).

Ceftriaxone, a third generation cephalosporin, has an excellent in vitro activity against nonenterococcal streptococci. Ceftriaxone is both effective and safe for the treatment of streptococcal endocarditis due to the some studies performed with nonenterococcal streptococci. Ceftriaxone is also able to penetrate the cerebrospinal fluid that may be a better choice than the first generation cephalosporins as alternative regimens to penicillin when central nervous system complications were seen in infective endocarditis (6,14).

Our patient was admitted to our service with a presumptive diagnosis of central nervous system infection. Ceftriaxone (2x2 g/day, i.v.) was initiated empirically due to the leucocytosis and shift to the left. In the cranial computerized tomography of our patient, minimally contrast media uptake increased was observed in the bilateral parietal and frontal lobes and in the anterior areas of right occipital lobe. The blood cultures results were positive for group G streptococcus. In the echocardiography of the patient, there was a mass on the posterior surface of the mitral valve which was 2x2.5 cm in length. It was thought that the echocardiographic appearance might belong to a vegetation. The patient was diagnosed as infective endocarditis according to the Duke criteria with the presence of two major and two minor criteria (including echocardiographic evidence, two persistently positive blood cultures yielding G Group beta hemolytic streptococcus, vascular phenomena, and fever >38 °C). Gentamicin (2x80 mg/day, i.v.) was added to the treatment at the eighth day of her hospitalization and Ceftriaxone was switched to 2x1 g/day, i.v. from 2x2 g/day, i.v. Ceftriaxone was given for 4 weeks and gentamicin was given for 2 weeks, and progressive improvement of our patient's condition was seen.

In summary, Group G streptococcal endocarditis is often more aggressive, and is more frequently associated with embolic, neurologic and cardiac complications. We reported this case because of its being an unusual form of infective endocarditis that was caused by Group G Streptococcus. We also want to emphasize that the patient who had neurological symptoms like meningitis had to be evaluated in details, in cerebral emboli cases, infective endocarditis should be encountered in differential diagnosis.

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