CASE REPORT

Infective Endocarditis Caused by an Indigenous Bacterium (Gemella morbillorum)

Hirokazu Terada, Kenkichi Miyahara, Hiroshi Sohara, Masahiro Sonoda*, Hitoshi Uenomachi*, Jun-ichi Sanada* and Terukatsu Arima*

A case of infective endocarditis (IE) caused by a rare pathogen, Gemella morbillorum, is presented. Because of persistent low-grade fever after dental treatment, the patient was given oral antibiotics. Whereas he was diagnosed as having aortic regurgitation by a cardiologist, and IE was not suggested unfortunately. After long-term chemotherapy over five months, he was aware of nocturnal dyspnea and Gemella morbillorum was detected by blood culture. Then, he was treated with intravenous administration of Penicillin-G, and underwent surgical operation for valve replacement. No cases of IE due to this organism have been reported in Japan. (Internal Medicine 33: 628-631, 1994)

Key words: superinfection, antibiotics, valvular heart disease

Introduction

Progress in antibiotics remarkably improved the recovery rate of various infectious diseases, whereas the risk of appearance of antibiotic-resistant bacterium has increased. Recently, we treated a case of infective endocarditis (IE) due to Gemella morbillorum, an indigenous bacterium in the upper respiratory and intestinal tract of healthy persons. The present case was regarded to be caused by the opportunistic bacterium due to a long-term treatment with antibiotics. Very few cases of IE caused by Gemella morbillorum have been reported, and the present case was recognized as the 28th in the world, and the first case in Japan, to our knowledge (1, 2).

Case Report

The patient, a male aged 64, visited our hospital with complaints of low-grade fever and nocturnal dyspnea in February 1993. About 6 months earlier, he was treated for dental caries. From that time, he could not be relieved of cold-like symptoms. He was treated with several oral antibiotics, but fever persisted. In December 1992, he was diagnosed as aortic regurgitation due to development of nocturnal dyspnea. But no special suggestion for its treatment was given unfortunately. On admission, his body temperature was 37.3°C. Blood pressure was 112/52 mmHg, and heart rate was regular and 88/minute. Anemic sign was observed in his palpebral conjunctivae, but jaundice, petechiae, and cyanosis were not observed. On auscultation, “to and fro murmur” (Levine 3/6) at the Erb’s area and systolic murmur at the apex were heard, and intermittent rale was audible at both lung bases. Hepatomegaly, splenomegaly, and peripheral edema were not observed.

In the examination, leukocytosis (10,700/μl; basophils 0%, eosinophils 0%, neutrophils 84%, lymphocytes 15%, monocytes 1%) was observed. C-reactive protein (CRP) was 8.5 mg/dl, and the erythrocyte sedimentation rate (ESR) was 47 mm/hr. Urinalysis revealed occult blood (2+) but no protein (Table 1). According to chest X-ray radiography, the cardio-thoracic ratio was 55% and a marked congestion was seen in bilateral lower lung fields (Fig. 1A). Electrocardiography showed sinus rhythm, and poor R progression in V2-3 (Fig. 1B). In an echocardiography, marked left ventricular dilatation, vegetation-like tumors on the anterior mitral leaflet and the aortic valve were observed (Fig. 2A). In addition, a reversed flow through the aortic valve reaching the apical region of the left ventricle, and a mild reverse flow through the mitral valve to the left atrial posterior wall were detected by color Doppler echocardiography (Fig. 2B). Serial blood culture was performed and gram-positive streptococcus was found, but no obligate anerobes were detected. By separating the arterial blood isolate was prepared. By use of a 24-hour culture at 37°C, each isolate was identified by the API 20 strep method based upon biochemical characteristics. No acid was produced from mannitol, sorbitol, inulin, and raffinose. Hippurate and esculin were not hydrolyzed. Ammonia was not produced from arginine. Then, Gemella morbillorum was detected in 3 con-
intravenous drip injection at a daily dose of 18 million units. Diuretics and cardiotonic agents were administered simultaneously. After 20 days of PCG treatment, peripheral white blood cell count (WBC) and CRP were normalized, and no bacteria were detected in blood culture. We concluded that the valvular replacement was applicable. Throughout his clinical course, no evidence of embolism due to IE was observed.

From his medical history, dental caries left untreated was highly suspected as the focus of infection. Therefore, a dental examination was carried out. Necessity of treatment for the infected route and pulpectomy were indicated, and then dental treatment was started. No recurrence of IE was observed thereafter, but on the 35th day of PCG treatment, WBC remarkably decreased to 1,900/μl (neutrophils, 15%) and PCG-induced granulocytopenia was highly suspected. PCG treatment was immediately stopped, and granulocyte stimulating factor was administered, but the patient was complicated with acute appendicitis (Fig. 3). After transfer to the surgery, leukopenia was improved, and appendicitis was subsidized by conservative therapy. Hence, replacement surgery of aortic and mitral valves was performed. No examination by cardiac catheterization was carried out before surgery.

In the surgery, vegetation and rupture of chorda were observed in the anterior mitral leaflet, and vegetation in the right coronary cusp and perforation in the left coronary cusp were found (Fig. 4). The surgical specimens, resected from mitral
Fig. 2. A) Echocardiography. Vegetation-like tumors (arrow) were revealed on the anterior mitral leaflet (left) and the aortic valve (right). B) Color Doppler echocardiography. Aortic regurgitation reaching the apical region and mitral regurgitation to the left apical posterior wall were revealed.

Fig. 3. Clinical course.

Fig. 4. Surgical findings. The perforation (arrow) was revealed in the left coronary cusp.

and aortic valves, were histopathologically examined. Fibrosis with hyalinization and partial mucous degeneration were observed on these valves. In part of the vegetation, proliferation of granulation tissue and calcification were seen, however, no bacterial flora was recognized. The patient recovered completely and was discharged on the 101st hospital-day.

**Discussion**

In 1988, *Streptococcus morbillorum* was reclassified and transferred to the genus *Gemella as Gemella morbillorum* based upon homology of DNA and 16SrRNA oligonucleotide (16SrRNA cataloging) (3). This bacterium is regarded as an indigenous species existing in the upper respiratory tract and intestine, and belongs to gram-positive and facultative anaerobic bacteria. This bacterium is thought to have very weak toxicity and pathogenicity to the human body. Therefore, infectious disease attributed to this bacterium is rare, furthermore IE caused by genus *Gemella* is considered very rare (2, 4–7).

To our knowledge, only 8 cases of IE caused by *Gemella morbillorum* have been published including the present case. Under the genus *Streptococcus*, 4 cases were reported (4, 5, 7, 8). Furthermore after reclassification, 1 case by Yasser and Craig (1) and 2 cases by Ewan (2) were reported. Other than the above cases, a retrospective review revealed 20 cases of IE caused by this organism (9–12). Hence, overall there were only
Gemella morbillorum Endocarditis

28 cases in total, including the present case. Upon review, there was little difference in clinical symptoms between variant streptococcus including Gemella morbillorum and Streptococcus viridans which is the common cause of IE. It is reported that the clinical course of IE caused by Gemella morbillorum develops subacutely or chronically, and it takes 3 weeks to 5 months from the appearance of infectious symptoms to diagnosis (1). Most of these reports stated that penicillin alone or combination therapy of penicillin and aminoglycoside was effective (1, 4, 6). Bisno et al (13) reported that the dosage and period of chemotherapy should be determined based on the susceptibility test of antibiotics according to the established treatment for IE caused by Streptococcus viridans. Regarding the prognosis, almost all cases were curative with a combination of penicillin and other antibiotics, and some cases including the present case required valve replacement.

In general, development of IE is determined by relationship between its bacterial toxicity and host immunity. Although the presence of underlying heart disease is common, IE can develop without any underlying heart disease if host immunity is disordered. Other than patients with heart diseases, the conditions of other compromised hosts such as malnutrition, advanced age, abuse of steroid hormones or immuno-suppressants, and severe consumptive diseases are thought to be a factor for easy development of IE. On the other hand, superinfection due to abuse or continuous use of wide-spectrum antibiotics and opportunistic infection are also important as a cause of the infection. In the present case, no immunodeficiency was observed, and several antibiotics were administered for about 5 months. In this case therefore, IE was considered to be caused by superinfection induced by long-term and incomplete treatment with antibiotics based on the non-treated dental disease and valvular heart disease.

Thus, proper selection and appropriate use of antibiotics are very important for cases with valvular heart disease. Additionally, it is important to be cautious against an oral disease which can be a focus of infection in patients with valvular heart disease. We would like to emphasize here that an opportunistic bacterium such as Gemella morbillorum could cause IE.

References