ISSN 2689-8268 | Volume 3

American Journal of Surgery and Clinical Case Reports

Case Report Open Access

Pulmonary Embolism and Global T-Wave Inversion as the Concomitant Presentation of IgA Vasculitis

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Received: 09 May 2021
Accepted: 25 May 2021
Published: 31 May 2021

Citation:

Zhang Q. Pulmonary Embolism and Global T-Wave Inversion as the Concomitant Presentation of IgA Vasculitis. Ame J Surg Clin Case Rep. 2021; 3(5): 1-4

Keywords:

Abdominal Computed Tomography; Symptoms; IgA vasculitis

1. Abstract

A 58-year-old woman was diagnosed as IgA vasculitis by typical symptoms and skin biopsy findings of IgA deposition. The admission ECG showed global T-wave inversion, while coronary CT angiography showed no obvious stenosis or plaque in the coronary artery. During hospitalization, the patient had dyspnea, CT pulmonary angiography showed embolism in the posterior basal segment of the right lower lung lobe. With the application of glucocorticoid and low molecular weight heparin, the patient got resolution of symptom. She has no flare in the subsequent 2 months.

2. Introduction

IgA vasculitis is an immune-mediated vasculitis associated with IgA deposition which predominantly affect the skin, joints, gastrointestinal tract, and kidneys. Thrombotic complications are uncommon. Pillebout et al [1] analyzed 250 cases of adult IgA vasculitis and none of them reported thrombotic complications. Mosalem et al [2] reported a case of adult IgA vasculitis complicated with pulmonary embolism, but did not show specific Electro Cardio Graph (ECG) changes. This case is the first adult IgA vasculitis patient with pulmonary embolism and global T wave inversion. Interestingly, there was no obvious plaque or stenosis in the coronary artery by coronary CT angiography (Figure 1). After treatment, the patient's symptoms disappeared and ECG returned as before.

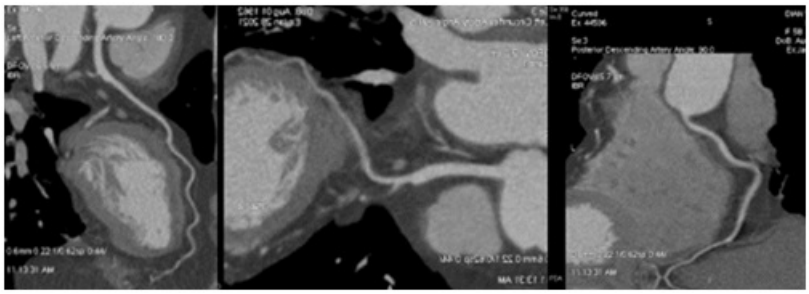

Figure 1: There was no obvious plaque or stenosis in the coronary artery by coronary CT angiography.Left Anterior Descending Artery(left), Left Circumflex Artery(middle), Right Coronary Artery(right).

3. Case Presentation

A 58-year-old woman presented to our rheumatology clinic with a 2-week history of rash and polyarthritis and 1-week history of intermittent abdominal pain on 25 January, 2021. The rash was symmetrically distributed on lower extremities and gradually spread to upper extremities and trunk, accompanied with polyarthritis of hands, wrists, elbows, knees and ankles. Then she developed intermittent abdominal pain. She denied fever, morning stiffness and pruritus. Physical examination revealed non-tender palpable purpura (Figure 2) symmetrically distributed predominantly on

anterior aspects of lower extremities. The upper quadrants of abdomen were tender without rebound tenderness. There was asymmetric edema in the lower extremities. The joints of hands, wrists, elbows, knees, and ankles were diffusely swollen and tender. The remainder of examination was unremarkable. The blood laboratory data were generally normal except for high D-Dimer/FDP (19.41 mg/LFEU/74.12 mg/L) and an increased acute phase reactant (erythrocyte sedimentation rate 42 mm/h, C-reactive protein 111.28 mg/dL). Urinalysis revealed proteinuria with 24-hour urine protein quantification to be 154 mg. The ECG (Figure 3) showed global T-wave inversion and ST-segment elevation in the aVR lead compared to the TP segment. The echocardiogram showed normal ejection fraction and no segmental ventricular wall motion abnormalities (LVEF=65%) and coronary CTA showed no significant coronary plaque or stenosis. Considering occasional dyspnea and high D-Dimmer, we performed Arterial Blood Gas (ABG) which revealed mild hypoxemia (PO2 73.6mmHg). No deep venous thrombosis was found, nevertheless, the CT Angiography of the Pulmonary Arteries (CTPA) suggested filling defects in the posterior basal segment of the right lower lobe of the lung (Figure 4). Abdominal Computed Tomography (CT) showed small bowel wall thickening and edema with suspected multiple mucosal erosions or ulcers. Skin biopsy suggested IgA deposition in the vessel wall (Figure 2). In this patient, the typical palpable purpura, polyarthritis, abdominal symptoms and the findings of IgA deposition, make IgA vasculitis quite conceivable. Eventually, the patient was diagnosed as IgA vasculitis with pulmonary embolism and specific ECG appearance. the patient's symptoms relieved with the application of intravenous methylprednisolone and Low Molecular Weight Heparin (LMWH). ECG returned as before (Figure 5). Until April 2021, the patient stayed stable, still in subsequent follow-up.

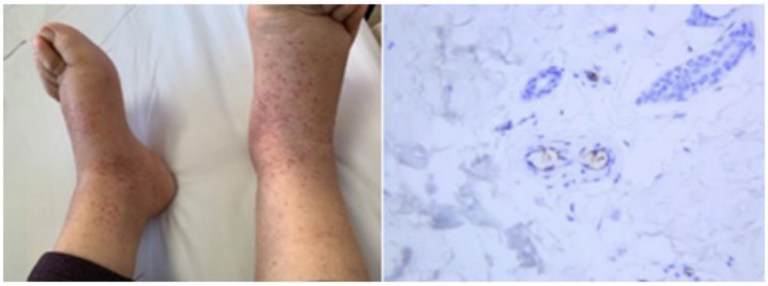

Figure 2: purpura on lower extremities（left）,pathology of the skin showed IgA deposition in the vessel wall(right).

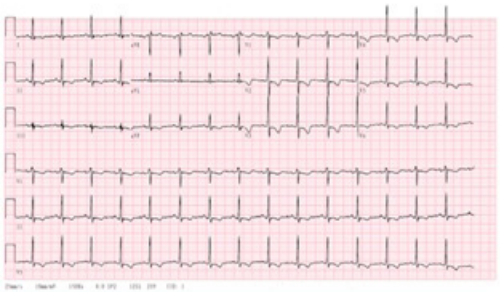

Figure 3: (above) Admission ECG global T-wave inversion with normal QT interval.

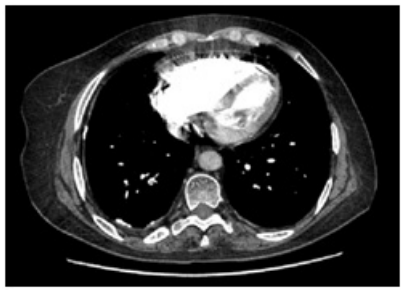

Figure 4: Pulmonary embolism in the posterior basal segment of the lower lobe of the right lung

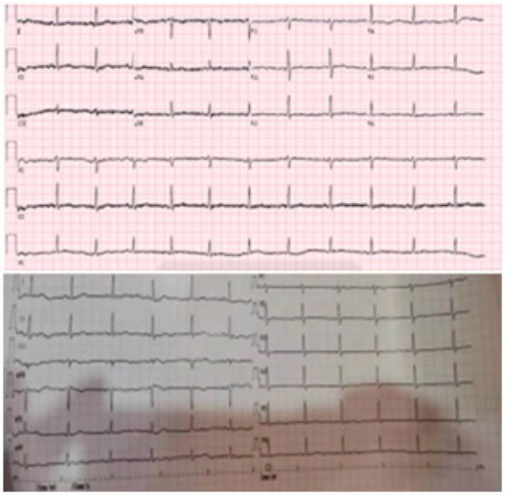

Figure 5: (above) Pre-discharge ECG of the patient Sinus rhythm, no deviation of electrical axis; the bottom Electrocardiogram of the patient on physical examination in September 2020.

4. Discussion

IgA vasculitis is characterized by a tetrad of clinical manifestations that include palpable purpura with neither thrombocytopenia nor coagulopathy, arthritis, abdominal pain and renal disease, which is less common in adults [3]. The etiology and pathogenesis of this disease are still unclear and may be related to infection, food, psychiatric, drug, immune complex deposition, and complement activation [4]. Most studies still favor that the disease is an immune reactive disease involving multiple inflammatory cells, inflammatory mediators, cytokines, adhesion molecules and oxidative stress mediated by IgA [5].

A few case had reported the thrombosis in IgA vasculitis patients. Canpolat [6] reported a 33-year-old IgA vasculitis patient combined with myocardial infarction caused by right coronary occlusion. Sari [7] reported a 37-year-old man with IgA vasculitis combined with dorsal penile vein and femoral vein thrombosis. Mosalem et al [2] had ever reported a case of adult IgA vasculitis complicated with pulmonary embolism, but did not show specific Electro Cardio Graph (ECG) changes. Patients reported previously usually had no risk factors for embolism such as tumor, hyperlipidemia, or prolonged bed rest. The hypercoagulable state of blood in patients with IgA vasculitis may be associated with impaired activation of the activated coagulation system [8]. The patient in this case was admitted with a full-lead T-wave inversion, an ECG manifestation that was first proposed by Walder and Spodick back in 1991 [9], and the exact mechanism regarding this manifestation is still unclear. Its possible etiologies include acute myocardial infarction, stress cardiomyopathy, myocarditis, pericarditis, hypertrophic cardiomyopathy, pulmonary embolism, acute cerebrovascular disease, and pheochromocytoma. In this case, as myocardial enzymology stayed normal, along with the normal echocardiography and coronary CTA examination, we excluded acute myocardial infarction, stress cardiomyopathy, hypertrophic cardiomyopathy, myocarditis, and pericarditis. The patient was clear in consciousness and neurological examination showed no abnormalities. Thus, acute cerebrovascular disease was excluded. The patient's vital signs stayed normal and no adrenal occupying lesion was seen on enhanced CT of the abdomen, which did not support pheochromocytoma tumor. The ECG T-wave inversions can be seen in pulmonary embolism patients, but usually in the inferior wall leads and V1 lead. When combined with prolongation of QT interval [10], we can see global T-wave inversion in pulmonary embolism patients, which may be related to increased ventricular load. In this case, the patient's electrocardiographic QT interval was normal, suggested the global T-wave inversion in this patient may be most likely due to the immune mechanism in IgA vasculitis. With the application of glucocorticoids and LMWH, the patient's ECG gradually returned to the preadmission state.

This case is the first adult IgA vasculitis patient with pulmonary embolism and global T wave inversion which suggest that we need pay attention to the large vessel involvement in patients with IgA vasculitis, and of course, it is worthwhile to think about the anticoagulant drugs use in patients with IgA vasculitis.

5. Conclusion

IgA vasculitis in adults can be combined with vascular complications, which requires our further attention in the future. There may be immune mechanisms involved in the manifestation of global T-wave inversion in the ECG.

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