Necropsy showed that the immediate cause of death was a large pulmonary embolus. There was moderate left ventricular hypertrophy, and the kidneys, apart from slight surface granularity, were normal. The right adrenal gland weighed 25 g., being enlarged by the presence of a bright yellow cortical adenoma; the left adrenal was macroscopically normal. Histologically the kidneys showed the changes of benign hypertension, and the adrenal adenoma consisted of large lipid-laden cells resembling zona fasciculata and very similar to those illustrated in earlier reports (Milne *et al.*, 1957).

Comment

Although aldosterone output was not measured, there seems little doubt that primary hyperaldosteronism was the cause of this patient's hypertension and hypokalaemia, particularly as she was found to have a characteristic adrenal adenoma at necropsy. Cushing's syndrome may produce similar biochemical changes, but there were no other signs suggestive of this condition, and the slightly raised 24-hour urinary hydroxycorticosteroid excretion rate could be accounted for by the concurrent illness.

The electrocardiographic findings are of interest in view of recent reports of similar abnormalities in association with cerebrovascular accidents (Harrison and Gibb, 1964; Menon, 1964; Srivastava and Robson, 1964). In most of these the plasma electrolytes have been specified as normal, but the E.C.G. changes have been identical with those occurring in this case, consisting mainly of ST segment depression, T-wave inversion, and prominent U waves.

The association of cerebrovascular accidents with hypertension due to Conn's syndrome seems rare, since only three examples were found in a recent survey of 145 cases (Conn *et al.*, 1964). However, it is important not to overlook the possibility, since, granted survival from the cerebral episode, the hypertension can usually be cured by removal of the adrenal adenoma (Conn *et al.*, 1964; Smithwick *et al.*, 1964).

I should like to thank Dr. T. Simpson, under whom the patient was admitted, for his encouragement to describe this case.

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Iodine-induced Hypothyroidism Due to Benziodarone (Cardivix)

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The occurrence of jaundice in several patients under treatment with the long-acting coronary vasodilator benziodarone has been reported recently (Lee and Devey, 1964). Because of this the manufacturers have now withdrawn the drug from the market pending further investigations. We wish to describe a different complication which occurred in a patient during treatment with benziodarone.

Case Report

The patient, a man aged 56, had been complaining of intermittent claudication and mild angina of effort for about 18 months. At the beginning of 1964 his practitioner prescribed benziodarone, 300 mg. daily. Despite this, his symptoms continued and he was referred to hospital in May 1964 for further opinion. At that time the only abnormal clinical finding was absence of pulsation in the dorsalis pedis and posterior tibial vessels. He was clinically euthyroid. The serum cholesterol was 295 mg./100 ml. The electrocardiogram was normal. Radiological examination showed the cardiac shadow to be normal in size and configuration; there was extensive calcification in the arteries of both legs. The diagnosis was thought to be intermittent claudication associated with peripheral vascular disease and mild angina pectoris due to chronic ischaemic heart disease. No change in therapy was recommended and he continued to have benziodarone as before.

In August he began to notice new symptoms, which progressed during the next two months. He lost interest in his work and his memory became poor. He developed intolerance to cold, numbness and paraesthesiae of his hands, hoarseness, and impairment of hearing. When next seen by us in October he showed obvious clinical features of hypothyroidism. He was mentally and physically slow, with hoarse slurred speech. His skin was slightly coarse and his face puffy. No goitre could be felt. His tendon reflexes were myotonic. The serum cholesterol was 435 mg./100 ml. There was generalized flattening or inversion of the T waves in the limb and chest leads of the electrocardiogram. Radiological examination of the chest showed slight cardiomegaly. He was admitted to hospital, where further investigations were carried out, with the following results: serum protein-bound iodine greater than 25 μg./100 ml.; serum thyroxine, measured by a resin method (Pileggi *et al.*, 1961), 2 μg./100 ml.; thyroid clearance 19 ml./min.; plasma inorganic iodine 650 μg./100 ml.; absolute iodine uptake 2,900 μg./hr. These results are typical of iodine-induced hypothyroidism (Harrison *et al.*, 1963).

Administration of benziodarone was stopped and within three weeks he felt better and more alert. He became less hoarse and noticed the cold less. The paraesthesiae of his hands and the puffiness of his face disappeared. He was allowed home on 31 October. When seen again at the end of November he was clinically euthyroid. The electrocardiographic changes had regressed and the cardiac size had returned to normal. The serum cholesterol had fallen to 272 mg./100 ml. The plasma thyroxine had risen to a normal level (3 μg./100 ml.). Serum P.B.I. was still greater than 25 μg./100 ml.

Comment

One of us took a single dose of 100 mg. of benziodarone. During the next four hours the urinary excretion of iodine was approximately 600 μg., over 95% of which was inorganic. It has been shown previously that the basic abnormality in patients who develop iodine-induced hypothyroidism is an inability of the thyroid gland to regulate its iodine uptake in accordance with the plasma levels of iodide presented to it (Harrison *et al.*, 1963). Benziodarone contains about 46% iodine, a fraction of which is converted to inorganic iodine in the body. There can be little doubt that our patient developed an iodine-induced hypothyroidism as a result of continued administration of this drug during a period of about nine months. After withdrawal of the drug the condition cleared up spontaneously within two months.

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