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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 polyarthritides. 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

fully descended during infancy but where orchidopexies had been done after referral as a result of school medical examinations. One of these boys had had two operations for hypospadias and the other, a premature infant, was followed up by one of us (TML) for two years.

Comment

It is generally agreed that surgery for undescended testis should be performed by not later than 5 years of age. All but one of our consultant colleagues agreed with this, as did 75% of the general practitioners, yet only 32% of operations were done by the recommended age. Why does this happen and what does it mean?

The incidence of undescended testis after the age of 1 year is about 0.8%.^{1,2} Between infancy and puberty, however, retractility of the testis may make diagnosis difficult; one survey found that 6% of boys between the ages of 5 and 11 appeared to have undescended testes but by the age of 14 this figure had fallen to 0.6%.³ A paediatric surgeon mentioned this problem and said that he was reluctant to operate on boys in whom the diagnosis of undescended testis had not been documented during infancy.

Chilvers *et al* studied the increasing orchidopexy rate over the past 20 years and suggested that surgery on boys with retractile testes could explain the increase.⁴ We have documented two cases where this occurred and believe this explanation to be correct. Hence about 10 000 unnecessary operations—75% of the total—are performed in England and Wales every year; these cause considerable discomfort, endanger fertility, and, with an average stay in hospital of 4.4 days, cost about £4m.⁵

This unsatisfactory state of affairs could be avoided by two measures. Firstly, neonates with undescended testis should be followed up by a consultant paediatrician for one year; if the testis remains undescended the boy should be referred directly to a surgeon. Secondly, an older boy thought to have an undescended testis and for whom no infant record is available should be examined by an experienced surgeon—if necessary under general anaesthesia—so that retractile testes can be identified.

We thank our colleagues who collected data for our survey and also D I Beeby, P G Girolami, R W Hoile, D G Jenkins, and P J Jennings for permission to study patients under their care. We are also grateful to Mrs T Redknapp for secretarial help.

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Haemophilus parainfluenzae and H influenzae respiratory infections: comparison of clinical features

Haemophilus influenzae, although usually non-capsulated in the respiratory tract of adults,¹ is regarded as a possible pathogen when so isolated.² The role of *H parainfluenzae* is less clear and, despite causing invasive infection,³ its effect as a pathogen has been doubted.⁴ We report a study of 981 sputum isolates of haemophilus and relate the respiratory disease, clinical features, and antibiotic sensitivity to the organism isolated.

Materials, methods, and results

Using laboratory records to identify patients who had haemophilus isolated from the respiratory tract, we conducted a retrospective study of patients' case notes to record the clinical features at the time the specimen was produced. We also recorded bacteriological features of the isolate. Specimens

were inoculated on to horse blood agar and heated horse blood agar containing 10 units bacitracin/ml and incubated for 18 hours at 37°C in 7% carbon dioxide. Non-haemolytic haemophilus colonies were identified by typical appearance and the organism confirmed by Gram stain and satellitism with *Staphylococcus aureus*. Differentiation between *H influenzae* and *H parainfluenzae* was achieved by their ability (*H parainfluenzae*) or failure (*H influenzae*) to metabolise δ -aminolaevulinic acid.

The monthly frequency of isolations of *H influenzae* and *H parainfluenzae*, the underlying diagnosis and presenting symptoms, the antibiotic sensitivities, and the frequency of mixed growths were recorded and compared using χ^2 statistical analysis.

There were 689 sputum isolates of *H influenzae* and 292 of *H parainfluenzae*; *H influenzae* had its peak in March and *H parainfluenzae* in November. The patients were aged 12-98 years (mean 60 (SD 18) years).

The most frequently recorded symptoms associated with isolation of haemophilus were cough (with or without sputum production), dyspnoea, increased sputum purulence, and wheeze, and these varied little among the diagnostic groups of chronic bronchitis, asthma, pneumonia, bronchial carcinoma, and bronchiectasis. There was no difference in the symptoms produced by *H influenzae* or *H parainfluenzae* in these groups or in total (figure). In patients with chronic bronchitis 35% of *H influenzae* and 37% of *H parainfluenzae* isolates were obtained when there was no clinical evidence of exacerbation of the disease (NS).

Mean frequency of symptoms recorded for all biotyped hospital episodes. (No of episodes with symptom recorded/total No of episodes $\times$ 100%.)

Other organisms were isolated from 20% of specimens yielding *H influenzae* and 13% of those yielding *H parainfluenzae*, but this difference was not significant ($0.1 > p > 0.05$). Mixed growths were most common in bronchial carcinoma ($p < 0.001$); and in patients with pneumonia, 10 out of 38 had mixed growths, seven with *Streptococcus pneumoniae*, all but one of the serotypes (type 33) having been recorded in association with pneumonia in this hospital.⁵

Antibiotic sensitivities were determined in 954 isolates. Ampicillin resistance occurred in 9% of *H influenzae* (60% of these producing β -lactamase) and 7.5% of *H parainfluenzae* (58% β -lactamase producers). Fourteen per cent of *H influenzae* isolates were resistant to tetracycline, 1.5% to co-trimoxazole, and 0.3% to chloramphenicol, the corresponding figures for *H parainfluenzae* being 17%, 1.5%, and 0.4%.

Comment

We have shown that *H parainfluenzae* is associated with the same clinical spectrum of illness as *H influenzae* and that symptoms produced, occurrence in mixed growths, and the antibiotic sensitivities are the same for the two organisms. It is difficult to define an exacerbation in chronic bronchitis, as sputum purulence may not be a factor, and we relied on clinical impression at the time of presentation; again there was no difference in presentation whether *H influenzae* or *H parainfluenzae* was isolated. Whether haemophilus initiates infection or is merely a secondary infection is uncertain but there is no evidence that *H influenzae* and *H parainfluenzae* are different in this regard. In patients with pneumonia haemophilus may not have been solely responsible in all cases, since in seven *Str pneumoniae* was isolated.

H parainfluenzae and non-capsulated *H influenzae* are clinically indistinguishable; hence we suggest that *H parainfluenzae* should be reported by bacteriology departments and be considered pathogenic in a patient who has evidence of a respiratory tract infection if the organism is obtained in the presence of pus cells and in pure growth from the respiratory tract.