prick test, it seems that another mechanism is operating.

In 1974, Dolovich et al. described late skin reactions that were considered to be mediated by IgE and where histology of the skin test site revealed edema, mast cell degranulation and mainly eosinophilic perivascular infiltration. There were few neutrophils and no evidence of intravascular thrombi; neither did immunofluorescence reveal immunoglobulin or complement deposition. All these observed features are not characteristic of an Arthus reaction.

We believe we have observed the same reaction to animal allergens and suggest that a late skin reaction mediated by IgE be termed a Dolovich reaction.

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To the Editor:

It has been observed that beta-adrenoceptor agonists exert an anti-inflammatory action in skin reactions of experimental animals. In man, the response to intradermal injection of histamine provides a convenient model for study of altered vascular permeability and has been used to investigate the anti-inflammatory potential of a beta-adrenoceptor agonist in normal volunteers.

Histamine (0.5μg), histamine and terbutaline (100μg) or histamine, terbutaline and propranolol (20μg) were injected (0.1ml) in three sites in volar forearm skin. After 15 minutes, areas of skin swelling (mm²) were (mean ± SEM):

128.8 ± 5.0 for histamine (n = 24)
96.3 ± 7.1 for histamine + terbutaline
(n = 15) (P < 0.0005)
114.0 ± 8.6 for histamine + terbutaline + propranolol
(n = 15, n.s.).

These results demonstrate that when a beta-adrenoceptor agonist is applied locally, there is significant reduction of the histamine-induced skin lesion. This effect of terbutaline may be attributed to beta-adrenoceptor stimulation, since it is blocked by the beta-adrenoceptor antagonist propranolol. Our findings suggest an anti-inflammatory action of locally administered beta-adrenoceptor agonists by opposition of the permeability component of plasma protein extravasation.

This property of terbutaline could contribute to the therapeutic effect of inhaled terbutaline and other beta-adrenoceptor agonists in obstructive lung disease.

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To the Editor:


Response of Myelomatous Pleural Effusion to Chemotherapy

To the Editor:

Pleural effusion due to plasma cell infiltration of the pleura is an uncommon manifestation of multiple myeloma. We describe a patient with IgG myeloma and bilateral
pleural effusions containing numerous plasma cells who showed significant improvement of the effusions with chemotherapy.

CASE REPORT

A 49-year-old white man was found to have IgG multiple myeloma and bilateral pleural effusions. Thoracentesis yielded yellow fluid containing 6.9 g of protein, LDH 1,042 with 2,500 red blood cells/cu mm and 6,500 white blood cells/cu mm, 96 percent of which were identified morphologically as plasma cells (Fig 1). Pleural biopsy showed plasma cell infiltration. Systemic chemotherapy with vincristine, cyclophosphamide, adriamycin and prednisone was administered at three-four week intervals with resolution of the pleural effusions after three courses associated with a decrease in serum protein level. The patient did well for the next three months, but subsequently developed spinal cord compression, sepsis and expired seven months after diagnosis and onset of pleural effusions.

DISCUSSION

Pleural effusion in multiple myeloma is relatively infrequent and effusion due to pleural plasma cell infiltration is unusual. Diagnosis can usually be made by simple cytologic examination of the fluid using Wright's stain. Previous reports of myelomatous pleural effusion have indicated a poor prognosis with survivals usually less than four months. The present case had a somewhat longer survival (seven months).

We propose that a major determinant in the development of myelomatous effusion is the production of large quantities of immunoglobulin by malignant plasma cells in and near the pleura which raises the colloid osmotic pressure of the fluid such that normal absorption cannot take place. In myeloma a successful response to chemotherapy includes a significant decrease in production of monoclonal protein by the malignant clone of cells. One would then expect a decrease in pleural effusion with successful chemotherapy to the extent that the effusion is in fact dependent on protein production by the plasma cells. In this case, the effusions cleared as the blood level of IgG decreased with chemotherapy.

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REFERENCES


Pseudomembranous Colitis and Rifampicin

To the Editor:

To our knowledge, only four cases of rifampicin-associated pseudomembranous colitis are reported in the literature. Some reports do not document bacteriologic data. We report an additional case of pseudomembranous colitis apparently related to the administration of rifampicin in which Clostridium difficile and its toxin were isolated.

CASE REPORT

A 18-year-old woman was admitted on November 29, 1980 with a ten-day history of diarrhea and abdominal pain without fever and tenesmus. Since October 13 she had been treated for pulmonary tuberculosis with ethambutol (1,200 mg daily), rifampicin (600 mg daily) and isoniazid (300 mg daily). On October 30, isoniazid was stopped because of INH resistance; it was replaced by streptomycin (1 gm daily).

During the three weeks before institution of antibacterial chemotherapy she had been successively treated for intercurrent infections (urinary tract, genital tract), with TMP-SMZ, ampicillin, gentamicin and tinidazole and finally minocyclin (discontinued on October 20).

A coloscopy performed on November 26 disclosed pseudomembranous colitis. Cl difficile and its toxin were isolated in the stool. Rifampicin was withdrawn and pyrazinamide (2 gm daily) and cholestyramine (12 gm daily) were administered. After three weeks of treatment, the patient became completely symptom-free. Cl difficile was no longer isolated, but the toxin remained present for two more weeks. Cholestyramine was discontinued on December 18.

The patient was readmitted 20 days later for treatment of tuberculosis meningitis that necessitated reinstitution of rifampicin; at the same time, pyrazinamide was withdrawn. The diarrhea did not return, but Cl difficile and its toxin reappeared transiently positive.

DISCUSSION

Rifampicin can be implicated in this case of pseudomembranous colitis because the patient had not received antimicrobials other than the antituberculosis chemotherapy for three weeks before she developed colitis. On the other hand, clinical and bacteriologic improvement had been observed when rifampicin was stopped. It is actually thought that proliferation of Cl difficile and production of its toxin occur when the normal bowel flora is suppressed or disturbed by antimicrobials. Ethambutol and streptomycin (parenteral) do not have great activity on bowel flora. The patient was treated with cholestyramine at the same time that rifampicin was withdrawn, but recent double-blind studies show that this agent is not more active than placebo in the treatment of pseudomembranous colitis. When the patient was given rifampicin again, the stools were moni-