fund, Memorial University of Newfoundland; the Gerontology Centre; Abbott Laboratories, Montreal, Canada; and Ross Laboratories, Columbus, Ohio.

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Fast atrial fibrillation induced by treatment of psoriasis with azathioprine

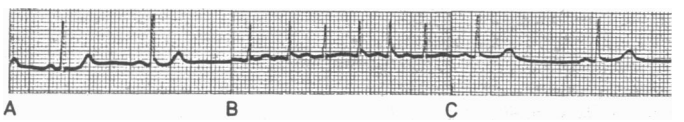
We describe a patient with psoriasis who developed fast atrial fibrillation in an idiosyncratic reaction to azathioprine.

Case report

A 60 year old man was referred for treatment of widespread psoriasis. He had suffered from the condition for over 20 years and had received various topical treatments and photochemotherapy. The response to psoralens and ultraviolet A treatment had been unsatisfactory after some initial improvement, and immediately before his referral to our department he had been using potent topical steroid ointments. He had a history of heavy alcohol consumption. Examination showed widespread confluent plaques of psoriasis, which had a glazed, thinned appearance. No abnormal cardiovascular signs were found, and he was in sinus rhythm at 60 beats/min with a blood pressure of 140/90 mm Hg. An electrocardiogram on admission (figure (*A*)) and chest *x* ray film were normal. We considered him to be unsuitable for routine treatment with dithranol, and azathioprine 50 mg three times daily was started. He received no other drug.

Four days later he became febrile, and over the next 48 hours his temperature reached 40°C and was accompanied by a sinus tachycardia. Azathioprine was stopped, and repeated cultures of blood and urine yielded negative results. Three days later, after the fever and tachycardia had completely settled, azathioprine was reintroduced. Within five hours he developed rigors and a fever (39°C) and was found to be in fast atrial fibrillation (figure (*B*)). Azathioprine was again immediately stopped, and the atrial fibrillation resolved within hours on bed rest. Tests of thyroid function yielded normal results, and serum electrolyte concentrations and a later electrocardiogram (figure (*C*)) were normal.

Subsequent inquiries about previous treatment at another hospital showed that he had developed a fever while taking azathioprine seven years before. It had recurred on reintroduction of the drug.



Electrocardiogram (lead II) (*A*) on admission; (*B*) five hours after azathioprine 50 mg; and (*C*) after bed rest and withdrawal of azathioprine.

Comment

Azathioprine is a widely used immunosuppressive agent that is effective in severe and disabling psoriasis.[1] Toxic effects include depression of bone marrow, hepatotoxicity, gastrointestinal upsets, reduced resistance to infection, malignant tumours, and teratogenicity. Because patients show better tolerance of and clinical response to methotrexate azathioprine has become a second line agent in severe psoriasis that is used when other treatments have failed or are contraindicated.[1]

Febrile reactions to azathioprine have been reported and can, as in our case, develop within hours after administration of the drug. The mechanism is unclear, but the fever may be associated with polyarthritis. We have found no reports of cardiac side effects of azathioprine, and neither the manufacturers (Wellcome Medical Division, United Kingdom) nor the Committee on Safety of Medicines has received reports of any such side effects in toxic reactions. Episodes of atrial fibrillation in normal hearts can be precipitated by other drugs, including alcohol,[2] inappropriate thyroxine treatment,[3] and nicotine,[4] as well as by non-cardiac events such as surgery, pneumonia, burns, mediastinal carcinoma, lymphoma, pulmonary embolism, and renal and biliary colic.[5] There was no precipitating event in our patient other than the administration of azathioprine, and the heart was clinically and electrocardiographically normal before and after the episode.

It is important for physicians using azathioprine to be aware of this serious but reversible adverse effect.

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Orchidopexy: theory and practice

Only 32% of orchidopexies are done by the recommended age of 5 years. The number of operations is four times greater than the incidence of undescended testis, suggesting that boys with retractile testes have unnecessary surgery. We decided to investigate further.

Present survey and results

With the help of colleagues in 12 hospitals in different parts of Britain we recorded the ages of 1285 boys undergoing orchidopexy between 1981 and 1983. The proportions who had surgery before the age of 5 ranged from 7% to 51%, with a mean of 32%.

We took the incidence of undescended testis as 1% and calculated the expected number of cases for six district hospitals with known catchment populations. In these the numbers of orchidopexies were two to four times greater than expected (table).

All general practitioners in the Medway district were asked when they would refer boys with undescended testis for surgery; of the 92 who replied, 69 (75%) said that they would do so before the age of 5 years.

We examined the hospital and, wherever possible, neonatal and community child health records of 58 boys who had had orchidopexies in 1983. In 15 cases the diagnosis had been made at the neonatal examination but none of these children, if otherwise well, had been followed up by paediatricians. In 22 cases the referral letter stated that the diagnosis had been made by a community medical officer, but in many cases the separate neonatal, hospital, child health clinic, and school medical records made it impossible to elicit this information.

Among these cases we found two where the testes had been noted to be

Expected and actual numbers of orchidopexies performed in six district general hospitals

Hospital	No of male births a year	No of orchidopexies a year	
		Expected	Performed
1 (Medway)	2500	25	52
2	1000	10	22
3	1900	19	43
4	1800	18	65
5	2000	20	87
6	1400	14	62
England and Wales, 1981	315 000	3150	14 070