As the sole blood supply to the lungs in this case was through the LPA, its stenosis assumed a very critical role and the RV failure made it imperative to relieve the stenosis. The pulmonary arteries stretch before the intima and media tear during balloon dilatation, and under these conditions a stent proves advantageous since it prevents elastic recoil and leads to permanent vessel enlargement[1]. Also, lesser vessel wall trauma may reduce the chances of restenosis. It seems safe to balloon dilate and stent a branch pulmonary artery even if the other branch is absent; however, safety is limited in cases with complications.

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Reference

Mitrail, aortic and tricuspid valvu- lar heart disease associated with ergotamine therapy for migraine

Ergotamine has been associated with numerous vascular complications but only rarely with valvular heart disease. We present a patient whose ergotamine abuse resulted in claudication of the legs followed by gangrene as well as valvular heart disease; the latter led to triple-valve replacement.

A 45-year-old woman presented with non-progressive weakness, orthopnoea, and ankle oedema. Her pertinent medical history included the use, for more than 5 years, of Cafergot suppositories (ergotamine tartrate 1 mg, caffeine 100 mg), up to 10 per day for migraine headaches. She also had a childhood history suggesting scarlet or rheumatic fever.

At the time of writing, 18 months ago the patient underwent mitral valve replacement for fourth degree mitral regurgitation. Two months later a de Vega tricuspid annuloplasty was necessary due to tricuspid regurgitation, and 6 months ago the patient underwent aortic and tricuspid re-
placement due to tricuspid stenosis and second to third degree aortic regurgitation. Furthermore, a permanent pacemaker (VVI) was implanted as a result of symptomatic bradycardia absoluta. After tricuspid replacement and re-location of the epicaldial lead, the first lead was left on site.

On admission, we found typical signs of congestive heart failure: tachycardia, dyspnoea, weakness, ankle oedema and radiological signs of oedema of the lung. Echocardiography demonstrated left atrial (LAD 42 mm) and right ventricular dilatation (end-diastolic diameter 38 mm), but normal left ventricular size (LVEDD 48 mm) and function. The diastolic pulmonary artery pressure measured 45 mmHg, but there was no sign of haemodynamically relevant valvular or prosthetic dysfunction.

Due to claudication of the leg and progressing gangrene, angiography of the lower extremity was performed. It demonstrated severe diffuse narrowing of the iliac and femoral arteries without any localized stenosis. The right foot was amputated. The specific medical history revealed abuse of ergotamine suppositories used in the treatment of migraine for more than 5 years.

We re-examined the pathological and histopathological findings acquired by other institutions. The excised mitral valve displayed severe diffuse leaflet and chordal thickening with commissural fusion. No appreciable calcification was evident. Proliferation of fibroblasts and smooth muscle cells was identified by standard light microscopy; features included aberrant nuclear shape and cytoplasmonic eosinophilia. These cells formed a thick coating that surrounded the normal leaflet and chordal element. There were no signs of acute inflammation.

All three aortic valve cusps demonstrated mild thickening, without commissural fusion or calcification. The tricuspid valve showed commissural fusion, but no calcification. The histopathological findings were similar to, but less severe than, those of the mitral valve.

Although it cannot be proven conclusively that ergotamine caused the valvular lesions described in this patient, it is the most likely aetiologic agent. The patient had no history suggestive of rheumatic fever, scarlet fever, congenital heart disease, or infective endocarditis. The patient developed the signs of claudication and of congestive heart failure concomitantly while using Cafergot suppositories (ergotamine tartrate 1 mg, caffeine 100 mg). Angiography showed vasoconstriction, but no localized stenosis. Vasculitis was not detected in any specimen.

The pathogenesis of ergotamine-associated proliferative valvular process is still unclear[2-3]. The cardiac valvular lesions resemble those described for carcinoid heart disease. Among the patients with carcinoid heart disease, levels of circulating serotonin are elevated markedly, and serotonin and other vasoactive amines seem to be instrumental in the formation of carcinoid plaques. It has been speculated that the serotonin antagonist, tioni, may also have agonistic properties in the cardiovascular system.

The valvular dysfunction, e.g. tricuspid and aortic regurgitation, most likely resulted from reduced mobility of the thickened valves. In addition, a reduced valve area resulting from scarring is possible.

In summary, although valvular heart disease has only rarely been associated with chronic ergotamine toxicity[4], chronic ergotamine abuse must be considered as an aetiologic agent[5], especially in the case of mitral regurgitation.

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