

Giant Left Atrium with a Large Thrombus

*Muhammad A. Sadiq¹ and Hafidh A. Ba Omar²

أذين أيسر عملاق به خثرة كبيرة

محمد آثار صادق وحافظ عقيل باعمر

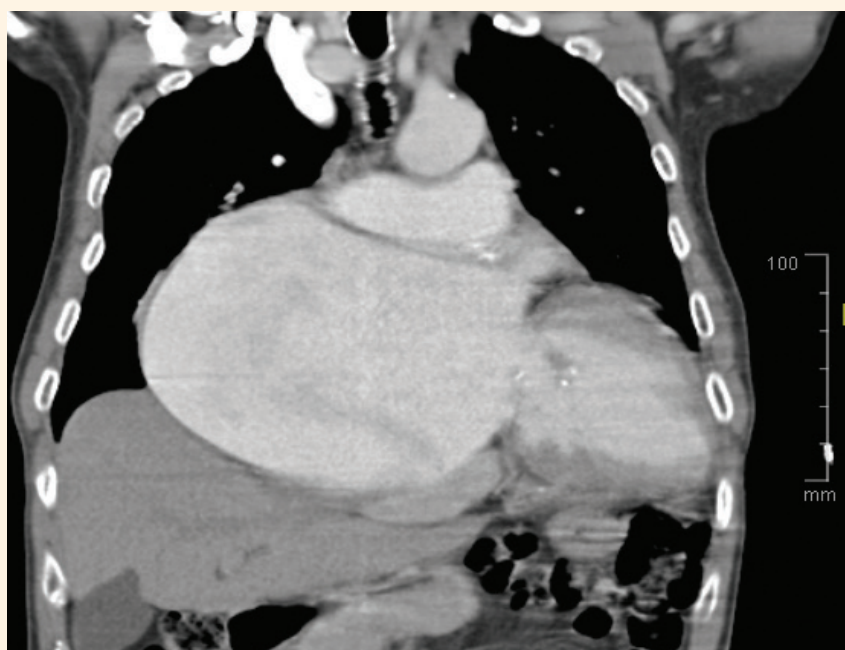


Figure 1: A non-contrast computed tomography two chamber view of the chest of a 73-year-old male patient showing a gigantic left atrium.

A 73-YEAR-OLD MALE PATIENT WITH KNOWN diabetes, hypertension and atrial fibrillation (AF) presented to a tertiary care centre with a transient ischaemic attack. He was referred to cardiology service for evaluation of his AF and to rule out a cardioembolic event. At the time of admission, the patient was on treatment with warfarin with an international normalised (INR) ratio of 2.5.

Transthoracic echocardiography revealed severe mixed mitral valve disease and giant left atrium (GLA) with profuse and dense whirling spontaneous echo contrast in the left atrium. There was a suspicion of a thrombus in the left atrium. Cardiovascular magnetic resonance (CMR) imaging revealed a markedly dilated left atrium measuring 17×11 cm. There was a large filling defect at the left atrium's posterior aspect, which was not visualised adequately in CMR black blood sequence due to severe stagnation of blood. A non-

contrast computed tomography scan was performed, which showed a GLA with a large hypodense filling defect (thrombus) measuring $4.1 \times 3.6 \times 8.2$ cm in its widest dimensions [Figures 1 and 2]. The patient's left ventricular ejection fraction measured by cardiac MRI was 49%. He was offered mitral valve replacement (MVR) and left atrial volume reduction surgery, but he refused surgery.

Comments

GLA is a rare condition with a reported incidence of 0.3%.¹ It is defined as an excessive dilatation of the left atrium by >8 cm.² GLA is usually associated with rheumatic mitral pathology, but there are few reports of GLA in patients with non-rheumatic aetiology.¹ It has been hypothesised that fibrosis with chronic inflammation combined with progressive

¹Department of Medicine, Sultan Qaboos University, Muscat, Oman; ²Department of Radiology, Sultan Qaboos University Hospital, Muscat, Oman
*Corresponding Author's e-mail: matharsadiq@gmail.com

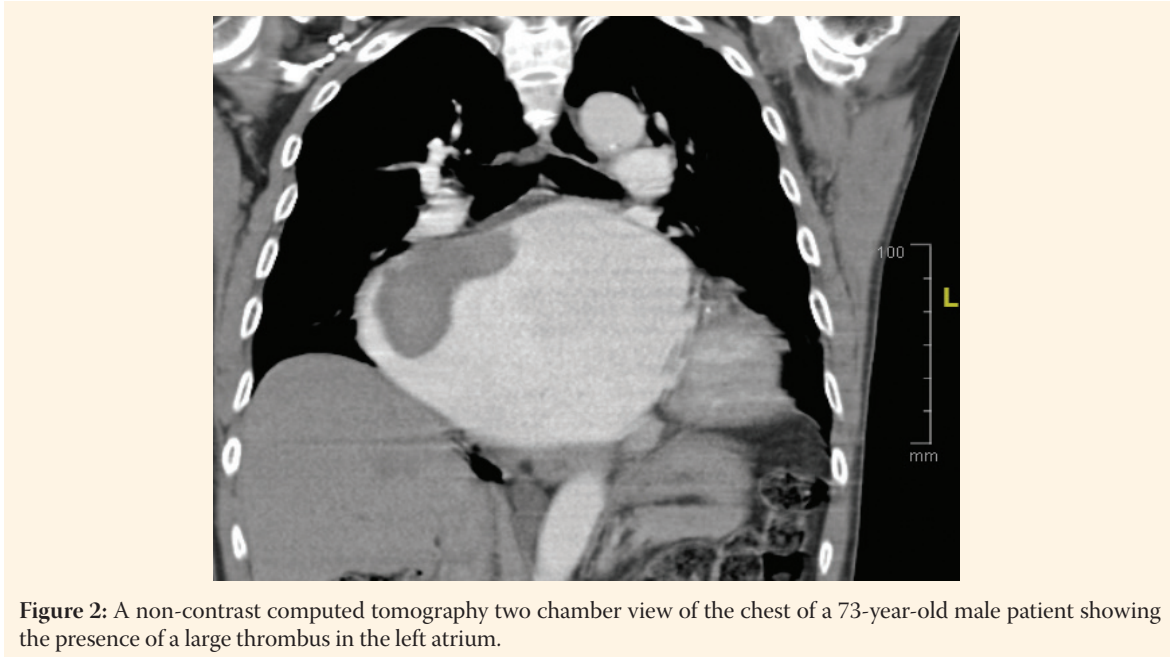


Figure 2: A non-contrast computed tomography two chamber view of the chest of a 73-year-old male patient showing the presence of a large thrombus in the left atrium.

atrial enlargement resulting from chronic volume and pressure overload is likely the cause of gross atrial enlargement.

Patients with GLA usually present with dyspnoea, dysphasia and thromboembolic events. The current patient complained of dyspnoea on minimal exertion, but there was no dysphagia. Although the risk of thromboembolism increases with LA dimensions and is independent of anticoagulants' administration, the published evidence of large thrombus formation in patients with LA size of >8 cm is very scarce. There was a large LA thrombus formation in the current case despite having therapeutic INR with standard vitamin K antagonist therapy. There is a therapeutic dilemma when managing such patients. It is unknown whether the increase in the INR therapeutic range of ≥ 3.0 will resolve GLA patients' clot. Contrary to current American Heart Association (AHA) guidelines that do not recommend using novel oral anticoagulants (NOAC) in patients with intracardiac thrombus and valvular AF, several reports are published on the use of off-label NOACs resulting in resolution of thrombus in patients with enlarged LA.^{3,4} In a case report by Masarova *et al.*, recurrent thrombus formation with therapeutic INR of 2.89 was observed in their patient with GLA after surgical removal of the thrombus. A combination of rivaroxaban and clopidogrel resulted in partial thrombus regression in their patient.⁴ Left atrial

volume reduction surgery in AF patients with enlarged LA have shown superior freedom rates from AF up to five years compared with patients in the non-reduction group.⁵ AF plays a vital role in LA thrombus formation along with an increase in left ventricular (LV) volume overload and enlargement. Freedom from AF with LV volume reduction surgery may play an essential role in managing patients with GLA and large thrombus formation. The current patient was offered MVR, LA volume reduction surgery and thrombus removal, but he refused and was lost to follow-up.

References

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