

***Pasteurella multocida* Infection with Bacteremia Mimicking Acute Coronary Syndrome**

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Abstract

Pasteurella multocida is found in the oral cavity and gastrointestinal tracts of domestic livestock, pets, and wild animals. It is uncommon in humans, but is associated with animal bites and scratches, and can serve as an opportunistic pathogen. Although infection in humans is rare; infected individuals may display symptoms of septicemia, septic arthritis, and pneumonia. We report the case of a cirrhotic patient, who was bitten by a dog and presented with septicemia, disseminated intravascular coagulation, and bleeding. He had symptoms of intermittent chest tightness with elevated cardiac enzymes levels under stable hemodynamically and was suspected to have acute coronary syndrome on hospitalization. Coronary artery angiography showed no abnormality. However, the above symptoms of the patient were relieved after administration of antibiotics. *P. multocida* septicemia mimicking acute coronary syndrome was highly suspected. (J Intern Med Taiwan 2015; 26: 299-302)

Key Words: Acute coronary syndrome, *Pasteurella multocida*, Septicemia, Toxin, Vascular smooth muscle

Introduction

Pasteurella multocida is a zoonotic, gram-negative coccobacillus found in the oral cavity and gastrointestinal tracts of cattle, buffaloes, sheeps, goats, pigs, poultry, and other wild animals.¹ Infection in immunodeficient humans is usually caused by animal bites, and it may present as cellulitis, spontaneous bacterial peritonitis, or septic arthritis. Septicemia caused by *P. multocida* has rarely been

noted in previous medical reports.

Here, we report a case of *P. multocida* infection that mimicked acute coronary syndrome.

Case Report

A 43-year-old man, without any history of systemic disease, was admitted to our hospital. The patient, who was a technician, a compulsive drinker and a heavy smoker. The onset of symptoms occurred 48 h after a dog bite to the right forearm



Figure 1. Healed dog-bite wound over the right forearm.

(Figure 1). The initial symptoms included poor appetite, general malaise and dizziness. Admission at a local medical clinic revealed hypotension and hypothermia. The patient was transferred to our emergency room with a suspected septic shock.

On arrival, hypothermia (35.2°C), hypotension (mean arterial pressure < 90 mmHg), and disturbance of consciousness (Glasgow coma score, E4M6V3) were noted. Physical examination showed mildly icteric sclera, tympany on percussion of the abdomen, and a scar on the right forearm caused by the dog bite. Initially, electrocardiography (EKG) showed normal sinus rhythm without any ischemic change, and blood tests revealed white blood cell count 4000/uL (80% neutrophils), C-reactive protein 9.72 mg/dL, alanine transaminase (ALT) 44U/L, aspartate transaminase (AST) 93U/L, creatine kinase (CK) 1017 U/L, creatine kinase-MB (CK-MB) 60.4 U/L, troponin-I 0.16 ng/mL, total bilirubin 1.83 mg/dL, albumin 2.87 g/dL, and prothrombin time 12.8 sec. However, the patient was also diagnosed with early stage cirrhosis (Child–Pugh score, 5) via abdominal sonography examination and blood sampling.

After admission, the patient displayed chest tightness, cold sweating and respiratory distress with stable hemodynamically. Due to persistent elevation of cardiac enzyme levels (CK, 1972–3172



Figure 2. Normal electrocardiogram.



Figure 3. No critical lesions on coronary arteriography.

U/L; CK-MB, 82.3–102.6 U/L; troponin-I, 0.61–2.1 ng/mL) and non-specific dynamic ST changing on the EKG (Figure 2), acute coronary syndrome was highly suspected. Coronary artery angiography revealed some atherosclerotic change without significant stenotic lesions (Figure 3). Triglyceride levels were 363 mg/dL, cholesterol 105.6 mg/dL, fasting blood sugar 100 mg/dL, brain natriuretic peptide (BNP) 683 pg/mL, d-dimer 23.07 mg/L, fibrin degradation product (FDP) 66.4 ug/mL, and fibrinogen 567 mg/dL. Transthoracic echocardiography showed mild to moderate tricuspid regurgitation with preserved left-ventricular ejection fraction. Infective endocarditis was considered a less likely diagnosis.

The patient was initially administered intensive antibiotics with ceftriaxone, but this was changed to meropenem trihydrate, due to the development of septicemia and disseminated intravascular coagulation two days later. Blood culture revealed *P. multocida*, which is sensitive to all the usual antibiotics. Therefore, amoxicillin/clavulanic acid were administered on the fifth day of hospitalization, and the septicemia was successfully controlled. Both clinical symptoms and septicemia remained under control after 14 days of medical treatment.

Discussion

According to a previous report, patients with immunocompromised status have a higher risk of *P. multocida* infection or bacteremia than other patient groups. Further, patients with cirrhosis, chronic renal failure, or those who receive chemotherapy are more susceptible to this bacterium.^{2,3,4} In data collected over the past 5 years, from our general hospital, this is the only case of *P. multocida* bacteremia identified by biochemical tests.

Several *Pasteurella* species are pathogenic to humans, but most infections are caused by *P. multocida* and involve the skin, soft tissues, and subcutaneous abscesses.⁵ So, local inflammation after animal bite of humans, especially dog or cat, deserves a search for *P. multocida*.¹ Although less frequent, the pathogen can also induce arthritis, osteomyelitis, and pneumonia. In rare cases, it can lead to bacteremia, central nervous system infection, peritonitis, and endocarditis. Previous studies have reported that the infective cardiomyocyte by *P. multocida* would express abnormal signal expression and induce coronary spasm or angina pectoris which caused by cytokine or other mediators modulation.^{6,7} but this phenomenon was not observed during cardiac catheterization in our case.

According to a previous study, rho-like small GTPases play an important role in the actin cytoskeleton, smooth muscle cell contraction, and endo-

thelial permeability.⁸ Cytotoxic necrotizing factor-1 from *Escherichia coli* and *P. multocida* toxin are specific activators of rho-proteins.⁹ Recent studies report that *P. multocida* toxin can activate rho-kinase, which inactivates myosin light chain phosphatase. This results in increase in myosin light chain phosphorylation causing endothelial cell retraction and an increase in endothelial permeability.^{5,10,11} These results indicate that rho-kinase is upregulated and plays a key role in vascular smooth muscle hypercontraction by inhibiting myosin phosphatase through the phosphorylation of its myosin-binding subunit.^{12,13}

P. multocida toxin may, therefore, play a role in acute coronary syndrome modulation, as observed in our patient.

In conclusion, *P. multocida* infection should be considered in immunodeficient patients presenting with septicemia and disseminated intravascular coagulation, particularly if there is evidence of an animal scratch or bite. The pathogenesis and possible complications such as vascular and nervous system damage, which are caused by *P. multocida* infection, are not entirely clear; hence, inadequate immune response or cell damage may be induced in the infected individuals. Our findings reveal that *P. multocida* infection may mimic acute coronary syndrome, therefore, appropriate control of septicemia is recommended.

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多殺性巴氏桿菌感染引發類急性冠心症

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摘 要

一名43歲男性，有肝硬化病史及長期喝酒習慣，此次因右前臂遭狗隻咬傷併全身倦怠至急診求診，診斷為嚴重敗血症入駐內科加護中心，住院過程中因發生胸悶及冒冷汗症狀且併有心肌酶素異常，而緊急安排心導管檢查術，檢查結果並未發現有任何冠狀動脈異常狀況，但住院期間病人偶發有類似冠心症之症狀，直至血液培養結果證實為多殺性巴氏桿菌且接受適當抗生素治療，病患胸悶症狀明顯緩解。據推測此症狀可能為多殺性巴氏桿菌分泌之毒素直接影響心肌細胞收縮，致使病患因遭受該菌感染而產生類似急性冠心症病症。