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## References

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## Response

We thank Emma Vardy and Stephen Todd for their interest in our AMC article on a clinical case review of 'Acute presentation of dementia with Lewy bodies' especially on contributing additional knowledge base and evidence in support. We note the comments expressed by Stephen Todd that the case does not meet the criteria for diagnosis. Based on this we revisited the clinical notes and have provided additional information below in support of our conclusion that this was a case of probable DLB using the guide by the Fourth consensus report of the DLB consortium.

Our conclusion was based on the evidence that the patient had more than one cardinal clinical feature of Parkinsonism – bradykinesia and cog wheel rigidity. These features were accentuated by the use of antipsychotics (haloperidol and risperidone) on different occasions; in retrospect these signs predated the use of medications and with heightened awareness and clinical suspicion might have been picked up earlier. The severe sensitivity to antipsychotics is a supportive clinical feature. Our article states that the patient had no visual hallucinations. This is incorrect as visual hallucinations were recorded on collateral history from family on admission and also noted on several instances on the ward. She saw dogs moving around during clinical interviews on the ward. There was also recorded evidence of fluctuating cognition during the course of her hospital admission which was not recorded in our original article. When combined, the presence of more than two core clinical features;  $\geq 1$  cardinal feature of Parkinsonism, recurrent visual hallucinations and fluctuating cognition or one core clinical feature and  $\geq 1$  indicative biomarker ie a positive biomarker (dopamine transporter scan) fits the diagnosis of probable dementia with Lewy bodies in line with the diagnostic criteria of the Fourth Consensus of the DLB Consortium as enumerated in our AMC article. The presence of microvascular changes on computed tomography or magnetic resonance imaging does not negate this conclusion more so as it is not unusual to have these changes coexist with other pathologies in older people.

The case is unique as conventional diagnosis of dementia will follow a course duration of months to years rather than weeks. The lesson of the case review is that clinicians should not be put off by a shorter period of presentation, as in this instance, albeit other causes of delirium including metabolic encephalopathies must

be excluded as was done in this case. In addition, Emma Vardy's written supplement and review on a similar case drives home some of the observations in our original article. ■

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## Oesophago-pericardial fistula

Editor – regarding Dutton *et al.*'s presentation of fatal oesophago-pericardial fistula with cerebral air embolism after elective atrial fibrillation ablation,<sup>1</sup> awareness of this complication is high amongst cardiac electrophysiologists but less so among other physicians. We therefore commend the authors for highlighting this tragic case and we would like to add some complementary insights.

Atrio-oesophageal fistulation should be considered the end stage of a spectrum that encompasses superficial oesophageal thermal injury, ulceration and perforation leading to oesophago-mediastinal, pericardial and atrial fistulation.<sup>2</sup> As the authors point out, a high clinical suspicion is required. This is particularly important for early diagnosis, which will dictate appropriate management. Any presentation between 5 days and 5 weeks following left atrial ablation with a recurrence of atrial fibrillation (AF), chest pain, gastro-oesophageal symptoms, fever and/or leukocytosis should warrant consideration. The development of systemic emboli represents advanced pathology and a very poor prognosis. This pattern may be misinterpreted as endocarditis but history of recent left atrial ablation (particularly AF ablation) should prompt consideration of oesophageal injury and trans-oesophageal echocardiography must be avoided.

The initial investigation of choice is computed tomography (CT) with intravenous and oral contrast. If oesophageal perforation is excluded then endoscopy can be performed to exclude significant oesophageal injury.<sup>3</sup> This is not recommended prior to CT as peri-procedural insufflation of the oesophagus in the presence of an oesophago-pericardial fistula can result in pneumopericardium and haemodynamic collapse.

Management is then guided by the presence or absence of mediastinitis. Although the authors are correct to point out the poor outcomes of medical management and stent placement, this is largely in patients with delayed diagnosis and a systemic inflammatory response suggestive of established mediastinitis.<sup>4</sup> Endoscopic surveillance allows the detection of oesophageal thermal injury prior to perforation and early stent placement in the event of progression. Similarly when fistulation is present without evidence of mediastinitis, stenting may be an effective option.<sup>3</sup> Importantly, where stent implantation is performed in the context of an oesophago-pericardial fistula a pericardial drain should be placed in advance to prevent iatrogenic pneumopericardium. If an atrio-oesophageal fistula or any evidence of mediastinitis are detected then surgical intervention is mandated.

Fortunately, this is a rare complication.<sup>5</sup> A high degree of clinical suspicion is crucial for early diagnosis and we would encourage discussion with cardiac electrophysiology colleagues in all patients presenting to hospital following a catheter ablation procedure

for atrial fibrillation particularly in the context of chest pain, fever and/or leukocytosis. ■

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