Letters to the editor

OVERVIEW

Please submit letters for the editor's consideration within 3 weeks of receipt of *Clinical Medicine*. Letters should be limited to 350 words, and sent by email to: clinicalmedicine@rcplondon.ac.uk

Mitral stenosis-related pulmonary embolism as a potential cause of vocal cord paralysis

DOI: 10.7861/clinmed.Let.21.2.1

Editor – In their lesson of the week article, Raja Shariff *et al* listed a differential diagnosis of Ortner's syndrome which should have included not just compression of the left recurrent laryngeal nerve (LRLN) by left atrial enlargement but also compression of the recurrent laryngeal nerve by a 'large thrombotic formation that completely occlude(s) the outflow tract of the pulmonary artery', as in the case of pulmonary embolism (PE) reported by Polverino *et al*.[1,2] Accordingly, for the sake of completeness, they should have evaluated their patient not only for left atrial enlargement but also for stigmata of PE.

The rationale for evaluation for PE even when left atrial enlargement has been documented by echocardiography is that mitral stenosis is a risk factor for PE (and, hence, for Ortner's syndrome) in its own right, and also a risk factor for mitral stenosis-related mortality.[3–5] The occurrence of mitral stenosis-related PE was exemplified by a 43-year-old man who presented with severe mitral stenosis, atrial fibrillation (AF) and haemoptysis. Contrast enhanced computed tomography demonstrated the presence of a left pulmonary embolism. Left atrial thrombus was also present, as shown by cardiac magnetic resonance imaging and by transthoracic echocardiography. Deep vein thrombosis was excluded by ultrasonography.[3] In the clinicopathological study of 51 cases of mitral stenosis published by Jordan *et al*, pulmonary emboli or infarcts were present at necropsy in 27 cases. In that study, 16 of the instances of PE and/or pulmonary infarct were associated with the presence of mural thrombi in the right atrium. Furthermore, peripheral venous thrombi were found in eight cases.[4] Pulmonary embolism was listed as a cause of death in six of the 59 patients with mitral stenosis-related mortality reported by Donzelot *et al*.[5]

PE-related Ortner's syndrome has not been reported in the context of mitral stenosis. Nevertheless, in view of the above observations and in order to maximise the impact of 'lesson of the week' as a 'teachable moment', evaluation for PE should be included in the evaluation of mitral stenosis patients with vocal cord paralysis even when left atrial enlargement is present.[2–5] The purpose of that exercise would be to rule out (or rule in) the possibility that LRLN compression might be attributable to mitral stenosis-related PE, given the fact that the latter can have a fatal outcome. At the very least, evaluation for PE should include an evaluation for peripheral venous thrombi (as a risk factor for PE) and a transoesophageal evaluation specifically to look for stigmata of PE. ■

OSCAR JOLOBE
Retired geriatrician, Manchester, UK.

References

1 Raja Shariff REF, Abidin HA, Kasim S. Lesson of the month: A forgotten classic: Delayed diagnosis of mitral stenosis presenting initially as Ortner's syndrome. *Clin Med* 2020;20:e267–8.
2 Polverino F, Ricciardi M. Voice box symptoms: A hitherto unknown presentation of pulmonary embolism. *Am J Respir Crit Care Med* 2013;187:108–10.
3 Zhao Q, Ye X, Liu J *et al*. Pulmonary thromboembolism in a patient with rheumatic mitral valve stenosis: A fortuitous association? *Ann Thorac Surg* 2011;92:2263–5.
4 Jordan RA, Scheifley CH, Edwards JE. Mural thrombosis and arterial embolism in mitral stenosis. A clinicopathologic study of fifty-one cases. *Circulation* 1951:3:363–7.
5 Donzelot E, de Balsac RH, Samuel P, Beyda E. Life expectation of patients with mitral stenosis with and without operation. *British Heart Journal* 1957;19:555–8.

JAK-inhibition as a therapeutic strategy for refractory primary systemic vasculitides

DOI: 10.7861/clinmed.Let.21.2.2

Editor – I read with interest the vasculitis update by Mooikhin Hng and colleagues who mention tociluzumab (anti-IL-6) as a therapeutic option in refractory giant cell arteritis (GCA), and wish to add that tocilizumab has been used successfully in refractory polyarteritis nodosa (PAN).[1,2] More importantly, patients with GCA who do not respond to biologics have few treatment options other than high doses of systemic corticosteroids.

These patients provide crucial learning experiences and the pressing need to understand pathogenesis of vasculitides in more detail. Inflammatory cytokines from effector T-cell subtypes allows self-sustained signalling in vasculitis and Janus-associated kinase inhibitors (JAKinibs; small molecules that inhibit JAK1, JAK2, JAK3 and Tyk2) have proven quite useful in controlling tissue inflammation in some refractory systemic vasculitis (supplementary material S1).[5–9] Immunophenotyping data in large vessel vasculitides show distinct characteristics between GCA and Takayasu's arteritis (TAK), but also have similarities such as Th1, Th-17 and Tfh cells involved in both disease relapses and such knowledge may help with personalised therapies.[3] The IL6/JAK/STAT3 axis in systemic sclerosis was one of the initial models where the efficacy of tofacitinib as a potential anti-fibrotic agent was recognised, and was then shown to reverse graft-versus-host