Infected Saphenous Vein Coronary Artery Bypass Graft

To the Editor:

The article by Douglas, Bulkley and Hutchins (Chest 75: 76, 1979) calls attention to a fatal complication resulting from disruption of an aorto-vein graft anastomosis due to infection. We have previously reported in Chest a patient in whom disruption of this anastomosis was recognized four months following the initial aorto-coronary bypass procedure.1

In our patient, evidence existed that the disruption had been present for some time prior to re-exploration. Bloody discharge was noted from the wound and dysplasia and mediastinal widening were present. At exploration total separation of the vein from the aorta was found.

At the time of re-exploration in our patient, indolent infection was thought likely to be the underlying cause for the disruption. Although not stated in our report, in view of the possibility of infection, postoperative closed irrigation was employed as described by us2 and cited in Douglas, Bulkley and Hutchins' article. Results of all cultures were negative, however, and healing occurred without evidence of infection. Despite occlusion of the second vein graft in our patient, no further revascularization was done because of the possibility of infection.

Our patient recovered uneventfully from reoperation. He was last seen three and one-half years after reoperation. Although revascularization was not accomplished, the patient has done reasonably well with only occasional anginal discomfort.

Graft disruption indeed appears to be a potential complication in patients undergoing saphenous vein aorto-coronary bypass grafts. It may not, however, be possible to incriminate active infection as the cause in every instance. In addition, the problem may present itself at some time after the immediate postoperative period.

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REFERENCES


Eosinophilia Caused by Rifampin

To the Editor:

Recently we treated a patient with rifampin-induced eosinophilia similar to the patient described by Mungall and Standing.1 We believe the following is the second report of documented eosinophilia due to rifampin.

CASE REPORT

A 28-year-old black woman was admitted to the hospital with an eight-month history of weight loss, night sweats, and fever. Chest x-ray film showed bilateral pulmonary infiltrations and cavities plus loss of left lung volume. Sputum smears and cultures confirmed the diagnosis of tuberculosis. The hemoglobin level on admission was 8.4 gm/100 ml, and peripheral blood smear showed hypochromic, microcytic anemia. The bone marrow showed erythroid hyperplasia and slight plasmacytosis. Hematologic studies revealed a thalassemia and sickle cell trait.

She was started on INH 300 mg/day, ethambutol 800 mg/day, and rifampin 600 mg/day on July 28, 1978. Within six days she developed eosinophilia of 6 percent which increased to a maximum of 41 percent after 26 days (Table 1). Stool examination was negative for ova and parasites. No new clinical symptoms or signs appeared. Rifampin was discontinued on August 25, 1978, while INH and ethambutol were continued. The eosinophil count decreased within three days and eosinophilia disappeared within 18 days after rifampin was discontinued.

DISCUSSION

We believe our patient most likely had rifampin-induced eosinophilia, since no other cause could be found and the eosinophilia promptly decreased when rifampin was discontinued. The time sequence of developing eosinophilia within six days of starting rifampin and the peak absolute eosinophil concentration of 5500/cu mm are similar to that of Mungall and Standing's patient. When rifampin was discontinued, eosinophilia disappeared. Neither Mungall and Standing's patient nor ours had new signs, symptoms, or abnormalities associated with a drug reaction.

Eosinophilia due to rifampin is rare, but should be considered a possibility in patients who develop eosinophilia shortly after starting rifampin.

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REFERENCE


Table 1— White Blood Cell Count and Eosinophil Percentages

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