Herz 2012 · 37:801–803
DOI 10.1007/s00059-012-3597-x
Received: 8 November 2011
Revised: 28 January 2012
Accepted: 30 January 2012
Published online: 25 February 2012

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Myocardial infarction due to coronary thrombosis in a patient with Henoch–Schönlein purpura

Introduction

Henoch–Schönlein purpura (HSP) is a leukocytoclastic vasculitis of the small vessels characterized by nonthrombocytopenic purpura, polyarthritis, localized subcutaneous edema, glomerulonephritis, and gastrointestinal manifestations [1]. Cardiac involvement is extremely rare [2], although it is considered to be one of the possible features of severe HSP [3]. The association of acute myocardial infarction (MI) and HSP was rarely reported in the literature [2, 3, 4, 5]. Herein, we present a patient with HSP and acute inferior MI.

Case report

A 33-year-old man with HSP, who had been in the remission for 5 years, was

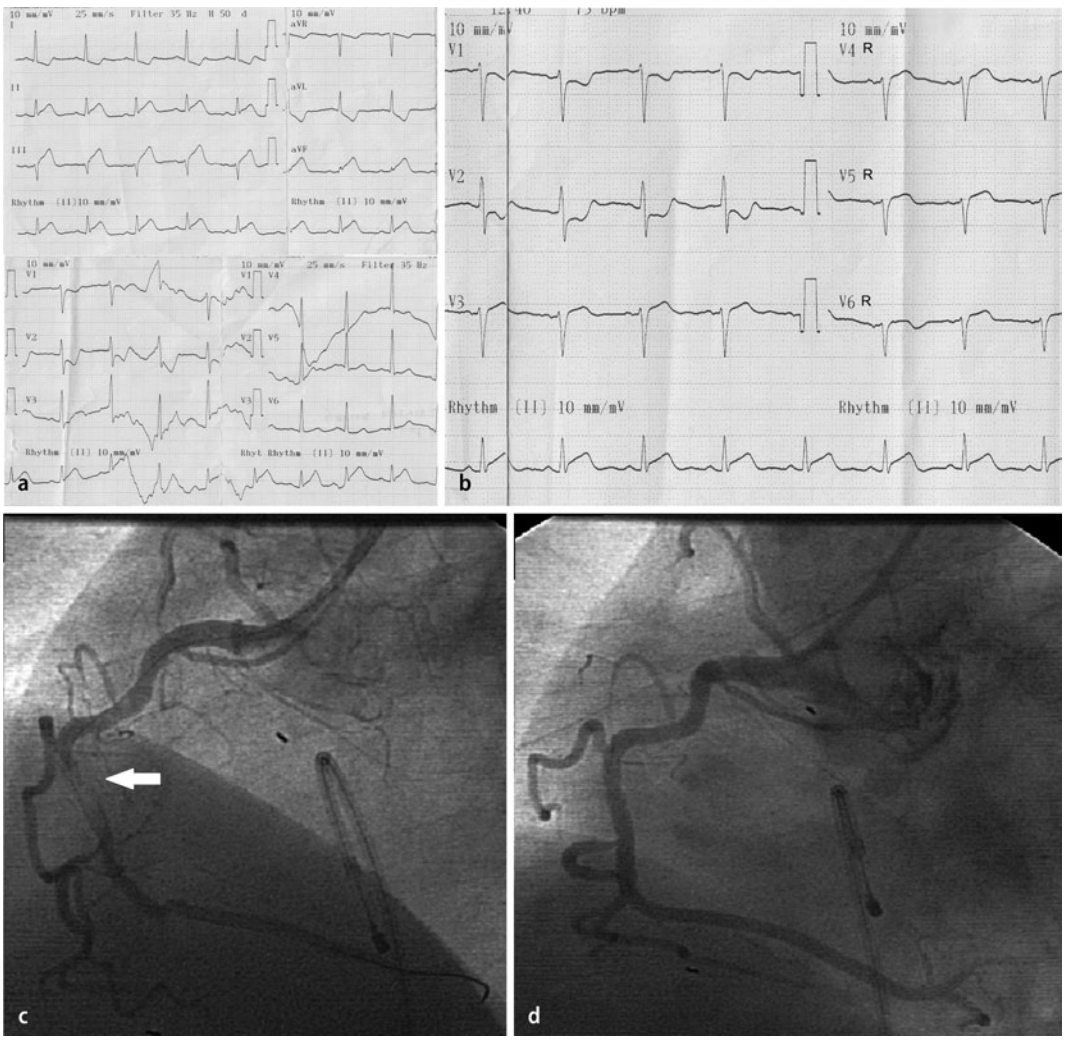

Fig. 1 ◀ **a** Initial electrocardiogram showing ST segment elevation in leads II, III, and aVF; reciprocal ST segment depression in leads I, aVL, and V1-3. **b** Right-sided ECG showing ST segment elevation in leads V_{4R-6R}. **c** Coronary angiogram revealed near total occlusion of the right coronary artery with high thrombus burden. **d** Coronary angioplasty and subsequent stent implantation resulted in TIMI 3 flow with no residual stenosis

admitted to our emergency department with sudden onset chest pain that was stabbing in character and radiating to his left arm. He also had nausea and vomiting before admission. He had been diagnosed with HSP at the age of 8 years and was taking medication, i.e., azothiopurine and prednisolone. He underwent renal transplantation because of glomerulonephritis on the ground of HSP 4 years previously. On admission to the emergency department, his medication included azothiopurine, prednisolone, and tacrolimus. He had no additional cardiovascular risk factor.

Physical examination revealed blood pressure of 110/70 mmHg, pulse 70 bpm, purpuric rash over the lower extremities, and unremarkable findings in other systems. The initial electrocardiogram showed sinus rhythm (75 bpm), 2 mm ST segment elevation in leads II, III, and aVF and reciprocal ST segment depression in leads I, aVL, and V_{1-3} (**Fig. 1a**). In addition, the right-sided ECG demonstrated 1.5 mm ST segment elevation in V_{4R-6R} (**Fig. 1b**). Aspirin (300 mg), clopidogrel (600 mg), unfractioned heparin (4,000 IU bolus, 800 IU/h maintenance dose), and atorvastatin (80 mg) were initiated. The patient developed bradycardia and 3° atrioventricular block with no hemodynamic instability during follow-up. Thus, the patient was diagnosed as inferior ST segment elevation myocardial infarction (STEMI) and transferred to the catheterization laboratory immediately for primary percutaneous coronary intervention (PCI) because of ongoing chest pain (door-to-balloon time 20 min). Coronary angiography revealed a normal left coronary system and near total occlusion at the mid segment of the right coronary artery with high thrombus burden and TIMI 1 flow in the distal segment (**Fig. 1c**). No difficulties were encountered by the guidewire during advancement throughout the right coronary artery (RCA). Thrombus aspiration was unsuccessful to provide TIMI 3 flow. Tirofiban was administered as an intracoronary bolus injection (10 µg/kg over 3 min) before balloon angioplasty. Successful predilatation with a 2.5 × 20 mm balloon (Maverick, Boston Scientific, Natick, MA, USA) and subsequent 2.75 × 28 mm drug-eluting stent (Genius® TAXCOR I™, EuroCor GmbH, Bonn, Germany) implantation was performed with optimal angiographic results (**Fig. 1d**).

After the procedure, the patient was transferred to the coronary care unit for further observation. He was in sinus rhythm 4 h after PCI. Intravenous tirofiban infusion after PCI was maintained at a rate of 0.15 µg/kg/min for 24 h. Further evaluation for thrombophilia (including antiphospholipid antibodies, prothrombin G20210A mutation, protein C, protein S, anti-thrombin III, lupus anticoagulant, activated protein C, factor V Leiden mutation, methylene tetrahydrofolate reductase (MTHFR) C677T mutation, and plasma total homocysteine level) revealed negative test results. In addition, C3 and C4 levels, erythrocyte sedimentation rate, and C-reactive protein levels for disease activity assessment revealed normal results. Thus, no additional immunesuppressive therapy was given to the patient. The patient was discharged from the hospital uneventfully. He was asymptomatic at the 6-month follow-up visit with ongoing dual antithrombotic agents and anti-ischemic treatment.

Discussion

Acute ST elevation myocardial infarction (MI) is uncommon in young individuals. A 10-year incidence rate of MI was reported in 0.52% and 1.29% of young women and men, respectively [6]. In older age groups, atherosclerotic coronary artery disease is the main cause of acute coronary syndrome in young adults, accounting for around 80% of events [7]. Fatty streaks begin at early ages and lead to coronary lesions. It takes several decades for progression to fibrous plaques that may be complicated by erosions or rupture with concomitant thrombosis. This is the main pathophysiologic mechanism of acute coronary syndromes [8, 9]. Myocardial infarction in young patients may have some characteristics that are different from those in older patients. This could indicate a higher prevalence of nonatherosclerotic causes of MI (congenital coronary artery anomalies, myocardial bridging, spontaneous coronary artery dissection, vasculitis, "dilated coronaropathy", coronary lesion secondary to radiotherapy, or coronary embolization), hypercoagulable states, vasospasm, or drug use [10, 11, 12, 13, 14, 15, 16]. Connective tissue disorders, vasculitism and other autoimmune diseases cause myo-

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Abstract
Henoch–Schönlein purpura (HSP) is characterized by vasculitic involvement of small-sized vessels and results in multisystem manifestations. Cardiac involvement is extremely rare and myocardial infarction with coronary thrombus formation in those patients has also rarely been reported. Herein, we report a 33-year-old man with acute myocardial infarction due to coronary thrombus formation and HSP.

Keywords
Purpura, Henoch–Schoenlein · Myocardial infarction · Thrombus · Vasculitis · Glomerulonephritis

Myokardinfarkt durch Koronarthrombose bei einem Patienten mit Purpura Schönlein-Henoch

Zusammenfassung
Die Purpura Schönlein-Henoch (PSH) ist gekennzeichnet durch eine Vaskulitis kleinkalibriger Gefäße und manifestiert sich multisystemisch. Eine kardiale Beteiligung ist extrem selten, auch ein Myokardinfarkt mit Thrombusbildung bei PSH wurde nur selten dokumentiert. Wir berichten von einem Patienten mit durch einen Thrombus und PSH bedingten akuten Myokardinfarkt.

Schlüsselwörter
Purpura Schönlein-Henoch · Myokardinfarkt · Thrombus · Vaskulitis · Glomerulonephritis

cardial damage by several mechanisms, e.g., coronary artery or aortic dissection, coronary artery aneurysm formation, and thrombus formation.

The presented case is important because of the discrepancy between absence of traditional cardiovascular risk factors for coronary artery disease and development of acute MI with coronary thrombus burden in a young patient. This relationship was previously shown in patients with thrombotic thrombocytopenic purpura without any chest pain [17] and also in a young patient with HSP [5]. In accordance with the recently published guidelines, the patient underwent primary PCI and drug-eluting stent implantation due to high restenosis risk of our patient (long lesion, male gender, STEMI, vasculitis) [18]. We suspect that macroangiopathic involvement and coronary thrombus formation in a young patient with HSP in remission for several years may be secondary to systemic vasculitic involvement due to HSP. The rarely observed findings of HSP as coronary thrombus formation and acute MI development make this case report interesting.

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Conflict of Interest. This article was not funded by any institution. The corresponding author states that there are no conflicts of interest.

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