A 38-year-old woman with Down syndrome and mitral valve prolapse presented with 2 weeks of fever and cough. She had presented to an outpatient clinic 1 week earlier and was prescribed empirical antibiotics for a suspected respiratory tract infection. Despite this therapy, she continued to have high spiking fevers and developed a fulminant rash, prompting emergent admission for further evaluation. She had previously been healthy without other risk factors for infective endocarditis.

On admission to the intensive care unit, she was febrile, confused, tachycardic, and hypotensive. Examination showed peripheral signs of septic embolization, including Janeway lesions (Figure, A), splinter hemorrhages (Figure, B), Osler nodes (Figure, C), and conjunctival petechiae (Figure, D). An IV/VI plateau–shaped holosystolic murmur was heard in the mitral area with radiation to the axilla. Laboratory studies revealed neutrophilic leukocytosis and lactic acidosis. Transthoracic echocardiography showed myxomatous morphology of the mitral valve with holosystolic prolapse of the anterior mitral leaflet (Figure, E and Movie I in the online-only Data Supplement). Color-flow Doppler showed severe eccentric mitral regurgitation with a posteriorly directed regurgitant jet (Figure, F and Movie II in the online-only Data Supplement). Jet velocities in excess of 4 m/s were measured on continuous-wave Doppler interrogation (Figure, G).

Although there were no obvious vegetations on the mitral valve, a filamentous 4-cm-long oscillating mass was seen originating from free wall of the left atrium at the site of impact of the high-velocity jet (Figure, H and Movie III in the online-only Data Supplement). Transesophageal echocardiography used to further study the intracardiac mass (Figure, I and Movie IV in the online-only Data Supplement) showed a small vegetation at the base of the anterior mitral leaflet (Figure, J and Movie V in the online-only Data Supplement). Noncontrast computed tomography of the chest and abdomen showed multiple pleura-based pulmonary nodules suspicious for septic emboli (Figure, K). Magnetic resonance imaging of the brain using T1-, T2-, and diffuse-weighted sequences, these lesions displayed subtle diffusion restriction on diffuse-weighted imaging, consistent with acute embolic infarcts (Figure, L).

A diagnosis of left atrial mural endocarditis secondary to mitral valve jet lesion was made. The pathogenesis of the underlying mitral valve prolapse was thought to be myxomatous degeneration, with the abnormal substrate predisposing to concomitant native valve endocarditis. Emergent therapy was initiated with endotracheal intubation, intravenous vasopressors, and broad-spectrum antibiotics, including vancomycin and gentamicin. Serial blood cultures grew methicillin-sensitive Staphylococcus aureus, establishing the diagnosis of definite endocarditis by fulfilling 2 major modified Duke Criteria.1 The patient’s clinical condition rapidly improved over the next 2 days, with weaning of mechanical ventilation and circulatory support. Extensive discussion with the patient’s family about the necessity of surgical intervention followed, but the family remained adamant about wanting medical therapy only. The patient was eventually discharged home 2 weeks later on long-term intravenous antibiotics.

Mural endocarditis secondary to jet lesions is very rare and can easily be missed during echocardiography if the index of suspicion is low. From a pathophysiological standpoint, high-velocity intracardiac jets can lead to endothelial injury and mural endocarditis by multiple mechanisms.2 In the case of aortic coarctation, patent ductus arteriosus, arteriovenous fistulas, and ventricular septal defects, the vena contracta at the transition zone between a high-pressure chamber and a low-pressure sink can produce Venturi effect–related flow disturbances and indirect endothelial trauma. In the case of regurgitant aortic or mitral valvular lesions, the direct impact of the high-velocity jet against a cardiac chamber or valve tissue can cause endothelial denudation. Regardless of the mechanism of formation, the sites of endothelial injury are highly thrombophilic. Deposition of platelets and fibrin on these lesions produces MacCallum plaques, which can serve as a nidus for endocarditis during transient bacteremia. The echocardiographic recognition of these lesions is of critical clinical importance for 2 reasons. First, the diagnosis of infective endocarditis may depend entirely on their presence,
especially when valvular vegetations are absent. Second, complete removal of these lesions during surgery is necessary to clear the bacteremia, to decrease embolic complications, and to prevent relapse after the completion of antibiotic therapy.

Disclosures

None.

References

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