Journal of Clinical Cases

A Case Of Isolated Splenic Infarction After Paroxysmal Atrial Fibrillation

Yanchao Li,[1,2*] , Jian Zhang[1,2*], Dexin Han[1,2], Gang Wang[1,2], Mengyue Yang[1,2], Yue Zhang,[1,2], Jialu Guo[1,2], Dan Qiang[1,2], Qi Liu[1,2] and Jingbo Hou[1,2]

[1]Department of Cardiology Organization, The Second Affiliated Hospital of Harbin Medical University, Harbin, Heilongjiang 150086, China
[2]The Key Laboratory of Myocardial Ischemia Organization, Chinese Ministry of Education, Harbin, Heilongjiang 150086, China
*Yanchao Li and Jian Zhang contributed equally.

Correspondence to:
Qi Liu, M.D and Jingbo Hou, M.D,
Department of Cardiology Organization, The Second Affiliated Hospital of Harbin Medical University, Harbin, Heilongjiang 150086, China
Department of Cardiology Organization, The Second Affiliated Hospital of Harbin Medical University, Harbin, Heilongjiang 150086, China
E-mail: jingbohou@163.com, doctorliu9128@126.com

Received Date: 06 July 2024
Accepted Date: 17 July 2024
Published Date: 23 July 2024

Citation:
Qi Liu and Jingbo Hou. A Case Of Isolated Splenic Infarction After Paroxysmal Atrial Fibrillation. Journal of Clinical Cases 2024.

1. Abstract

Systemic embolism in Atrial fibrillation (AF) occurs predominantly intracranially, with extracranial embolism being less common. This case report presents a case of paroxysmal AF with an isolated splenic artery embolism. The patient's main manifestation was sudden severe pain in the left hypochondrium region. The main trunk of the splenic artery was diagnosed and finally improved after effective analgesia, anticoagulation, anti-inflammatory therapy, and restoration of sinus rhythm. In patients presenting with acute abdominal pain and paroxysmal AF, it is crucial to consider the possibility of extracranial embolism, such as isolated splenic infarction, as early as possible.

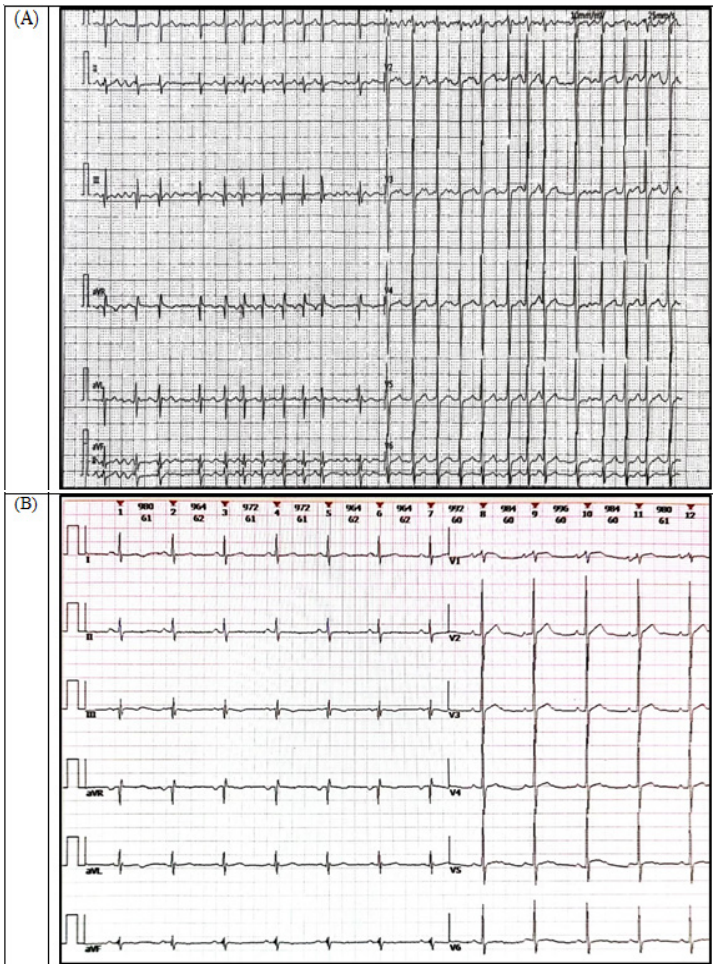
2. Keywords
Atrial fibrillation; Splenic infarction; Artery embolism

3. Introduction

Systemic embolism in AF occurs predominantly intracranially, whereas embolisms in the abdominal organs caused by paroxysmal atrial fibrillation are less common, with isolated splenic infarction being even rarer. Here we present a case of isolated splenic infarction caused by paroxysmal AF. In this case, accurate and timely diagnosis and treatment improved the patient's clinical symptoms and prognosis.

4. Case report

A 54-year-old male patient (height: 176cm, weight: 90kg, BMI: 29.1 kg/m2) was admitted to hospital with "paroxysmal palpitations for more than 10 years, worsening for 2 years". The patient had paroxysmal palpitations more than 10 years ago, which were not treated. In the last 2 years, the palpitations were frequent and he was diagnosed with a psychoneurotic disorder at the local hospital, which improved slightly after symptomatic drug treatment. Twelve days before admission, the patient developed palpitations and was diagnosed with paroxysmal atrial fibrillation at the local hospital because of an ectopic rhythm and rapid atrial fibrillation on the electrocardiogram (Figure 1). The patient had no symptoms other than palpitations during the course of the disease.



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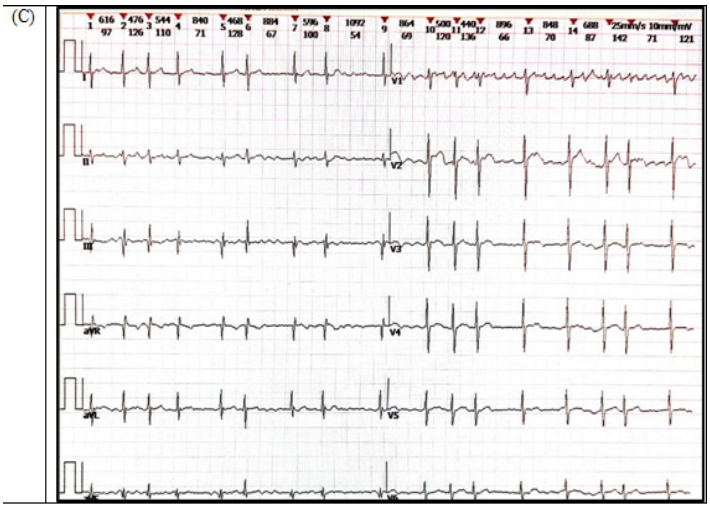

Figure 1: Atrial fibrillation on pre-hospital electrocardiogram (A), Electrocardiogram on admission (B), Electrocardiogram of atrial fibrillation after splenic infarction (C).

He had smoked and drunk alcohol intermittently for several decades. No other medical history was available. Previous coronary angiography at the local hospital showed 40-50% stenosis of the coronary arteries. He was currently taking long-term oral bisoprolol (5mg qd) for ventricular rate control and intermittent oral aspirin enteric-coated tablets. He was admitted to our hospital to complete the relevant investigations and anticoagulation therapy, and to undergo elective radiofrequency ablation of atrial fibrillation. On admission, he was started on rivaroxaban 20mg tablets once daily. On the afternoon of the second day of admission, the patient complained of severe pain in the area of the left hypochondrium, which gradually increased in intensity and was colicky in character, and which was not associated with eating or changes in posture, without pain radiating from the back of the shoulder and the inguinal or perineal areas. There was no significant relief with oral analgesics alone. Vital signs: Body temperature 36. 5 ℃, pulse 99 times/min, respiration 16 times / min, blood pressure 168/110 mmHg. Other regular physical examination, including cardiac examination, was unremarkable. Laboratory tests: Biochemical indices: alanine aminotransferase (ALT) 78 U/L, γ-glutamyl transferase (GGT) 135 U/L. N-terminal b-type natriuretic peptide precursor, NT-pro BNP, was 160 pg./ml. Low-density lipoprotein LDL-C 3.77 mmol/L. Other indicators such as complete blood count, urinalysis, stool analysis, and electrolytes are all within normal range.

Ancillary tests: No atrial fibrillation on routine 12-lead electrocardiogram at the time of the attack or on 24-hour ambulatory electrocardiogram on the first day of admission. Echocardiography: mildly enlarged left atrium, thickened left ventricular wall, mild bicuspid and tricuspid regurgitation, normal left heart function. CT of the whole abdomen showed no urinary stones or pancreatitis. To rule out embolism in the somatic circulation, aortic CTA was performed and showed luminal occlusion of the distal segment of the splenic artery, embolization of the main trunk of the splenic artery near the splenic hilum, and poor splenic perfusion. The patient's abdominal pain resolved after 3 day of active conservative treatment, and a repeat abdominal enhanced CT scan showed that the spleen had multiple scaly hypodense areas, and the infarct area was not significantly enlarged compared with the previous one (Figure 2).

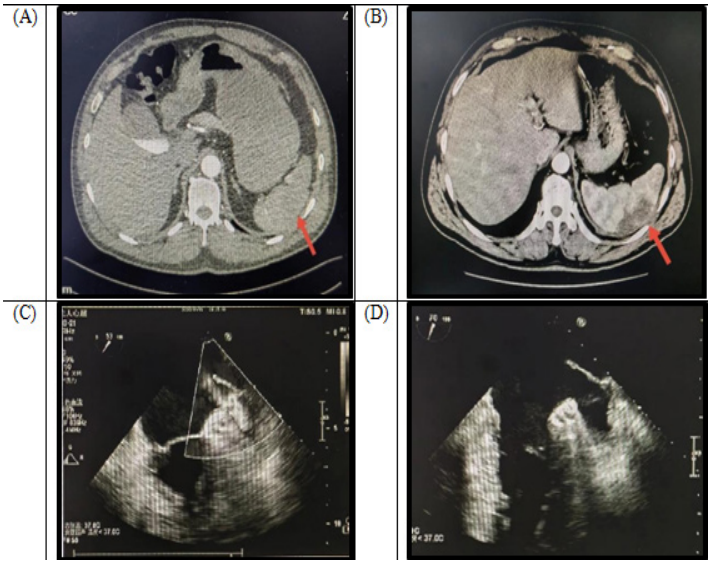

Figure 2: Diagram showing aortic CTA showing splenic infarction on day 1 (A), abdominal enhanced CT showing splenic infarction on day 8 (B) and 3-dimensional transesophageal echocardiography showing no thrombosis in the left atrium, left auricle (C) and (D).

Taking into account all factors, the most probable cause of the sudden onset of pain in the left hypochondrium region during hospitalization was splenic artery embolism caused by thrombus dislodgement as a result of paroxysmal atrial fibrillation. In this case, the patient had hypertension and thrombosis, history of coronary artery disease, so the patient's CHA2DS2-VASc score was 4, HAS-BLED score was 1 due to history of intermittent aspirin use, age <60 years, history of coronary artery disease, and history of smoking, so the SAMe-TT2R2 score was 4. Morphine analgesia was given; In addition to low-molecular-weight heparin 4000IU, subcutaneous injection, 12h/times anticoagulation therapy; prostaglandin 10ug, static, once/day vasodilator therapy; antibiotics were given to prevent infection and splenic abscess formation after splenic tissue necrosis. After 48 hours of abdominal pain, the monitor detected transient atrial fibrillation again, and amiodarone 0.3 g was given once temporarily to restore sinus rhythm. After active conservative management, the patient's pain symptoms improved significantly. After the patient's condition was stabilized, radiofrequency ablation was performed.

5. Discussion

Atrial fibrillation is the most common persistent arrhythmia1. Large-scale epidemiological survey studies show that there are about 12 million patients with atrial fibrillation in China and the prevalence of atrial fibrillation increases with age2.The risk of death in patients with atrial fibrillation is 1.5-1.9 times higher than in patients without atrial fibrillation, and the mechanism is closely related to thromboembolism, followed

by an increased risk of heart failure and the coordinated effects of co-morbidities3. AF is an independent risk factor for stroke4. The incidence of stroke, transient ischemic attack (TIA) and venous embolism in patients with atrial fibrillation who are not receiving anticoagulation therapy is approximately 34.2/1000 person-years5, which is 305 times higher than in people without atrial fibrillation. Although paroxysmal atrial fibrillation was first diagnosed 14 days ago and the electrocardiogram and Holter study before and on the day of the abdominal pain always showed sinus rhythm, the actual history of atrial fibrillation should be up to 2 years, considering that the patient had frequent episodes of palpitations in the past 2 years and did not take oral anticoagulants regularly. Systemic embolism in AF occurs predominantly intracranially, extracranial embolism is less common. Based on an analysis of the databases of four large randomized clinical trials, extracranial embolism accounted for 11.5% of thrombotic events in patients with AF6. Splenic infarction, a relatively rare clinical thromboembolic disease, occurring in approximately 0.004% to 0.01% of hospitalized patients, accounts for only 3% of extracranial embolisms.7. Early splenic infarction is characterized by Osler's triad: left upper abdominal pain, splenic tenderness and mild splenomegaly8. The patient had a sudden onset of left upper abdominal pain after lunch with tenderness in the left hypochondrium, and CT of the whole abdomen suggested only gallstone, cholecystitis and mild dilatation of the gastric lumen, not an abnormality in the splenic region, so the initial diagnosis was very difficult. The patient had persistent abdominal pain that was not relieved by analgesics, and the diagnosis was confirmed by aortic CTA, which showed distal splenic artery lumen occlusion and poor splenic perfusion. Unexplained sudden abdominal pain in the left hypochondrium, not related to feeding or change of posture, and no abnormality on plain CT should be considered in the setting of splenic infarction.

Previous studies have shown that the most common causes of splenic infarction are coagulopathies due to hematological disorders such as sickle cell anemia, lymphoma and chronic granulomatous leukemia, followed by cardiac disorders such as atrial fibrillation, bacterial endocarditis and rheumatoid endocarditis, infectious diseases, cancer embolism, aneurysm and atherosclerosis9. In the present case report, the laboratory tests for hematology, rheumatology, immunology, oncology, tuberculosis and fungus were unremarkable, and the patient had no history of smoking, diabetes mellitus or other risk factors for atherosclerosis, which, together with the patient's current admission for paroxysmal atrial fibrillation, led to consideration of the diagnosis of atrial fibrillation leading to splenic infarction. In fact, the diagnosis of atrial fibrillation after an occult ischemic event is very high7, 10, and in this case the patient had a recurrence of atrial fibrillation the day after the splenic infarction. To date, there are no guidelines on antithrombotic therapy for splenic infarction, and anticoagulation is the necessary and classic therapy for arterial infarction. Splenectomy should be considered for large splenic infarcts, recurrent splenic infarcts, complications of intrasplenic hemorrhage, splenic rupture and hemodynamic instability11. Smaller splenic infarcts should be treated aggressively with conservative therapy and management of the primary disease. In this case, the patient had hypertension and thrombosis, with a history of coronary artery disease, and the benefit of preventing thrombosis was greater than the risk of bleeding. After the patient was admitted to hospital, he was treated with regular anticoagulation with rivaroxaban 20mg, and trans esophageal echocardiography was completed to remove the thrombosis in the external atrium, and he did not have any bleeding manifestations of splenic infarction, so he was maintained on sedation, anticoagulation, vasodilatation, antibiotics to prevent infection. In conclusion, our case illustrates the importance of the presence of splenic infarction as a complication of paroxysmal atrial fibrillation with unexplained abdominal pain in the left quaternary region. This case also suggests that the risk of embolization in paroxysmal atrial fibrillation is comparable to that in persistent atrial fibrillation.

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