"Compendium des produits et spécialités pharmaceutiques" de 1983[7] sous la rubrique "Vibramycine" ne mentionne ni cet effet secondaire ni les précautions à prendre afin de l'éviter. Il est probable que l'oesophagite et l'ulcère oesophagien soient plus fréquents qu'on ne le croit à la suite de la prise de doxycycline. Il faudrait systématiquement recommander aux malades soit (a) de boire un grand verre d'eau si la prise est au coucher, ou encore de manger quelque chose après la prise, car la motilité oesophagienne diminue en position couchée, soit (b) de prendre la doxycycline durant le repas du soir, puisque cet antibiotique serait bien absorbé même en présence d'aliments ou de lait.

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Psychosis induced by sustained-release procainamide

Only three cases of procainamide-induced psychosis have been described in the literature,[1,2] although psychotic reactions have been reported with other antiarrhythmic agents, including propranolol,[3] lidocaine[4] and disopyramide.[5] To our knowledge the following case is the first detailed description of acute psychosis produced by sustained-release procainamide.

Case report

A 74-year-old man was admitted to hospital because of orthostatic dizziness, for which he had previously been hospitalized several times. He had had an inferior wall myocardial infarction 1 year earlier and had a history of lung cancer and alcohol abuse. There was no history of psychiatric symptoms. He had chronic obstructive pulmonary disease. He was initially treated with quinidine, aminophylline, digoxin, nitroglycerin and metoprolol. A 24-hour electrocardiogram done while he was on this regimen showed a baseline normal sinus rhythm with occasional sinus tachycardia, occasional wandering pacemaker, occasional multiform atrial tachycardia, occasional premature ventricular contractions and three episodes of ventricular tachycardia (up to five beats each).

In the third week of hospitalization the quinidine and metoprolol were stopped, and therapy with sustained-release procainamide, 750 mg given every 6 hours, was started. Three days later the patient became agitated and fearful. He removed his cardiac monitor and refused medical and nursing care. On two occasions that day, 6 hours after he had taken the drug the following levels were found: plasma procainamide 10.6 and 11.5 μg/mL (45 and 49 μmol/L), plasma *N*-acetylprocainamide 9.3 and 12.4 μg/mL (34 and 45 μmol/L), serum creatinine 2.3 and 2.1 (normally 0.7 to 1.4) mg/dL (203 and 186 [normally 62 to 124] μmol/L), blood urea nitrogen 19 and 21 (normally 8 to 22) mg/dL (serum urea 6.8 and 7.5 [normally 2.8 to 7.8] mmol/L), serum alkaline phosphatase 336 and 367 (normally 111 to 294) U/L and serum glutamic oxaloacetic transaminase 39 and 47 (normally 7 to 40) U/L. Procainamide was discontinued, and 25 mg of haloperidol was administered over the next 8 hours. The patient now sat quietly, although he continued to refuse care.

Forty-eight hours after procainamide was discontinued the patient's paranoid behaviour stopped, and 4 days later he was discharged on metoprolol therapy. Symptoms of psychosis did not recur, and he had a normal score on two mental status examinations.[6,7]

Comments

The patients described by Giardina and colleagues[8] usually had lower "predose" levels of procainamide and *N*-acetylprocainamide than did our patient, and all of the adverse effects disappeared within 24 hours after sustained-release procainamide was discontinued. Perhaps the hepatic and renal impairment in our patient explains the decreased clearance of the drug and its metabolite.

Patients usually receive 3 g of sustained-release procainamide per day initially, but most require between 3.3 and 6.3 g per day to achieve a 75% reduction in the frequency of ventricular premature depolarizations.[8] Our case shows that psychosis can occur at lower dosages and suggests caution in treating elderly patients who have hepatic or renal impairment.

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No crossover of hypersensitivity between zimelidine and fluoxetine

Zimelidine hydrochloride, a potent and relatively specific blocker of serotonin reuptake,[1] was recently removed from use as an antidepressant because of hypersensitivity reactions in the early stages of treatment, followed at times by Guillain–Barré syndrome.[2,3] Fluoxetine, also a specific blocker of serotonin reuptake,[4] has not yet been associated with such reactions. We report the only case to our knowledge of a patient in whom hypersensitivity to zimelidine developed and who subsequently was treated with fluoxetine, with no similar effects.

Case report

A 41-year-old woman who had a long history of depressive episodes was treated unsuccessfully with various tricyclic antidepressants, lithium, tryptophan and neuroleptics. She had a history of an anaphylactic reaction to administration of dye for intravenous pyelography but no known drug allergies.

The patient began therapy with zimelidine, 200 mg/d, 1 week after all other medication had been cleared from her system. Ten days later she complained of joint pain, chills and headache. A physical examination gave unremarkable results. The levels of serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) and lactic dehydrogenase (LDH), all of which had been within normal limits before therapy with zimelidine was started, were found to be mildly elevated (SGOT, 53 [normally 0 to 47] U/L, SGPT, 57 [normally 0 to 47] U/L and LDH, 241 [normally 110 to 230] U/L). Zimelidine was discontinued. Four days later the patient complained only of mild joint pain, and the liver enzyme levels had returned to normal.

One week after discontinuation of zimelidine all the symptoms had resolved, and therapy with fluoxetine was started, 20 mg the first day, 40 mg the second day and then 100 mg/d. The patient continued receiving the drug for 4 months without symptoms of hypersensitivity or change in the liver enzyme levels.

Comments

The lack of development of hypersensitivity to fluoxetine after hypersensitivity to zimelidine had developed in this patient suggests that the toxic reaction to zimelidine was related to some mechanism specific to the latter. Thus, the potent and specific serotonergic reuptake blockade common to the two drugs does not appear to be a factor in the development of hypersensitivity to zimelidine.

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Swimmer's lung

Swimmer's ear is well known, but I believe there is another syndrome — swimmer's lung. Many of my young patients come to my office with irritating coughs that seem to last throughout the summer. Clinical findings are absent, but there is a strong correlation between such a cough and the use of swimming pools that have been chlorinated.

Most public swimming pools are overchlorinated in my opinion — one can smell the chemical from a distance. This, one would presume, can cause a chemical pulmonary irritation with resultant irritant cough throughout the swimming season.

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Crohn's disease in a mother, father and son

The incidence of familial ulcerative colitis and Crohn's disease appears to be increasing. In a study in Alberta it was found that about 30% of cases of these two diseases are familial (M. Grace: personal communication, 1980). Genetic and environmental influence may play a role in inflammatory bowel disease. Kirsner[1] described a case in which Crohn's disease developed in a man 3 years after he had married. Sacroileitis and then ulcerative colitis subsequently developed in his wife. Whorwell and colleagues[2] reported the development of Crohn's disease in both a man and his wife after more than 30 years of marriage. Zetzel[3] reported ulcerative colitis in a man, Crohn's disease in his wife and ulcerative colitis in two of their three children.

Other cases recorded in the literature include that of a German family in which the father, son and two daughters all had Crohn's disease.[4] A Chicago family was reported in which the mother had ulcerative colitis; a similar condition developed in her 7-year-old son, and a similar disease of the terminal ileum developed in her husband 9 years later.[5] A Mississippi family was reported in which Crohn's disease developed in four of the five children.[6]

I report another family in which Crohn's disease developed, in the mother, father and son.