CASE REPORT

Gemella endocarditis: An aggressive entity

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Abstract The authors present a rare case of subacute endocarditis caused by Gemella morbillorum. A 72-year-old man, with a history of hypertension, aortic valve disease and upper and lower endoscopy six months previously, was admitted due to fever and abdominal pain. He also complained of long-standing dyspnea on exertion and petechiae on his lower limbs. Imaging scans showed a consolidation in the lower left lung field, a splenic infarct and a left subphrenic abscess. Transthoracic echocardiogram findings were highly suggestive of endocarditis affecting three valves, with destruction of the mitral valve anterior leaflet. G. morbillorum was identified in three blood cultures and was considered the etiologic pathogen. Due to the patient’s worsening condition, he underwent cardiac surgery, aiming to control the infection and to resolve the associated mechanical complications. This case highlights the need for a complete and thorough history to arrive at likely diagnostic hypotheses that, together with complementary exams, will lead to correct diagnosis and the prompt institution of appropriate therapy.

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PALAVRAS-CHAVE

Endocardite; Gemella morbillorum; Embolização séptica

Endocardite por Gemella: uma entidade agressiva

Resumo Os autores apresentam um caso de endocardite subaguda causada por um agente raro, Gemella morbillorum. Trata-se de um homem 72 anos com hipertensão arterial e valvulopatia aórtica, submetido a endoscopias digestivas seis meses antes. Admitido por febre e dor abdominal. Concomitantemente apresentava clínica compatível com insuficiência cardíaca descompensada associada a petéquias com semanas de evolução. Os exames imagiológicos mostraram consolidação da base pulmonar esquerda, enfarte esplênico e acesso subfrênico esquerdo. O ecocardiograma transtorácico mostrou alterações sugestivas de endocardite de três válvulas com destruição do folheto anterior da válvula mitral. A embolização esplênica, em...
Case report

A 72-year-old man was admitted due to fever and abdominal pain. His medical history included hypertension and unspecified aortic valve disease. He had undergone upper and lower endoscopic studies six months previously, which revealed only chronic atrophic gastritis. His medication included a proton pump inhibitor, an ACE inhibitor and a thiazide diuretic. He complained of dyspnea with progressively less effort, orthopnea, paroxysmal nocturnal dyspnea and reduced urine output, as well as significant and progressively worsening edema of the lower limbs with dispersed petechiae, which started six weeks before admission. One day before admission fever and abdominal pain had begun.

On admission he was febrile with visible jugular distension; cardiopulmonary auscultation revealed a mitral-aortic murmur, grade IV/VI, and bilateral stasis. He presented diffuse abdominal pain but no signs of peritoneal irritation. He had peripheral edema up to the thighs with dispersed petechiae on the lower limbs.

Blood work showed hypochromic microcytic anemia, with low iron and transferrin saturation but with elevated ferritin and normal lactic dehydrogenase. A peripheral blood smear test showed slight anisocytosis and elliptocytes. His erythrocyte sedimentation rate was 116 mm/h, C-reactive protein was 10.32 mg/dl and B-type natriuretic peptide was >4000 pg/ml. Other blood tests were unremarkable, including platelet count, coagulation studies and renal and hepatic function tests. Thoracic-abdominal computed tomography (CT) scan showed a consolidation in the lower left lung field, a left subphrenic abscess, hepatomegaly and a moderately sized splenic infarction. Transthoracic echocardiography revealed thickening of the aortic valve cusps and two nodular formations causing mild to moderate aortic regurgitation. It also showed a 15-mm vegetation on the anterior leaflet of the mitral valve causing severe destruction of tissue and severe mitral regurgitation, tricuspid valve leaflet thickening with a 13-mm mobile vegetation and severe tricuspid regurgitation. The pulmonary valve cusps were also thickened and mild to moderate pulmonary regurgitation was visualized; global left ventricular hypokinesis was noted. Brain scans were unremarkable. *Gemella morbillorum* was isolated in three blood cultures drawn on admission.

The patient had one major (evidence of infection on echocardiogram) and four minor criteria (predisposition, fever, vascular phenomena and positive blood culture) of the modified Duke classification and a definite diagnosis of endocarditis was made. A course of six weeks of vancomycin and two weeks of gentamicin was started and the patient underwent open-heart surgery with mitral and aortic valve replacement (biologic prostheses) and tricuspid valve annuloplasty. No microorganisms were isolated in native valve cultures. Follow-up imaging scans documented pulmonary consolidation and resolution of the subphrenic abscess, with small splenic infarct sequelae being discernible in abdominal scans. Blood tests showed normal hematocrit and significant reduction in inflammatory parameters. The patient was discharged after concluding antibiotic therapy with complete resolution of heart failure symptoms and no need for diuretic therapy, and under oral anticoagulant and antiplatelet therapy which he maintained for three months. He was followed in the internal medicine and cardiothoracic outpatient clinic.

Discussion

Endocarditis has an annual incidence of 3–10 cases per 100 000, predominantly affecting individuals between 70 and 80 years old, most of whom are male. In recent decades its incidence has risen steadily, due not only to advances in diagnostic techniques but also to nosocomial infections (Figures 1–4).

*Gemella* is a genus of six Gram-positive bacteria species, found in the mucous membranes of various animals, including humans. Endocarditis caused by *G. morbillorum* is a rare condition, with about 24 cases being reported in the relevant medical literature. This anaerobic bacterium, with negative catalase test, is very difficult to identify microbiologically by standard methods and has a highly variable presentation. Part of the normal oropharyngeal, gastrointestinal and urinary tract bacterial flora, it is rarely responsible for infections in humans. However, it can cause septicemia, meningitis, arthritis and endocarditis, which usually follow a subacute or chronic course. More rarely, it may also be responsible for pulmonary infections, including abscess, necrotizing pneumonia and pleural empyema.

*G. morbillorum* endocarditis particularly affects males between 20 and 80 years old, usually with poor dental hygiene or with a history of recent dental procedures, digestive tract examination, immunosuppression, intravenous drug use, diabetes, hepatic and renal impairment or cardiac disease such as valve disease, myxoma, or hypertrophic cardiomyopathy.
Figure 1  The atrial face of the mitral valve anterior leaflet shows a mobile echogenic structure compatible with vegetation about 15 mm in size. The anterior leaflet is thickened, with tissue destruction and likely perforation, causing severe mitral regurgitation.

Figure 2  Aortic valve with two adherent nodular formations, causing poor coaptation and mild to moderate aortic regurgitation.

Figure 3  Tricuspid valve with mobile echogenic structure compatible with vegetation measuring over 13 mm.

Figure 4  Abdominal computed tomography scan showing splenic infarction.

Notwithstanding the institution of prompt and appropriate tailored antibiotic therapy, the patient had an initially unfavorable outcome, and urgent surgery was needed due to acute heart failure caused by the destruction of the anterior leaflet of the mitral valve and regurgitation of the other valves. Surgery also substantially reduced the risk of new septic emboli arising from the presence of >10-mm vegetations.

With this case, the authors wish to highlight the insidious and variable nature of this clinical entity, and the need for a high level of clinical suspicion, based on a thorough and methodical history, as well as appropriate complimentary exams for a correct diagnosis. Prompt institution of antibiotic therapy and, when appropriate, cardiac surgery, confer a favorable prognosis.

Conflicts of interest

The authors have no conflicts of interest to declare.
References