Clinical Case Report

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A case report of Guillain Barré syndrome revealing underlying infective endocarditis due to *Cardiobacterium hominis*

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Abstract

Rationale: Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy presumed to result from an infection-triggered autoimmune reaction.

Patient concerns: This paper describes a 53-year-old man admitted to hospital for deterioration of his general condition.

Diagnosis: He developed GBS, confirmed by lumbar puncture and electromyogram, which recovered after intravenous immunoglobulins. A grade 2 aortic regurgitation was detected by transthoracic echocardiography upon diagnosis of GBS, but in the absence of fever, no further investigations were conducted. A few weeks later, the patient presented with fever and infective endocarditis (IE) was diagnosed after the identification of vegetation on the aortic valve with transesophageal echocardiography. The etiologic agent was identified as *Cardiobacterium hominis* based on 3 positive blood cultures and DNA detection in valvular material.

Interventions: IE was cured with a 6-week course of antibiotics and aortic valve replacement.

Outcomes: The patient completely recovered from Guillain-Baré syndrome and IE.

Lessons: This case of GBS associated with *C hominis* endocarditis, emphasizes the importance of blood cultures and transesophageal echocardiography for the detection of IE and highlights the insidious nature of *C hominis* endocarditis which is often diagnosed late.

Abbreviations: GBS = Guillain-Barré syndrome, IE = infective endocarditis.

Keywords: blood culture, *Cardiobacterium hominis*, Guillain-Barré syndrome, infective endocarditis

1. Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy presumed to result from an infection-triggered autoimmune reaction. Actually, in about more than two-thirds of the cases, an episode of infectious disease is observed in the weeks before GBS onset.[1] Various micro-organisms have been suspected to trigger GBS but a strong association has been established for only a few of them, especially *Campylobacter jejuni* and more recently Chikungunya and Zika viruses.[2] To date, only few cases relating GBS to infective endocarditis (IE) have been reported,[3] none of them due to *Cardiobacterium hominis*.

2. Case

A 53-year-old man was admitted to hospital for deterioration of his general condition. He reported he had had fever before admission but his temperature was normal upon admission. Six months earlier he had undergone dental extraction. He complained of paresthesia in his lower limbs appeared gradually, ataxia, and transient diplopia. Clinical neurological examination showed proprioceptive ataxia without motor deficit, absent Achilles and patellar reflexes, a positive Lasègue straight-leg raise test, and no bladder sphincter disorders. Whole blood cell count was normal and serum C-reactive protein was 60 mg/L. No blood cultures were performed. Cerebrospinal fluid was optically normal, with 4 leukocytes/mm^3, glucose 0.69 g/L, and protein 0.72 g/L. A clinical diagnosis of GBS was made according to the GBS classification proposed by van den Berg et al.[4] The electromyogram was consistent with GBS. Although the patient had no heart murmur, a transthoracic echocardiography was performed, showing a left ventricular ejection fraction of 60%, a grade 2 aortic regurgitation, and no vegetation. The patient was given human polyvalent immunoglobulins intravenously 400 mg/kg/day for 5 days. His neurologic symptoms progressively improved and he was discharged from hospital. Serologic tests for

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The patient provided informed consent for publication of the case.

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C jejuni, Epstein-Barr virus, Cytomegalovirus, and human immunodeficiency virus (HIV) were negative.

Two months later, the patient was readmitted to hospital for acute fever. Because of a diastolic aortic murmur, a transesophageal echocardiography was performed, which showed a severe aortic regurgitation and a 13-mm aortic vegetation on the right coronary cusp of the aortic valve. A total-body CT-scan showed no evidence of septic emboli. Serum CRP and procalcitonin levels were 116 mg/L and 0.32 ng/mL, respectively. The patient was treated empirically with amoxicillin-clavulanate and gentamicin after blood cultures were drawn. Three aerobic blood cultures were reported positive after 65 hours of incubation for a slow-growing Gram-negative rod that was identified as *C hominis*, which confirmed the diagnosis of IE. The patient underwent valve replacement surgery. Valve cultures remained negative whereas 16S ribosomal deoxyribonucleic acid (rDNA) targeting PCR was positive and the corresponding 560-bp sequence showed 100% homology with *C hominis* ATCC 15826^T (GenBank accession no. M35014).

Endocarditis was cured with no relapse after 4 weeks of antibiotics (amoxicillin-clavulanate and gentamicin for 2 weeks and ceftriaxone for 2 more weeks).

3. Discussion

We report on a case of IE due to *C hominis* that was diagnosed a few weeks after the diagnosis and treatment of GBS. The diagnoses of GBS and IE are both unquestionable and we hypothesize that these 2 rare events were related. Although the diagnosis of GBS was made before that of IE, we make the assumption that IE preexisted to GBS but remained undiagnosed at the time of GBS. In line with this hypothesis are the facts that a dental extraction that was performed 6 months before GBS and that aortic regurgitation was diagnosed at the time of GBS are several. The diagnosis of IE was not established at that time because no blood cultures were obtained and echocardiography was transthoracic instead of transesophageal. IE due to *C hominis* often has an indolent course before they are diagnosed.[5,6] In 1 study, the median duration of symptoms before diagnosis of *C hominis* IE was 138 days ± 128.[7]

C hominis is a gram-negative bacillus that belongs to the HACEK group, a group of fastidiously-growing bacteria from of the oropharyngeal microbiota.

GBS is a postinfectious illness that develops after an acute infection, as a result of an aberrant immune response, which causes damage to the peripheral nerve.[1] The most common GBS-causing infection is *C jejuni* enteritis.[8] Although rarely, IE has already been reported as a potential cause of GBS. After literature search, we identified 5 case reports of IE that likely triggered GBS: 2 were due to *Staphylococcus aureus*,[9,10] 1 was due to *Coxiella burnetii*,[11] 1 was due to viridans streptococci,[12] while the last case was culture negative.[13] To our knowledge, this is the first time *C hominis* IE is reported in association with GBS.

Author contributions

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