dehydration, shock, acidosis, fever, and hypothermia.

Their final 2 patients again provide strong presumptive evidence that acute renal failure in some cases of LD is due to rhabdomyolysis and myoglobinuria. However, in these cases the conclusive link between elevated levels of CPK and subsequent renal failure is missing; that is, they give us no information on the presence of myoglobin in the urine or what early urinalyses of the patients showed.

We agree with their conclusion that the incidence and relationship of rhabdomyolysis and renal failure in LD should be examined prospectively to facilitate early identification and treatment of this previously unrecognized complication of LD.

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REFERENCES

Recurrent Form of Amiodarone-Induced Pneumonitis

To the Editor:

We have read with interest the letter by Joelson et al particularly because we are following up a similar clinical case.

A 68-year-old man diagnosed as having amiodarone-induced pneumonitis was treated with suppression of the drug and prednisone (1 mg/kg/day) with rapid and good response. He was maintained on gradually tapering dose of prednisone and 1 month after its suppression (nearly 8 months after its introduction and 9 after the cessation of the amiodarone) the patient noticed fever (37.8°C), dry cough and exertional dyspnea. His physical examination showed moderate tachypnea and crackling rales at both lung bases. A slight restrictive pattern (FVC: 75% pred), and a marked reduction in Dco (52% pred) and in Kco (45%) was apparent on lung function tests. The chest x-ray film showed bilateral lung infiltrations with moderate to severe mottling in the right lung apex. We started prednisone (1 mg/ kg/day) and 5 weeks later a clinical, radiologic and functional improvement was obtained. We progressively tapered the prednisone to 0.30 mg/kg/day which is his actual dose. The patient has slight dyspnea on exertion and bilateral basal rales and his chest film shows bilateral moderate mottling in both bases. Lung function tests indicate normal lung volume, but decrease in Dco (62% pred) and Kco (58%).

We believe that like other forms of drug induced pneumonitis, amiodarone may induce 2 patterns of disease: an acute one, responding rapidly to large doses of prednisone and/or suppression of the drug, and a subacute or chronic form where this therapeutic approach cannot achieve a complete clinical, functional and radiologic remission.

Sometimes one has to decide whether to increase or maintain large doses of prednisone in order to get a complete response or maintain lower doses with a subtotal objective response. In these cases, the addition of an immunosuppressive agent may help to control the drug-induced alveolitis as it is the case in other forms of alveolar inflammation.3

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REFERENCES

To the Editor:

I read with interest the letter from Manresa et al describing an apparent recurrence of amiodarone pulmonary toxicity following corticosteroid therapy.

There were several parallels between their case and ours; specifically, both patients developed cough, dysnea and pulmonary infiltrates after the withdrawal of chronic corticosteroid therapy for presumed amiodarone-induced pulmonary toxicity. As in our case, improvement of symptoms and of objective parameters in their patient followed the resumption of prednisone therapy.

Unfortunately, I do not feel the authors furnished sufficient evidence to implicate amiodarone as the causative agent of their patient’s pulmonary symptoms. In this regard, additional data would be of considerable interest, namely: exclusion of several interstitial pulmonary disorders which may respond to corticosteroid therapy and histologic evidence of amiodarone-induced alveolitis. In addition, the duration and cumulative dosage of amiodarone therapy prior to the onset of symptoms (possibly correlated with chronic persistence of circulating drug) was not revealed. Finally, clinical follow-up to determine the duration of steroid dependency in this patient would be useful in evaluating the long-term sequela of amiodarone-induced pulmonary toxicity.

Mild cases of amiodarone pulmonary toxicity may respond solely to discontinuation of the offending agent, whereas many cases appear to require corticosteroid therapy. I know of no evidence suggesting that the addition of immunosuppressive therapy would be effective in the management of particularly severe or prolonged pulmonary toxic reactions associated with amiodarone. In fact, it is not even established that amiodarone-induced pulmonary toxicity is mediated by an immunologic process.1

There is presently a dearth of published information on amiodarone pulmonary toxicity, and the nature of this adverse reaction remains poorly understood. Further reports hopefully will clarify the pathophysiology and optimal management of this perplexing process.

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REFERENCE