

An Autopsy Case of *Erysipelothrix Rhusiopathiae* Endocarditis

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Abstract

A 58-year-old man was admitted to our hospital with fever. The vegetation was confirmed by echocardiography on the tricuspid valve and *Erysipelothrix rhusiopathiae* was isolated by blood culture. The patient died due to heart failure, and tricuspid valve vegetation was confirmed on autopsy and the sample of Gram's staining showed Gram-positive microcolonies. Although about 60 cases of *E. rhusiopathiae* endocarditis have been reported, Japanese cases are extremely rare.

Key words: *Erysipelothrix rhusiopathiae*, endocarditis, rapidly progressive glomerulonephritis

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Introduction

Erysipelothrix rhusiopathiae, a facultative anaerobic, Gram-positive bacillus, is an important animal pathogen, but is rarely implicated in humans. In recent years, *E. rhusiopathiae* has been recognized as a cause of serious systemic infections in humans, although most such infections are related to occupational exposure (fishermen, fish handlers, butchers, slaughterhouse workers, veterinarians, etc.). We report here a fisherman who developed infective endocarditis caused by *E. rhusiopathiae* with rapidly progressive glomerulonephritis.

Case Report

A 58-year-old man had the chief complaints of fever, general fatigue, systemic edema and pruritic skin rash on both legs. There was no record of cardiac abnormalities in his past medical history. Physical examination findings on admission (April 2005) were as follows: height, 160 cm; body

weight, 59 kg; body temperature, 38.4°C; blood pressure, 104/70 mmHg; and pulse, 80 beats per minute. Consciousness was alert. On auscultation, respiratory and cardiac sounds were normal, with no abnormal sounds noted. Systemic edema and purpura were observed on both legs. Laboratory findings on admission are shown in Table 1. Hematuria, proteinuria, anemia and rapidly progressing renal failure were subsequently noted, and the patient was diagnosed with rapidly progressive glomerulonephritis (RPGN). Liver dysfunction was caused by alcohol abuse. Chest X-ray showed no infiltrates, but bilateral pleural effusion was present.

On the day after admission, hemodialysis was initiated, and methylprednisolone (1,000 mg/day) was administered for three days, followed by oral prednisolone at 40 mg/day. Echocardiogram revealed a vegetation on the tricuspid valve (Fig. 1) and gram-positive bacillus was isolated by blood culture on day 8. The patient was treated with carbapenems (meropenem for the first 2 weeks, and panipenem/betamipron for a further 2 weeks). *E. rhusiopathiae* was identified by APICoryne[®] (BioMerieux, Craonne, France) on day 35,

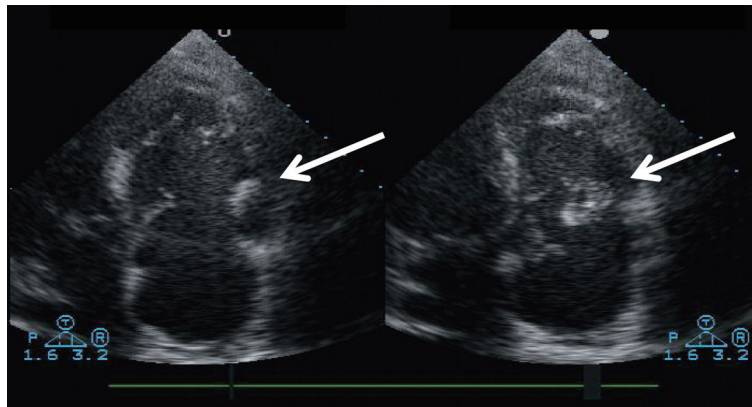
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Table 1. Laboratory Data on Admission

WBC	9800 /mm ³	TP	5.7 g/dl	CRP	9.9 mg/dl	Urinalysis
Seg	78 %	Alb	2.3 g/dl	IgG	1710 mg/dl	U-pro (+)
Ly	14 %	AST	46 IU/l	IgA	268 mg/dl	U-OB (3+)
Mo	8 %	ALT	27 IU/l	IgM	141 mg/dl	
Eo	1 %	γ GTP	84 IU/l	ANF	11.7	RBC many/F
RBC	265 × 10 ⁴ /mm	CK	33 IU/l	Anti ds-DNA Ab	13.7 IU/ml	WBC 8-10/F
Hb	7.9 g/dl	LDH	363 IU/l	Anti ss-DNA Ab	23.3 AU/ml	
Ht	23.2 %	BUN	67.2 mg/dl	CH50	6 U/ml	
Plt	3.6 × 10 ⁴ /mm	Cr	6.9 mg/dl	C3	48 mg/dl	
PT	98.5 %	UA	11.0 mg/dl	C4	3 mg/dl	
FDP	53.9 μ g/ml	Na	140 mEq/l	PR3-ANCA	137.0 EU	
Fib	254 mg/ml	K	3.5 mEq/l	MPO-ANCA	1.3 EU	
		Cl	98 mEq/l	Anti-GBM Ab	3.0 EU	

**Figure 1. Echocardiogram showing vegetation on tricuspid valve (arrow).**

and 16S rRNA analysis was performed at Gifu University. Infective endocarditis caused by *E. rhusiopathiae* was diagnosed. The patient was treated with ampicillin for a further 4 weeks (Fig. 2). The antibiotic sensitivity tests were performed using a broth microdilution test (Table 2). The treatment with antibiotics improved clinical symptoms and markers of inflammation but did not decrease the size of vegetation. While tricuspid valve replacement was considered, the patient refused surgery and was discharged on day 78. He was readmitted on day 140 (August 2005), because of heart failure and exacerbated renal failure was worse. Although antibiotics were re-administered, the patient died due to heart failure. Autopsy confirmed tricuspid valve vegetation (Fig. 3), and Gram-positive microcolonies were seen.

Discussion

E. rhusiopathiae, first isolated from mice by Koch in 1878, was established as a human pathogen in 1909 by Rosenbach, and was first reported to cause endocarditis by Gunther in 1912 (1). *E. rhusiopathiae* is found worldwide as a commensal or pathogenic organism in animals, with the major reservoir being swine. It is also found in fish, surviving in their mucoid slime. The organism may cause a disease in humans after direct contact. Human infection is associated with three clinical types (2): a localized cutaneous

eruption known as erysipeloid; a generalized cutaneous form; and a serious, but rare, systemic complication with septicemia and endocarditis. Most infections are occupation-related, and are seen in, for example, fishermen, fish handlers, butchers, slaughterhouse workers, and veterinarians (3-6). Since the occupation of the patient was a fisherman, septicemia might have developed from percutaneous infection of the minor scratches on his fingers.

A total of about 60 cases of *E. rhusiopathiae* endocarditis (7) have been reported, however only one study was reported in Japan (8). A characteristic feature of *E. rhusiopathiae* endocarditis is its predilection for native left-side valves, particularly the aortic valve (9, 10). About 40% of patients have heart disease as underlying disease, while 33% of patients have a history of alcohol abuse. *E. rhusiopathiae* endocarditis has significant morbidity and a much higher mortality rate (33-38%) than endocarditis due to other organisms (3, 5). This is mainly due to complications such as mycotic aneurysm, valve perforation, myocardial abscess, glomerulonephritis, meningitis or septic shock. Heart failure occurs in 80% of cases.

With regard to the diagnosis of RPGN in the present patient, although PR3-ANCA was positive, typical crescent formation was not seen at the time of autopsy. RPGN was caused not by crescentic glomerulonephritis, but by *E. rhusiopathiae* endocarditis.

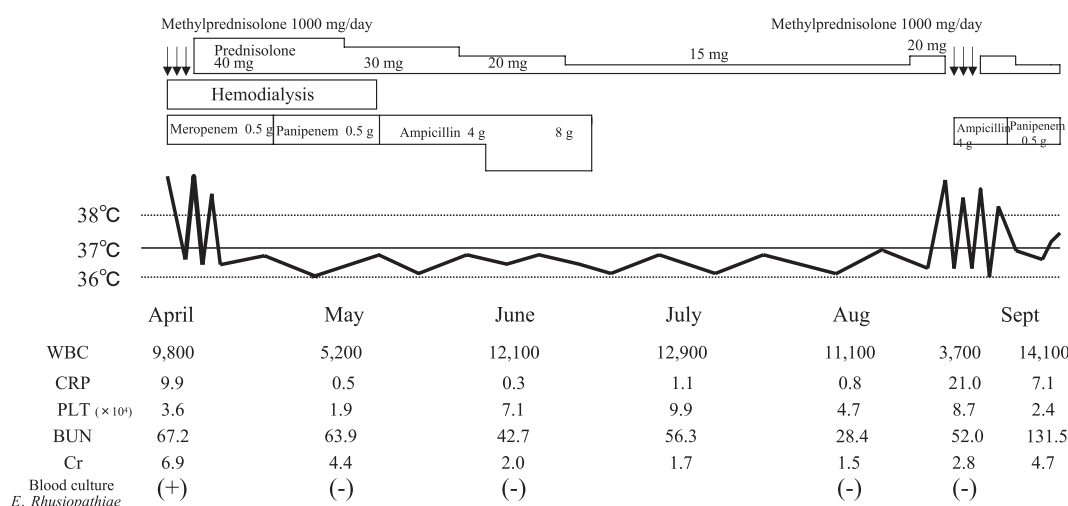


Figure 2. Clinical course.

Table 2. Antibiotic Sensitivity Tests

Antibiotic	MIC (μ g/ml)
Ampicillin	≤ 0.125
Cefazolin	≤ 0.25
Cefotiam	≤ 0.25
Cefpirome	≤ 0.25
Meropenem	≤ 0.25
Panipenem	≤ 0.25
Levofloxacin	≤ 0.25
Erythromycin	≤ 0.25
Clindamycin	≤ 0.25
Minocycline	≤ 0.5
Gentamicin	> 32.0
Arbekacin	> 32.0
Vancomycin	> 32.0
Teicoplanin	2.0

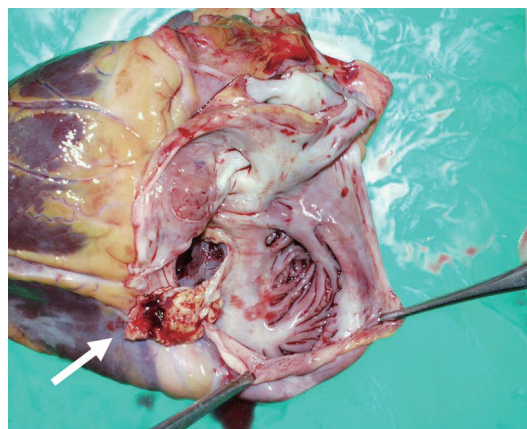


Figure 3. Macroscopic photograph of the heart taken during autopsy. In the anterior cusp of the tricuspid valve, a 2x3-cm vegetation with an irregular surface is visible (arrow).

E. rhusiopathiae is susceptible to penicillin, cephalosporins, carbapenems, quinolones and clindamycin, but is resistant to vancomycin, aminoglycosides, and trimethoprim-sulphamethoxazole (11). In the present patient, *E. rhusiopathiae* was sensitive to β -lactams and new quinolones, but was highly resistant to aminoglycosides and vancomycin. Intravenous antibiotic therapy is recommended for 4-6 weeks, although 2 weeks of intravenous therapy followed by 2-4 weeks of oral therapy has also been successful. Valve re-

placement remains necessary in 36% of affected patients (3). An early cardiac surgery might have been needed for this patient.

E. rhusiopathiae should be considered in the differential diagnosis of infective endocarditis, particularly in those patients with associated characteristic skin lesions or in those with a history of occupational exposure to animals (particularly swine and fish).

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