A Case Report

Vessel Mimic – A Case Report

Sanjith S Saseedharan¹, Sonal P Karpe², Edwin J Pathrose³, Vaibhav R Kubal⁴

¹M.D. (Anaes), D.A. (Univ), D.A. (C.P.S.), I.D.C.C., F.N.N.C. (Israel), E.D.I.C. (Europe), FIMSA Consultant Critical Care Head-Intensive Care, S.L. Raheja Hospital (A Fortis Associate), Mumbai, Maharashtra, India

²M.D. Pulmonary medicine (Grant Medical College and Sir Jamshedjee Jeejeebhoy Group of Hospitals) Associate Consultant (Critical Care) – S. L. Raheja Hospital (A Fortis Associate), Mumbai, Maharashtra, India

³ M.B.B.S. (Smolensk State Medical Academy, Russia) ICU Registrar, S.L. Raheja Hospital (A Fortis Associate), Mumbai, Maharashtra, India

⁴ DNB Respiratory diseases (National Board of Examination - Delhi) Associate Consultant (Pulmonology) – S. L. Raheja Hospital (A Fortis Associate), Mumbai, Maharashtra, India

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ABSTRACT

The removal of a mediastinal drainage tube placed on day 3 for an uneventful coronary artery bypass graft resulted in a long vessel like tissue being found within the lumen. It was compressible and could be probed through its entire lumen mimicking a blood vessel. Histopathological examination revealed it to be a well organized clot.

Keywords:
drain, thrombus, coagulation

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INTRODUCTION:
In vitro clot formation is a normal phenomenon which we encounter in any clinical setup but when this occurs at sites we least expect and resembles a physiological structure in vivo such as a vessel it warrants a reporting. We hereby report a case of clot formation in one of the mediastinal drains which resembled an artery on gross anatomy.

CASE REPORT:
A 50 year old male underwent coronary artery bypass graft as an elective procedure at our hospital. The procedure was uneventful and patient was shifted to the critical care unit for post-surgery care and monitoring. The patient was transferred on an invasive ventilator with endotracheal tube and three intercostal drains in situ - right pleural, left pleural and one mediastinal drain. All the three intercostals drains were well placed as confirmed by a chest radiograph. The air columns were moving well and there was no air leak. He was on minimal inotropic support and hemodynamically stable. On the day of the surgery, negative suction was applied to all the three drains. On day 2 the drain was around 100 ml from each of the pleural drains and 50 ml from the mediastinal drain. The patient was extubated and chest physiotherapy was started. All the inotropes were discontinued and the suction attached to the drains was stopped. On day 3 the drain further reduced to 50 ml and 75 ml in each of the right and left pleural drains respectively and the mediastinal drain was 60 ml. Decision was taken to remove the mediastinal drain.

This seemingly simple procedure resulted in coming out of what seemed to be a vessel out of the drain at that time. The structure encountered during the drain removal looked exactly like an artery. It was 10 centimeters long, taut, non-collapsing with a patent lumen and openings at the periphery that resembled branches(Fig. 1). To check the luminal patency a probe was introduced which could be navigated along its entire length without any resistance. The hemodynamic stability of the patient meant that it was a vestigial struture not contributing to any of his circulation at the moment. It was deduced that it was probably a graft left behind accidently. The vessel was sent for histopathological examination. The histology revealed it to be a clot. There has been no reported case so far of a clot or thrombus resembling an artery that even an experienced cardiologist, the cardiothoracic surgeon and the intensivist were unable to explain its presence and true nature. Only the histology established the diagnosis. The reporting of such phenomenon should be encouraged as without a proper histology report it can have dire consequences if interpreted wrongly in an otherwise asymptomatic patient. It may lead to unnecessary surgery in the form of re exploration and cause increase in both the duration of icu stay and trauma to patient in the form of surgery.

Fig. 1 Clot from the mediastinal drain mimicking a graft.
**DISCUSSION:** A thrombus is a fibrinous clot formed in a blood vessel or in a chamber of the heart. Thrombi have grossly (and microscopically) apparent laminations called lines of Zahn which represent pale platelet and fibrin layers alternating with darker red cell–rich layers. Such lines are significant in that they are only found in thrombi that form in flowing blood. Their presence can therefore usually distinguish antemortem thrombosis from the bland non laminated clots that form in the postmortem state. Although thrombi formed in the “low-flow” venous system superficially resemble postmortem clots, careful evaluation generally reveals ill-defined laminations.

The clot that we encountered was well defined elongated structure with luminal patency, non-collapsibility and small peripheral openings that resembled branches of a vessel. The absence of lines of Zahn indicated that it had been developed outside a vessel in a cylindrical structure and moulded accordingly. The apparent branches were the places where there were the eyes or openings of the intercostal drains.

Arterial thrombi are typically relatively rich in platelets, as the processes underlying their development (e.g., endothelial injury) lead to platelet activation. An increase in the activity of coagulation factors is involved in the genesis of most venous thrombi, with platelet activation playing a secondary role. Because these thrombi form in the sluggish venous circulation, they tend to contain more enmeshed red cells, leading to red or stasis thrombi. At autopsy, postmortem clots can sometimes be mistaken for venous thrombi. However, the former are gelatinous and due to red cell settling have a dark red dependent portion and a yellow “chicken fat” upper portion; they also are usually not attached to the underlying vessel wall. By contrast, red thrombi typically are firm, focally attached to vessel walls, and contain gray strands of deposited fibrin.

There could be few factors which could explain the presence of formation of the vessel like thrombus. Normal hemostasis comprises a series of regulated processes that maintain blood in a fluid, clot-free state in normal vessels while rapidly forming a localized hemostatic plug at the site of vascular injury.

Disorders of coagulation are disease states which can result in bleeding (hemorrhage or bruising) or obstructive clotting (thrombosis).[1] In all mammals, coagulation involves both a cellular (platelet) and a protein (coagulation factor) component.[2] Virchow’s triad describes the three factors that are thought to contribute to thrombosis.[3]

1. Hemodynamic changes (stasis, turbulence)
2. Endothelial injury/dysfunction
3. Hypercoagulability

Stasis [4] is marked reduction of blood flow or phenomenon of interrupted blood flow. Common conditions include venous stasis, mitral stenosis, prolonged immobility (as on a long plane or car ride, bed bound during hospitalization) and varicose veins. Turbulence contributes to arterial and cardiac thrombosis by causing endothelial injury or dysfunction, as well as by forming countercurrents and local pockets of stasis. Stasis is a major factor in the development of venous thrombi.

**CONCLUSION:** Endothelial injury is an important cause of thrombosis, particularly in the heart and the arteries, where high flow rates might otherwise impede clotting by preventing platelet adhesion or diluting coagulation factors. Examples of thrombosis related to endothelial damage are the formation of thrombi in the cardiac chambers after myocardial infarction, over ulcerated plaques in atherosclerotic arteries, or at sites of traumatic or inflammatory vascular injury (vasculitis). It is predominantly a surface phenomenon and contact with pro coagulant surfaces, such as bacteria, shards of foreign materials, biomaterials of implants or medical devices, membranes of activated platelets, and membranes of monocytes in chronic inflammation. Hyper coagulability means the excessive tendency of blood to clot.[5]

**Conflicts of Interest:** Nil from Authors

**REFERENCES:**


