Eosinophilia and Valvular Heart Disease

Sir,

The endocardial disease associated with idiopathic eosinophilia was first appreciated by Loffler\(^{[1]}\) in 1936. Since then, numerous reports and small series of patients have firmly established the association of these findings.\(^{[2,3]}\) Indeed, cardiovascular disease has been recognized as the major cause of morbidity and mortality in patients with idiopathic eosinophilia.\(^{[4]}\) Idiopathic hypereosinophilic syndrome is a rare systemic disease that can cause multiple organ failure by eosinophil infiltration. Cardiac involvement is characterized by endocardial fibrosis and overlying thrombus, leading to restrictive cardiomyopathy and valvular dysfunction. Surgical experience of patients with mitral dysfunction caused by this syndrome is limited, and valvular replacement is most often performed.\(^{[5]}\)

Rheumatic heart disease is the commonest cause for valvular heart disease and hence most patients are empirically labeled with this diagnosis. However, routine preoperative evaluation revealed hyper eosinophilia in three patients forcing a re-consideration of their etiology.

Three patients (two males and one female) in age group (18-35 years) were evaluated for exertional dyspnoea. Echocardiography revealed MS, MR with AS. The AR in two patients and MR in one patient. The patient with only MR also had a neurological deficit. The CT Brain revealed a parietal infarct. Hyper eosinophilia was seen in all patients ranging from 2 to 22 x 10\(^8\) \(^1\). Bone marrow revealed eosinophilia with normal cytogenetics. Ultrasound revealed mild splenomegaly in one patient. All three had elevated IgE levels. Connective tissue and malignancy workup was negative in all three. Two patients responded to steroids whereas the third required hydroxyurea and vincristine to control the eosinophilia. Two patients underwent double valve replacement and one had MVR. The valves used for replacement were Starr-Edwards and St. Jude valves, respectively. Surgery was uneventful. The HPE revealed myxoid changes with focal aggregates of lymphocytes in two patients. One patient who had MVR with Starr-Edwards valve was readmitted 8 months later with a left ventricular clot with an intact valve. The other two patients are alive and well on follow up.

Harley et al. in an analysis of a large population of patients with the idiopathic hypereosinophilic syndrome defines two patterns of clinical illness with respect to the development of heart disease. In one pattern, heart disease was accompanied by characteristics of a primary myeloproliferative type disease.\(^{[6]}\) In the other pattern, patients remained free of heart disease, the characteristics were more of allergic or hypersensitivity process. They tended to have elevated Ig, especially IgE, and elevated level
of circulating immune complexes.[4]

In our report on three patients, one had splenomegaly, and all three had elevated IgE levels, with two patients showing a dramatic response to steroids. Abnormal eosinophil morphology or eosinophilic infiltration of valves was not demonstrable in any patient. There was no evidence of rheumatic activity on Histopathological examination of the valves or in the blood tests. In our country where Rheumatic heart disease is the most common cause for valvular heart disease other causes such as eosinophilic myocarditis needs to be actively looked for before labeling a patient. A larger number of patients would further emphasize this point.

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REFERENCES


OLANZAPINE-INDUCED DOUBLE INCONTINENCE

Sir,

Urinary and faecal incontinence induced by olanzapine.

Olanzapine, a newer antipsychotic, is among the commonly prescribed antipsychotics around the world. The side effect profile of these newer antipsychotics is better than that of the typical antipsychotic medication. There is only one case report of fecal incontinence[5] and one report of urinary incontinence with olanzapine.[6] We would like to report a patient having urinary and fecal incontinence on olanzapine.

A 35-year-old man was admitted for the treatment of an episode of mania. His serum lithium level was 2.94 mEq/L for which he started on sodium valproate and olanzapine was stopped later. The patient is asymptomatic at the time of reporting this case.

The adverse drug reaction probability score[3] for the patient was nine denoting a definite adverse reaction due to olanzapine. Incontinence, reported with clozapine[6] and risperidone, is uncommon with olanzapine. Two cases of incontinence have been reported in patients taking olanzapine.[1,2] One report suggests the response of urinary incontinence with ephedrine.[2] An overall numerical increase in the incidence of urinary incontinence with olanzapine as compared to placebo has been reported among patients with Alzheimer’s disease.[5] The pathophysiology of this phenomenon is likely to be due to the action on central micturition pathways than the peripheral action. A study on anesthetized rats showed the inhibitory effects of olanzapine on the external urethral sphincter.[6] Although the antimuscarinic side effect would predict urinary retention, incontinence is due to its central mechanism.[6] The physician should be aware of this rare side effect of olanzapine.

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References