Authors' reply

The Editor,

I thank the author for the supportive comments and criticisms to our article "anaphylaxis during intravenous administration of amiodarone".[1] A trial fibrillation (AF) is the most common chronic cardiac arrhythmia occurring in 1-2% of the general population.[2,3] AF is an independent risk factor for stroke; in fact, it increases the risk of stroke approximately fivefold.[4] In patients with AF, major mortality and morbidity are secondary to stroke and systemic embolism. The goals of management of acute AF are to relieve patients' symptoms and to prevent thromboembolic complication especially stroke. Clinical evaluation of AF should include determination of the estimation of stroke risk and search for conditions that predispose to AF and for complications of the arrhythmia.[2,4] Many episodes of AF terminate spontaneously within the 1st h or days. Medical cardioversion is indicated in patients with recent-onset AF who remain symptomatic despite adequate rate control. Pharmacological cardioversion may be initiated by a bolus administration of an antiarrhythmic drug. The conversion rate with antiarrhythmic drugs is lower than with direct current cardioversion, but does not require conscious sedation or anesthesia and may facilitate the choice of antiarrhythmic drug therapy to prevent recurrent AF. Several agents are available for pharmacological cardioversion.[4,5] The Task Force the European Society of Cardiology[4] for the Management of AF recommends intravenous flecainide or propafenone for cardioversion of recent-onset AF when pharmacological cardioversion is preferred and there is no structural heart disease (Class I recommendation). In patients with recent-onset AF and structural heart disease, intravenous amiodarone is recommended (Class I recommendation). Digoxin, verapamil, sotalol, metoprolol, other beta-blocking agents and ajmaline are ineffective in converting recent onset AF to sinus rhythm and are not recommended (Class III recommendation).[4] Unfortunately, we did not have intravenous flecainide, propafenone or esmolol. Our patient was symptomatic even after rate control with administration of diltiazem. Therefore, we decided to administer amiodarone. Medical cardioversion was indicated in our patient, as she remained symptomatic even after adequate rate control. Therefore, amiodarone was administered for pharmacological cardioversion. It should be noted that The Task Force of the European Society of Cardiology has not considered metoprolol an effective drug for converting AF to sinus rhythm and not recommended.[4]

Hızır Okuyan, Cihan Altın, Okan Arıhan[1]

Department of Cardiology, Yenimahalle State Hospital, [1]Department of Physiology, Faculty of Medicine, Hacettepe University, Ankara, Turkey

Address for correspondence:
Dr. Hızır Okuyan,
Yenibatı Mah, 2026 Cad, PK: 06370 Batıkent, Yenimahalle, Ankara, Turkey.
E-mail: hizirokuyan@gmail.com

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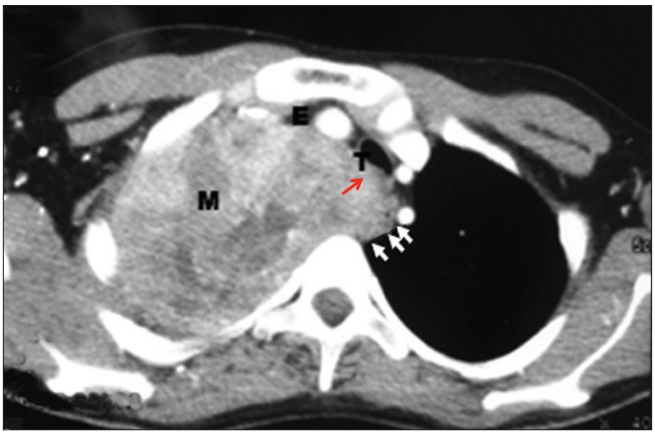

Figure 1: Axial contrast-enhanced computed tomography of the chest shows the heterogeneously enhancing right paravertebral lesion (M) extending anteriorly. The arrow shows the displacement and compression of the vessel and Trachea (T). The esophagus depicted in the original case report is marked as E. The actual position of the esophagus is marked as small arrows (The figure is taken from the indexed case report and published with the permission of Medknow publications, India)

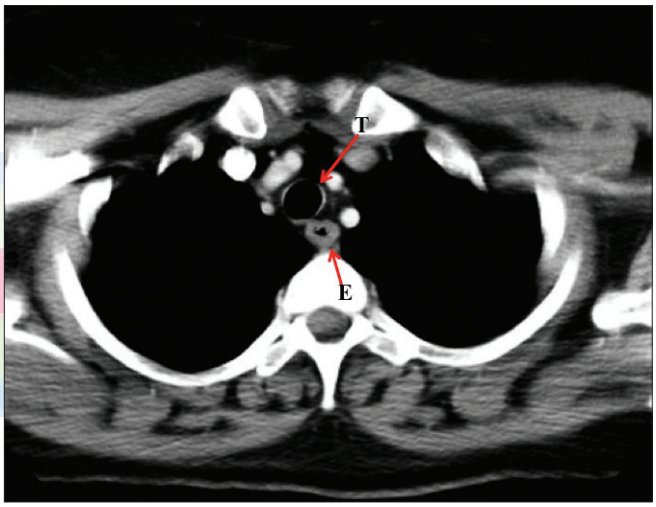

Figure 2: Axial contrast-enhanced computed tomography of the chest in an otherwise normal study. Note the contour and position of the trachea (T) and the esophagus (E)

In response to "Posterior mediastinal mass: Do we need to worry much ?"

The Editor,

We would like to put forth our view in response to "Posterior mediastinal mass: Do we need to worry much?" by Lalwani *et al*.[1] The article presents interesting facts. It introduces to the reader by stating that posterior mediastinal mass carries less anesthetic implications. The authors mention that these masses, with progression, can compress the vital structures including the trachea and the bronchus. The index patient has initially presented with pain, weakness and grade 1 dyspnea with hoarseness of voice. With the mention of the absence of orthopnea, non-productive cough, stridor etc. the authors, it seems, presumed that there is no airway compression. However, the given axial computed tomography image [Figure 1] shows evidence of airway compression. The scan clearly shows that the mass has started from the paravertebral region and extended anteriorly to push the esophagus and the trachea anteriorly. On comparison with a normal cross section image of the chest at this level, the displacement and loss of contour of the airway secondary to the compression by the mass is evident [Figure 2]. Even though, in the patient, there is lack of clinical evidence of the airway compression, the objective imaging evidence of airway compression should have warned