eventually required surgery, however, referral to an orthopaedic surgeon of all babies with unilateral loss of hip abduction in flexion is essential.

Appearances of plagiocephaly.

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¹ Dunn PM. Congenital postural deformities. *Br Med Bull* 1976;32:71-6.

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Reversible cholestatic jaundice and hyperamylasaemia associated with captopril treatment

Captopril is a useful drug in the management of intractable heart failure and hypertension unresponsive to conventional treatment. Its adverse effects include agranulocytosis in 0.3%, proteinuria in 1.2%, and pruritus or dermatitis in 7% of patients.¹ Although mild increases in liver enzyme activities have been noted in a few patients,² hepatitis is not a recognised complication of treatment with captopril and has been reported in only one patient.³

Case report

A 74 year old woman was admitted to hospital on 12 December 1982 for severe left and right sided heart failure. She had a five year history of ischaemic heart disease, including two episodes of myocardial infarction, and a pacemaker had been implanted for complete atrioventricular block. Her response to treatment, including high doses of frusemide, aminophylline, spironolactone, digoxin, and isosorbide, was not satisfactory. On 2 January 1983 captopril 25 mg by mouth thrice daily was started and was increased subsequently to 50 mg thrice daily, resulting in the disappearance of peripheral oedema and weight loss of 11 kg within three weeks.

On 28 January she was readmitted because of repeated episodes of absence manifesting as unresponsiveness to external stimuli without loss of consciousness and lasting for several minutes. On admission she was alert without dyspnoea or other signs of heart failure. The pulse was regular and blood pressure was 110/70 mm Hg. A neurological examination and electroencephalogram were normal. Urine analysis and a complete blood count yielded normal results. Her blood urea nitrogen concentration was 19 mmol/l (53 mg/100 ml) compared with 15 mmol/l (42 mg/100 ml) four weeks earlier. Serum aspartate transaminase activity was 208 IU/l (normal 10-45 IU/l) compared with 26 IU/l on her previous admission; alkaline phosphatase activity was 350 IU/l (normal 30-95 IU/l) compared with 135 IU/l; and total serum bilirubin concentration was 46 µmol/l (2.5 mg/100 ml). Erythrocyte sedimentation rate increased from 29 to 83 mm in the first hour.

During the following days her jaundice increased and generalised itching developed. There was no nausea, vomiting, or abdominal pain. The liver and spleen were not enlarged. Total bilirubin concentration increased to a maximum of 123 µmol/l (7.2 mg/100 ml) (direct 99 µmol/l (5.8 mg/100 ml)) and alkaline phosphatase activity to 1260 IU/l without a concomitant increase in serum aspartate transaminase or serum alanine transaminase activity. Serum amylase activity was 2000 IU/ml (normal 70-300) and urine amylase activity 30 000 IU/ml (normal 170-2000). She remained afebrile, and all blood cultures were negative. A technetium scan showed a normal liver and spleen.

After exclusion of extrahepatic biliary obstruction the possibility of drug induced cholestatic jaundice was considered, particularly because a few days earlier, when captopril had inadvertently been stopped for 24 hours, serum amylase activities had dropped from 2000 to 800 IU, increasing to 1790 and subsequently to 2400 IU with the resumption of captopril treatment. Treatment with captopril was stopped; the jaundice resolved within one week, and alkaline phosphatase and amylase activities returned to normal within two weeks. No other drugs had been added before the development of jaundice or withdrawn before its resolution. Throughout the ensuing three months she continued to enjoy good health and required only minimal diuretic treatment. At no time did she complain of abdominal pain or show any other signs of acute pancreatitis.

Comment

We did not carry out rechallenge followed by a diagnostic liver biopsy in view of our patient's age and her rapid improvement after treatment with captopril was stopped. The course of events was, however, suggestive of a causal relation between captopril and hepatitis. Extrahepatic obstruction was excluded, and the absence of fever or nausea, the negative serological findings, and the predominant cholestatic pattern of liver function tests were more in keeping with drug induced than viral hepatitis. Finally, the transient improvement in serum amylase activity after captopril was inadvertently stopped temporarily and the sustained improvement after it was finally stopped strongly support our contention that captopril was responsible for the observed abnormalities.

The importance of the raised serum and urinary amylase activities in our patient is not certain, particularly in view of the lack of any clinical manifestations and the normal endoscopic retrograde cholangiopancreatogram. Although painless pancreatitis cannot be ruled out, it seems unlikely in the circumstances.

Drugs that produce rashes often cause hepatic damage with both cholestatic and hepatocellular elements.⁴ Thus captopril might be associated with both of these complications. Our report thus emphasises the need for increased attention to hepatic function in patients receiving treatment with captopril.³

¹ Vidt DG, Bravo EL, Fouad FM. Drug therapy: captopril. *N Engl J Med* 1982;306:214-8.

² Elmenhary MM, Shaker A, Ramadan M, Hamza S, Tardos SS. Control of essential hypertension by captopril, an angiotensin converting enzyme inhibitor. *Br J Clin Pharmacol* 1981;11:469-75.

³ Vandenburg M, Parfrey P, Wright P, Lazda E. Hepatitis associated with captopril treatment. *Br J Clin Pharmacol* 1982;11:105-6.

⁴ Davis M, Williams R. Hepatic disorders. In: David DM, ed. *Textbook of adverse drug reactions*. Oxford: Oxford University Press, 1977: 146-72.

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