systemic lupus erythematosus who are negative for antiphospholipid antibodies makes this unlikely (Hojnik et al, 1996).

The valvular abnormalities in antiphospholipid syndrome are usually clinically silent and progressive valvular dysfunction is rare (Hojnik et al, 1996). Occasionally, however, significant valvular dysfunction occurs and there are reports of cardiac failure requiring valve replacement surgery (Bouma et al, 2010). Embolic phenomena are uncommon but, like this case, can be a cause of cerebral infarction as well as other systemic complications (Hojnik et al, 1996).

It may be relevant that this patient was positive for lupus anticoagulant which is a stronger risk factor for thrombosis than anticardiolipin antibodies in patients with antiphospholipid syndrome (Galli et al, 2003). Her presentation with deep vein thrombosis and cardioembolic cerebral infarction emphasizes the need for effective anticoagulation to prevent further thrombotic events. Continuing treatment with rivaroxaban was chosen because of uncertainty about the availability and safety of warfarin therapy in her homeland. Rivaroxaban is a factor Xa inhibitor and studies of its effectiveness in antiphospholipid syndrome are currently in progress. Early signs are good and an antiphospholipid syndrome study showed greater suppression of complement levels compared with warfarin leading to speculation that, in addition to its anticoagulant effect, rivaroxaban may benefit patients by limiting the activation of complement (Arachchillage et al, 2016). Hydroxychloroquine was added for protection against systemic lupus erythematosus and its proposed antithrombotic effects (Belizna, 2015). **BJHM**

LEARNING POINTS

- An aetiological diagnosis should always be sought in patients who present with deep vein thrombosis.
- Libman–Sacks endocarditis is not always a benign disorder and may be a source of cerebral thromboembolism.
- Associations of Libman–Sacks endocarditis with systemic lupus erythematosus are variable and may be absent.

Arachchillage DRJ, Mackie IJ, Efthymiou M et al. Rivaroxaban limits complement activation compared with warfarin in antiphospholipid syndrome patients with venous thromboembolism. J Thromb Haemost. 2016 Nov;14(11):2177–2186. https://doi.org/10.1111/jth.13475

Belizna C. Hydroxychloroquine as an anti-thrombotic in antiphospholipid syndrome. Autoimmun Rev. 2015 Apr;14(4):358–362. https://doi.org/10.1016/j.autrev.2014.12.006

Bouma W, Klinkenberg TJ, van der Horst ICC et al. Mitral valve surgery for mitral regurgitation caused by Libman-Sacks endocarditis: a report of four cases and a systematic review of the literature. J Cardiothorac Surg. 2010 Dec;5(1):13. https://doi.org/10.1186/1749-8090-5-13

Galli M, Luciani D, Bertolini G, Barbui T. Lupus anticoagulants are stronger risk factors for thrombosis than anticardiolipin antibodies in the antiphospholipid syndrome: a systematic review of the literature. Blood. 2003 Mar 1;101(5):1827–1832. https://doi.org/10.1182/blood-2002-02-0441

Hojnik M, George J, Ziporen L, Shoenfeld Y. Heart valve involvement (Libman-Sacks endocarditis) in the antiphospholipid syndrome. Circulation. 1996 Apr 15;93(8):1579–1587. https://doi.org/10.1161/01.CIR.93.8.1579

Levine JS, Branch DW, Rauch J. The antiphospholipid syndrome. N Engl J Med. 2002 Mar 07;346(10):752–763. https://doi.org/10.1056/NEJMra002974

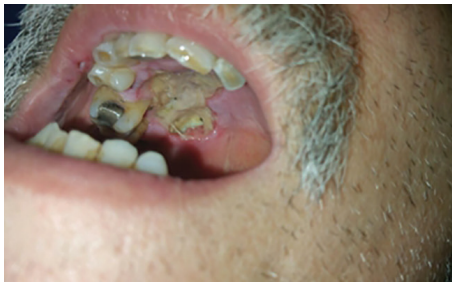
Pengo V, Tripodi A, Reber G, Rand JH, Ortel TL, Galli M, De Groot PG; Subcommittee on Lupus Anticoagulant/Antiphospholipid Antibody of the Scientific and Standardisation Committee of the International Society on Thrombosis and Haemostasis. Update of the guidelines for lupus anticoagulant detection. J Thromb Haemost. 2009 Oct;7(10):1737–1740. https://doi.org/10.1111/j.1538-7836.2009.03555.x

Ren X. Foster E, Ryan EW. 2014. Libman-Sacks endocarditis. The Heart.Org Medscape Cardiology. https://emedicine.medscape.com/article/155230-overview#a5

A man with black–brown nasal discharge

A 56-year-old man presented to the emergency department with a history of nasal fullness, brown nasal discharge and facial pain, and a past medical history of poorly controlled diabetes mellitus. His physical examination was significant for low grade fever, black and brown discharges and necrotic tissue on both walls of the nasal cavities, and necrotic scars on the hard palate (*Figure 1*). A computed tomography scan of the head and paranasal sinuses revealed mucosal thickening in the right ethmoid, maxillary and frontal sinuses, with soft tissue swelling around both orbits which supported the diagnosis of mucormycosis.

Figure 1. Necrotic scars on the hard palate.



Mucormycosis refers to several life-threatening diseases caused by fungal infection (mostly *Rhizopus* species) (Petrikkos et al, 2012). The patient was commenced on antimicrobial therapy (amphotericin B) and underwent immediate debridement of infected and necrotic tissue. Unfortunately he died 1 month later. **BJHM**

Dr Hadi Mirfazaelian, Assistant Professor, Department of Emergency Medicine, Tehran University of Medical Sciences, Tehran, Iran

Dr Atousa Akhgar, Assistant Professor, Department of Emergency Medicine, Tehran University of Medical Sciences, Tehran, Iran

Correspondence to: Dr A Akhgar (At.akhgar@gmail.com)

Petrikkos G, Skiada A, Lortholary O, Roilides E, Walsh TJ, Kontoyiannis DP. Epidemiology and clinical manifestations of mucormycosis. Clin Infect Dis. 2012 Feb;54 Suppl 1:S23-34. doi: 10.1093/cid/cir866