with Hypaque was performed before the expected date of ovulation, and laparoscopy before menstruation.[1] All but one of the laparoscopies were performed under local anaesthesia[2] with about 30 ml aqueous methylene blue injected under low pressure into the uterine cavity. On laparoscopy the dye could be seen traversing patent tubes and spilling into the pouch of Douglas.

Bilateral tubal patency was shown in 27 patients by hysterosalpingography and 33 by laparoscopy (see table). In 70% of the patients known to have some degree of tubal occlusion the two methods gave closely similar results, whereas periadnexal adhesions were detected in 57 patients (66%) by laparoscopy but in only four patients (4·7%) by hysterosalpingography. Laparoscopy also permitted an assessment of the severity of the adhesions and the degree to which other organs were affected. Thirteen patients who appeared to have either bilateral or unilateral patent tubes on their hysterosalpingograms were shown by laparoscopy to have fimbrial phimosis.

Comment

These results show the advantages of laparoscopy over hysterosalpingography despite the accuracy of the latter in detecting intrauterine anatomical defects. Six patients shown by hysterosalpingography to have bilateral tubal occlusions were found at laparoscopy to have patent tubes. This might have been due to tubal spasm at the time of hysterosalpingography resulting from anxiety and apprehension.[3,4] Two patients shown by both hysterosalpingography and laparoscopy to have bilateral interstitial tubal occlusions conceived within three months after the laparoscopy. They had macroscopically normal tubes with limited fine adhesions. Possibly the injection of the solutions opened the tubes.

There was good correlation between the two procedures in 70% of the patients with unilateral or bilateral tubal occlusions. Nevertheless, the finding at laparoscopy of 13 patients with fimbrial phimosis and 57 with periadnexal adhesions shows the advantage of this method in identifying peritoneal as well as tubal factors that may be responsible for subfertility in patients with patent tubes.

The results indicate the superiority of laparoscopy in the diagnosis of infertility, as it gives a better appraisal of tubal structure and excludes peritoneal factors.

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Alcohol-induced Cushingoid syndrome

Alcohol has long been known to affect adrenocortical activity.[1-4] A rise in hydrocortisone concentrations to such an extent that Cushing's syndrome is mimicked has been alluded to in various reports,[1,4] though well-documented accounts are lacking. Here we report the case histories of three alcoholic patients with biochemical and physical abnormalities strongly suggesting Cushing's syndrome.

Case reports

Case 1—A 39-year-old housewife enjoyed excellent health until July 1974, when she developed anorexia, weight loss, vomiting, hypertension (210/120 mm Hg), muscle wasting, and weakness, together with tremor. She was tremulous with a grossly Cushingoid appearance (moon face, periocular pigmentation, buffalo hump, supraclavicular fat pads, truncal obesity, crural ulcers, and obvious muscle wasting). Serum transaminase concentrations were double the normal. The morning plasma cortisol concentration was high (1662 nmol/l, 59 μg/100 ml) and inadequately suppressed (388 nmol/l, 14 μg/100 ml) by an overnight dose of 2 mg of dexamethasone. A diagnosis of Cushing's syndrome was considered. On admission to hospital three weeks later the physical findings and serum transaminase concentrations were unchanged. Glucose tolerance was impaired. Plasma cortisol concentrations were still high—1324-1656 nmol/l (48-60 μg/100 ml)—with a diminished circadian rhythm. Remarkably, a few days after admission the raised cortisol concentrations (along with the transaminase values) decreased and became normally suppressible by dexamethasone. The tremulousness, hypertension, and Cushingoid appearance also diminished.

The concurrent decrease of plasma cortisol and transaminase concentrations suggested alcohol abuse, which was finally admitted by the patient, who confessed that every day she drank 0·5 l of Dutch Jenever. Initially after discharge from the hospital her plasma cortisol levels remained normal, but later they were regularly found to be raised, strongly correlating with raised transaminase levels, suggesting that she had renewed her alcohol abuse.

Case 2—A 54-year-old milkman was admitted to hospital in November 1974 for evaluation of his hypertension (210/120 mm Hg), vertebral wedging, glucose intolerance, disturbed cortisol rhythm (3174 nmol/l, 115 μg/100 ml, at 8 am and 3060 nmol/l, 111 μg/100 ml, at 8 pm), and Cushingoid appearance. He said that he drank 0·5 l of Dutch Jenever daily. He appeared overtly Cushingoid with facial mooning, truncal obesity, and easy bruising. Serum transaminase concentrations were double the normal values. Basal morning plasma cortisol concentrations were initially high (1683-2208 nmol/l, 61-80 μg/100 ml) with either a dampened or absent circadian rhythm. Dexamethasone (0·5 mg four times daily for two days) inadequately suppressed plasma cortisol concentrations to 938 nmol/l (34 μg/100 ml). Cushing's syndrome was diagnosed. Two weeks after admission, however, plasma cortisol concentrations and blood pressure returned to normal (as did the serum transaminase concentrations) and remained so until discharge.

Case 3—A 30-year-old man was admitted in December 1974 for evaluation of his glucose intolerance and hypertension (150/100 mm Hg). He admitted to alcohol abuse. He was a plethoric young man with overt moon face, diffuse obesity, and purple abdominal striae. Serum transaminase concentrations were four times the normal values, while the histological examination of a liver biopsy specimen showed findings compatible with alcohol abuse. The morning plasma cortisol concentration was raised to 1021 nmol/l (37 μg/100 ml) and was inadequately suppressed by an overnight dose of 2 mg of dexamethasone to 386 nmol/l (14 μg/100 ml). During his stay in hospital, however, the plasma cortisol concentration returned to normal and appeared adequately suppressible by dexamethasone. Concurrently serum transaminase values became normal.

Discussion

Clinical and biochemical findings suggesting Cushing's syndrome were found in our three patients with proved alcohol abuse. Remarkably, most of the abnormalities compatible with Cushing's syndrome disappeared one to three weeks after abstinence from alcohol in hospital. The raised plasma cortisol concentrations returned to normal and became adequately suppressible by the time the initially raised serum transaminase concentrations had also returned to normal. Excessive alcohol intake was considered to account for both the liver impairment and hypercortisolism, which finally resulted in a Cushingoid appearance. The mechanism by which alcohol induces hypercortisolism is still not clear, though alcohol-mediated pituitary hypersecretion of corticotrophin[1,2,4] rather than impairment of liver function[2] seems to be responsible.[2,4] It remains conjectural why only some alcoholics show signs of adrenal overactivity and become Cushingoid.

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