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Case Report

Increase in serum triglyceride was associated with coronary plaque vulnerability in a patient with rheumatoid arthritis

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ABSTRACT

Rates of morbidity and mortality from cardiovascular disease are high in patients with rheumatoid arthritis (RA); however, the mechanisms and biomarkers that reflect coronary plaque vulnerability have not yet been established. We present a case of acute coronary syndrome (ACS) presumably caused by exacerbation of chronic inflammation of RA, in which an abrupt increase in serum triglyceride was seen on the day of onset of ACS but not during effort angina. This case suggests that RA patients with an abrupt increase in triglyceride need intensive care including anti-platelet and statin therapy for the prevention of coronary plaque rupture.

<**Learning objective:** Triglyceride might be a sensitive biomarker of activated macrophages and plaque vulnerability in patients with RA. RA patients with an abrupt increase in triglyceride might need intensive care including anti-platelet and statin therapy for the prevention of coronary plaque rupture.>

Introduction

Rheumatoid arthritis (RA) is a chronic progressive inflammatory joint disorder. Vasculitis is the most serious complication of RA leading to severe extra-articular organ failure due to microvascular insufficiency, which can result in a fatal outcome [1]. Patients with RA suffer significant morbidity and mortality from cardiovascular disease (CVD) [2,3]. CVD associated with RA includes coronary artery disease, stroke, congestive heart failure, and peripheral arterial disease, which are presumably caused by chronic vasculitis due to endothelial dysfunction; however, the precise mechanism by which an inflammatory joint accelerates endothelial dysfunction has not been clarified [4]. In addition, biomarkers that reflect coronary plaque vulnerability have not yet been established. Here, we present a case of acute coronary syndrome (ACS) presumably caused by exacerbation of chronic inflammation of RA, in which an abrupt increase in serum triglyceride was seen on the day of onset of ACS.

Case report

A 72-year-old man was admitted to our hospital in August 2007 for sudden onset of chest pain at rest and was brought to the emergency room of our hospital by ambulance. He had a history of diabetes and hyperlipidemia for 25 years and a history of RA complicated with rheumatoid vasculitis for 18 years and had been prescribed 15 mg of prednisolone and 50 mg of azathioprine daily. A 12-lead electrocardiogram on admission showed elevation of ST in leads V2–4 and depression of ST in leads II and aVF (Fig. 1A). ACS was therefore suspected. Emergency coronary angiography showed 99% stenosis with thrombosis at the proximal left anterior descending coronary artery (Fig. 1B). Thrombus aspiration and implantation of a bare metal stent (3.0 mm × 30 mm)

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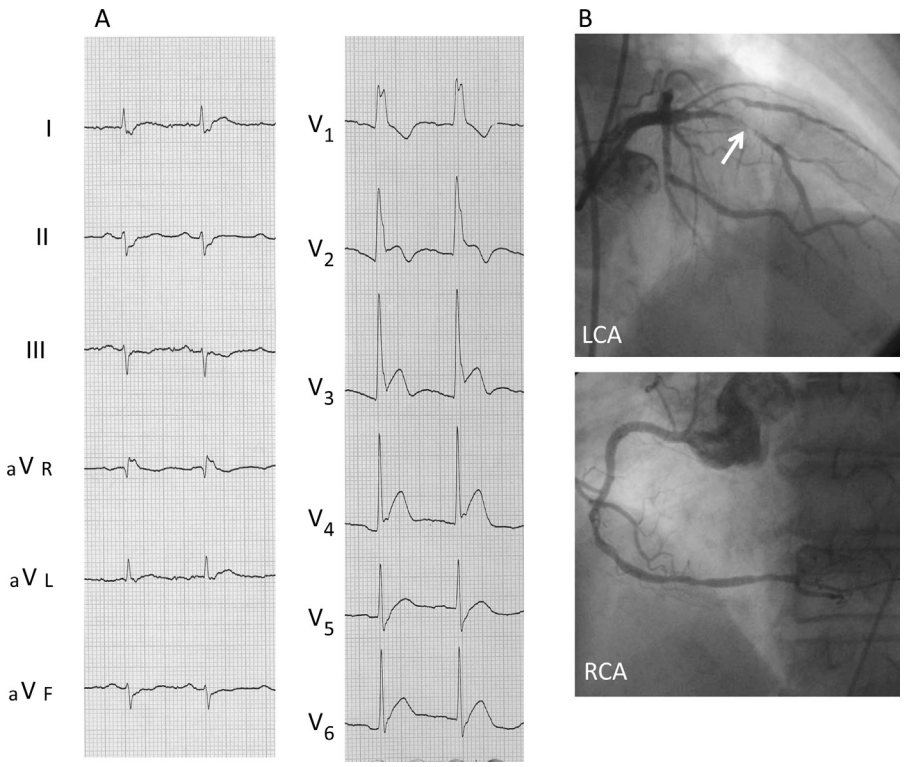

Fig. 1. (A) Twelve-lead electrocardiogram on admission. (B) Emergency coronary angiography showed 99% stenosis with thrombosis at the left anterior descending coronary artery (arrow). LCA, left coronary artery; RCA, right coronary artery.

were performed successfully, and creatine kinase peaked at 2627 U/L. Pathohistology of the thrombus showed accumulation of CD68-positive and CD3-negative inflammatory cells, indicating macrophage-dominant accumulation of inflammatory cells (Fig. 2).

Hyperlipidemia and diabetes had been controlled well with 10 mg of atorvastatin and insulin therapy, respectively. RA had been controlled with an adrenocortical steroid and immunosuppressant. However, the patient had gradually felt exacerbation of joint symptoms several months before the onset of ACS. The results of C-reactive protein (CRP) and rheumatoid arthritis particle agglutination (RAPA) tests showed elevation on the day of onset of ACS. Other inflammatory diseases including bacterial endocarditis, tumor, liver disease (acute and chronic hepatitis), and rheumatic fever were excluded from the medical interview and from laboratory and echographic findings. Of note, well-controlled fasting triglyceride had gradually increased 5 months before the onset of ACS and peaked at 626 mg/dL at the onset of ACS without any over-caloric intake (Fig. 3).

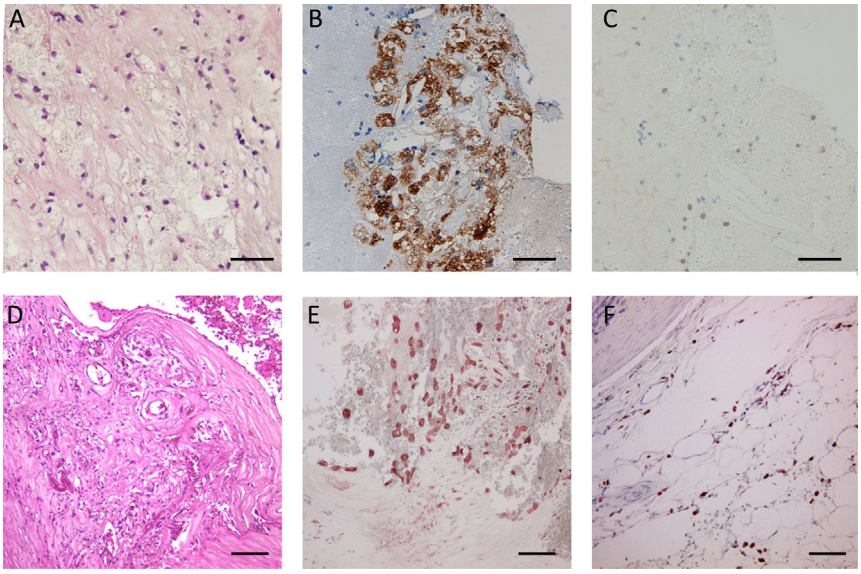

Fig. 2. Aspirated thrombus from the coronary artery shows accumulation of macrophage-dominant inflammatory cells. (A) Hematoxylin and eosin. (B) Immunohistochemistry for CD68. (C) Immunohistochemistry for CD3. The coronary artery from necropsy showed accumulation of CD68-positive inflammatory cells in neointima and pericoronal adipose tissue. (D) Hematoxylin and eosin staining of neointima. (E) CD68 staining of neointima. (F) CD68 staining of pericoronal adipose tissue. Black bar in A–F 100 μm.