Clinical Medicine.[1] In my own practice I have seen a similar case. A diagnosis of dementia with Lewy bodies (DLB) was suspected based on clinical history, collateral history and clinical examination, previous episodes of delirium, lack of response to treatment of identified causes of delirium and the protracted nature of the delirium. The patient was too unwell to undergo a dopamine transporter scan and the patient was trialled on rivastigmine with an excellent response. The patient was discharged home.

The published case highlights various aspects of the management of delirium. It is worth referring to the recently published Scottish Intercollegiate Guidelines Network (SIGN) guideline for risk reduction and the management of delirium.[2] The recommended tool for detection of delirium is the 4AT based on a comparison of different tools, and the National Institute for Health and Care Excellence (NICE) quality standards for delirium recommend assessing all those at risk newly admitted to hospital or long-term care.[2,3] It is worth noting that the investigation of acute and chronic cognitive impairment differ. For delirium the SIGN guidelines recommend good history, collateral history, clinical examination (including neurological) followed by basic and targeted investigations. The recommendation for computed tomography brain relates to various 'red flags' in the acute situation and for further consideration of brain imaging in the case of non-resolving delirium or where there are features to suggest primary nervous system pathology. Similarly, the NICE guidelines for dementia have clear guidance on assessment and investigation strategy in suspected dementia, which includes recommendations around imaging.[4] Some of the investigations listed in the approach to investigation by Akintade and Pierres, for example, autoantibodies would be appropriate only when a cause for delirium has not been found, the presentation is unusual or when not resolving as was the case for the patient presented. It is also worth emphasising that anti-psychotics would not be recommended first line in the management of delirium unless there is intractable distress, risk of harm to the patient or others and when benefits of these medications outweigh potential harms. Non-pharmacological treatment options should always be implemented first, use of more than one pharmacological agent would not be recommended, and it should be noted that only haloperidol is licensed for use in delirium when used without other drugs that prolong QT interval on electrocardiogram.[2]

I would also like to highlight that it has been increasingly recognised in the literature that in some cases of delirium there may be a diagnostic opportunity for DLB.[5,6] It has been suggested that a delirium-like illness may be a prodrome to a diagnosis of DLB.[7] This presentation by Akintade and Pierres is welcome in that DLB as a differential diagnosis for delirium that fails to resolve or is recurrent is highlighted. ■

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Dementia with Lewy bodies

Editor – Akintade and Pierres provide a useful review of the evaluation of cognitive impairment in their Acute Medical Care (AMC) report in July's *Clinical Medicine*.[1] However, the patient described in their report does not have dementia with Lewy bodies (DLB).

Akintade and Pierres cite the updated Fourth DLB Consensus Consortium criteria.[2] Application of these criteria to the clinical information provided leads to the conclusion that the patient is not presenting acutely with DLB.

Firstly, an essential criterion for a diagnosis of DLB is the presence of a dementia syndrome. The acute nature of the presentation at the time this patient was evaluated is not consistent with the typical durations usually considered necessary in standard diagnostic criteria.[2,3]

Secondly, the patient did not present with any of the core clinical criteria for DLB.[2] The Parkinsonism described was not spontaneous but drug-induced.[1] Fluctuating cognition, recurrent visual hallucinations and rapid eye movement sleep behaviour disorder are not present.

Thirdly, the case report suggests alternative explanations that make a diagnosis of DLB less likely, as noted by the DLB Consortium.[2] Akintade and Pierres report that significant ischaemia in the basal ganglia was noted on both computed tomography and magnetic resonance imaging brain studies.[1] Unfortunately, no images are provided for the reader to review. However, the reported degree of ischaemia confounds the brain dopamine transporter study and reduces confidence in a diagnosis of DLB. Additionally, DLB is reported more often to display both bilateral and uniform dopaminergic loss than Parkinson's disease[4] suggesting that the pattern seen on this patient's study is less consistent with DLB.

Finally, supportive biomarkers in this case for a diagnosis of DLB are conflicting. Absence of medial temporal lobe atrophy is reported but electroencephalogram findings are not consistent with DLB.[2] Reduced uptake on metaiodobenzylguanidine myocardial scintigraphy is increasingly recognised as useful in discriminating DLB from other forms of dementia[2] but is not reported in the current patient.

Overall, therefore, the patient described does not meet the threshold for even a 'possible' diagnosis of DLB based on the current standard criteria.[2] ■

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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; ≥1 cardinal feature of Parkinsonism, recurrent visual hallucinations and fluctuating cognition or one core clinical feature and ≥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,[1] 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.[2] 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.[3] 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.[4] 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.[3] 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.[5] 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