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Video 1. Cardiac catheterization demonstrating dilated distal coronary sinus lumen with ostial atresia and retrograde coronary sinus drainage into the innominate vein via thin left superior vena cava.

Address for Correspondence: Dr. Ayhan Çevik,
İstanbul Bilim Üniversitesi Tıp Fakültesi Hastanesi,
Çocuk Kardiyolojisi Bölümü,
Abide-i Hürriyet Cad. İstanbul-Türkiye

Phone: +90 532 657 10 42

E-mail: ayhancevik12@hotmail.com

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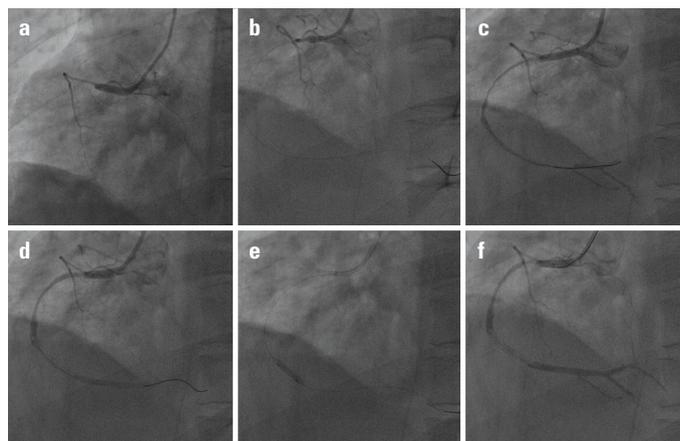


Figure 1. Angiography revealed occlusion of the right coronary artery (a). The blood flow did not improve after the crossing of the guidewire (b). There are still many thrombi and stenoses after aspiration (c, d). After balloon dilatation in the middle of the right coronary artery (e), the blood flow partially improved (f)

Case Report

A 40-year-old male admitted to another hospital with fever for half a month and pain in lower limbs for 1 week. Ultrasound results revealed deep vein thrombosis of the lower extremity, and laboratory examination revealed a high level of troponin I.

The patient was transferred to our hospital. Electrocardiography revealed ST elevation in leads II and III, AVF and V1–3, Q wave was found in leads II and III and AVF. Troponin I level was 4.402 ng/mL. Therefore, a diagnosis of myocardial infarction was confirmed. However, the blood cell count revealed an extremely high white blood cell (WBC) count ($81.9 \times 10^9/L$) and low platelet count ($50 \times 10^9/L$) that indicated leukemia. The patient underwent bone marrow aspiration that confirmed the diagnosis of acute myelogenous leukemia (M4).

The patient then underwent percutaneous coronary angiography, which revealed an occlusion of the proximal right coronary artery (Fig. 1a). After the guidewire crossing, there was still no blood flow in the right coronary artery (Fig. 1b). Intravascular ultrasound was performed, which showed multiple thrombi in the

Myocardial infarction caused by a leukemic clot: A case report

Jing Li, Liang Dong, Chang-ling Li, Ya-bin Liu, Li-jun Feng
Department of Cardiology, The Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou-China

Introduction

Leukemia complicated with myocardial infarction is well known. However, myocardial infarction caused by a leukemic clot has been rarely reported. We present a case of myocardial infarction with right coronary artery occlusion due to the leukemic clot.

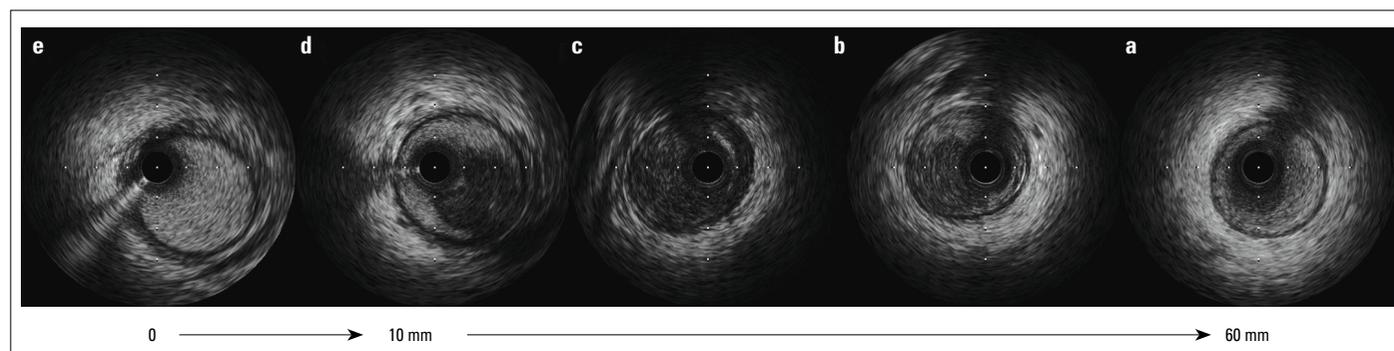


Figure 2. Intravascular ultrasound in the right coronary artery showed complete endarterium without atherosclerosis (a–e) but multiple thrombi (b, c, d) from distal to proximal

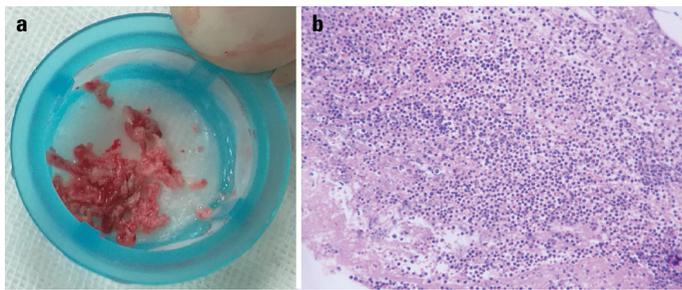


Figure 3. Macroscopic appearance (a) of aspiration material is different from that of normal fibrin platelet thrombus. Histological examination (b) showed abundant blast cells in coagulative necrosis (hematoxylin and eosin stain, $\times 200$)

right coronary artery from distal to proximal. The endarterium was complete without significant atherosclerosis (Fig. 2). After repeated thrombus aspiration, many thrombi were aspirated, but there were still many thrombi that could not be aspirated. There are still serial stenoses in the right coronary artery (Fig. 1c and 1d). To improve blood flow, balloon dilatation was applied in the middle of the right coronary artery, the site of most serious stenoses (Fig. 1e). After balloon dilatation, the blood flow partially improved (Fig. 1f). Because of the unusual appearance of the aspirated thrombi (Fig. 3a) and the history of leukemia, the thrombi were sent for histological examination. The histological examination revealed abundant blast cells in coagulative necrosis (Fig. 3b), according to the examination of the bone marrow. It indicated that the coronary artery was occluded by a leukemic clot.

The patient then received chemotherapy with idarubicin and cytarabine, but he finally died due to *Staphylococcus epidermidis* septicopyemia.

Discussion

There are several other mechanisms of myocardial infarction associated with leukemia, including thrombus caused by activation of the coagulation cascade, infiltration of the artery by leukemic cells, compressing coronary artery by leukemic infiltration of the pericardium, and low impediments induced by hyperleukocytosis (1-4).

The WBC count is associated with the risk of myocardial infarction. The risk of myocardial infarction in persons with WBC count $>9 \times 10^9/L$ is four times greater than that in persons with WBC count of $<6 \times 10^9/L$ (5). Hyperleukocytosis can decrease tissue perfusion because of its deleterious effect on blood rheology. The hemodynamic compromise due to hyperleukocytosis usually affects the microvasculature. There are only a few case reports on occlusion of a major artery, such as the distal aorta, carotid artery, and femoral artery, by a leukemic clot (7, 8).

In our case, the myocardial infarction was caused by multiple leukemic clots in the right coronary artery and was confirmed by histological examination. To the best of our knowledge, there is only one case report on the occlusion of a coronary artery due to a leukemic clot (9). In that case, the patient had acute myeloid leukemia (M4) and an occlusion of the right coronary artery that

was similar to our case. However, the blood flow restored after the transcatheter aspiration was different from that in our case. In our case, there were still many clots that could not be removed after repeated aspiration. The blood flow partially improved after balloon dilatation. An intravascular ultrasound revealed multiple clots and complete endarterium without atherosclerosis. But it was hard to differentiate the leukemic clot from normal fibrin platelet thrombus. It depended on the histological examination. The patient had a concomitant deep vein thrombosis of the lower extremity. Whether this had the same etiology as the myocardial infarction is not clear sure because of the lack of histological evidence.

Conclusion

Occlusion of the coronary artery by a leukemic clot can lead to myocardial infarction. In a patient with myocardial infarction complicated with leukemia, transcatheter inspiration and histological examination of inspiration material are very important for diagnosis and treatment.

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Address for Correspondence: Liang Dong, MD,
Department of Cardiology, The Second Affiliated Hospital,
Zhejiang University School of Medicine,
88 Jiefang Road, Shangcheng District,
Zip code: 310009 Hangzhou - China
Phone: +86 13858188861
Fax: +86 571 87315108
E-mail: dongl1979@hotmail.com

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