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SHORT REPORTS

Ovarian ectopic pregnancy with IUCD

We describe three cases of ovarian ectopic pregnancies with an intra-uterine device (IUCD) in situ, and suggest that prostaglandins may have played a major part in this combination. The IUCD alters the ratio of the prostaglandins secreted, so that tubal peristalsis is reversed and the fertilised ovum is carried the opposite way.

Case reports

Three multigravidae presented with varying degrees of abdominal pains. All of them had suffered from menstrual irregularity of recent duration, and all had had Lippes' loops inserted for over two years. X-ray films confirmed that the device was in position in each case. Laparotomy showed right-sided cystic ovarian swelling in one case and left-sided in the other two. The other ovary and both Fallopian tubes in each case were intact and uninvolved. Ovarian tissue was conserved in two cases, but the ovary had to be removed in one because of heavy, uncontrollable bleeding. The pathologist reported the histological features of pregnancy in the tissue excised.

Discussion

Ovarian pregnancy is very rare, a ratio of 0.7 ovarian per 100 ectopic pregnancies having been suggested.¹ Women who wear an IUCD appear to have higher incidence, a ratio of one ovarian to nine ectopic pregnancies having been reported.² Our cases fulfil the diagnostic criteria formulated by Spiegelberg.³

Very little is known about the mode of action of the IUCD. It has been claimed that it distorts and distends the uterine cavity and thereby impairs its tonicity, or that it causes a foreign body reaction with leucocytic infiltration resulting in an environmental hostility to the ovum or that the macrophages phagocytose the sperms. This spermatotoxic or blastotoxic hypothesis cannot explain the high relative frequencies of both tubal and ovarian ectopic pregnancies in patients fitted with IUCDs. Lehfeltdt *et al*¹ suggested that the anti-fertility effect is maximal in the endometrium, weaker in the tubes, and absent beyond that—that is, about the ovary. This pattern, they thought, suggested direct enzymatic or chemical action, possibly by retrograde flow from the endometrium outwards.

We suggest that perhaps the chemical action is due to a substance resembling prostaglandin, which is secreted abundantly from the endometrium causing contractions and preventing implantation. Prostaglandin is also secreted from the fallopian tubes but in lesser amounts. Nevertheless, it is not secreted from the non-contractile ovaries.

Alternatively, possibly the IUCD alters the ratio of the amount of the prostaglandins secreted. Therefore, instead of the normal contraction of the proximal part of the tube and relaxation of the distal segments, causing a suction action which favours the entrance of the ovum from the abdominal cavity into the tube, and its retention in the oviduct until fertilisation,⁴ the reverse occurs and the reversed suction and peristaltic action of the tube (due to the effect of the altered ratio of prostaglandins) will carry the ovum the opposite way. This hypothesis could also explain the reported cases of peritoneal pregnancies in women fitted with IUCDs.

In most of the reported ovarian ectopic pregnancies in women with IUCDs, the right side has been affected.⁵ For tubal pregnancies it has

been claimed that the right side is more often implicated possibly because of the proximity of the appendix. Such might also be the case in the ovarian ectopic pregnancy, but of our cases, two were left-sided and one right-sided; moreover, the free end of the Lippes' loop was found to be pointing towards the affected side in each case. Hence this might be an additional element in localising the condition.

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D C shock: spontaneous reversion to sinus rhythm during anaesthesia with propanidid

In the 1950s several derivatives of eugenol, the main constituent of oil of cloves and cinnamon leaf oil, were found to have short-acting anaesthetic properties.^{1 2} One of these, propanidid, causes sinus tachycardia and transient systemic hypotension, a decrease in stroke volume, and an increase in cardiac output as well as a slight fall in peripheral vascular resistance.^{3 4} We have used propanidid as an intravenous anaesthetic agent in nine episodes of arrhythmia in seven patients requiring cardioversion. In four episodes in two patients (cases 1 and 2) the arrhythmia reverted to sinus rhythm after injection of propanidid.

Case reports

Case 1.—A 19-year-old Indian girl presented with a ventricular tachycardia (200 beats/min) and hypotension (B.P. 80/70 mm Hg). She reverted to sinus rhythm during induction of anaesthesia with propanidid, 400 mg (see fig.). Two hours later the ventricular tachycardia recurred. It again reverted to sinus rhythm after a further 400 mg propanidid. Subsequent epicardial mapping confirmed the presence of a re-entry ventricular tachycardia. A myocardial biopsy showed histological changes compatible with a giant cell myocarditis.

Case 2.—A 60-year-old woman had a long history of paroxysmal supra-ventricular tachycardia. On previous occasions her arrhythmia had responded

Praecordial leads of 19-year-old girl (case 1). Upper: during episode of ventricular tachycardia. Lower: reversion to sinus rhythm during induction with 400 mg propanidid.

to intravenous practolol, but this failed during an admission in November 1974. She reverted to sinus rhythm soon after receiving 400 mg propanidid. In March 1975 she was readmitted with a similar tachycardia. Five minutes after receiving 500 mg propanidid she again reverted to sinus rhythm.

Case 3.—A 22-year-old woman with Fallot's tetralogy, reconstructed seven years earlier, developed a ventricular tachycardia. She failed to revert with propanidid though her ventricular rate fell from 190 to 170 beats/min.

Case 4.—A 64-year-old man with ischaemic heart disease developed ventricular tachycardia. He did not revert after 400 mg propanidid and D.C. shock was applied one minute later.

Cases 5 and 6.—These two patients with atrial fibrillation also failed to revert after propanidid.

Case 7.—This patient went into atrial fibrillation after a gastrectomy. The fibrillation spontaneously reverted to a supraventricular tachycardia. After 250 mg propanidid the patients went into atrial flutter.

Thus propanidid failed to control the rhythm disturbance in cases 3 to 7. In case 7 it converted supraventricular tachycardia to atrial flutter.

Discussion

Recognition of the antiarrhythmic properties of propanidid is not new. Johnstone and Barron³ found that it abolished for about two minutes ventricular arrhythmias that developed in 10 patients during light halothane anaesthesia. They concluded that propanidid reduced the risk of precipitating ventricular arrhythmia during endotracheal intubation. They suggested that it depressed the conducting system of the heart in a manner similar to that of quinidine and procaine. Two of their patients developed complete atrioventricular block after propanidid. However, Plagne and Barachet⁵ did not observe any antiarrhythmic effects from it in 258 patients before cardioversion.

Thus it seems that propanidid has an antiarrhythmic action which may be of benefit in certain arrhythmias. We are unable to determine whether this action is dependent on dose or rate of infusion or how long should be allowed for the conversion to occur before instituting D.C. shock. We believe that these properties warrant further investigation.

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Raised serum prolactin levels associated with hirsutism and amenorrhoea

Raised serum prolactin levels are usually associated with pregnancy, lactation, galactorrhoea, and hypophyseal tumours.^{1,2} The present study suggests that they may also be associated with hirsutism.

Patients, methods, and results

Out of 40 women with secondary amenorrhoea studied six were hirsute and 11 had galactorrhoea. The ovaries were enlarged in two patients (cases 1 and 3 (see table)) and genital hypoplasia was present in four (cases 2, 4, 5, and 6).

The circulating prolactin levels were measured by a specific radioimmunoassay (National Pituitary Agency standard V.L.S.1). The upper level in normal women was 30 $\mu\text{g/l}$. Urinary excretion of 17-ketosteroids, 17-hydroxycorticosteroids, and pregnanetriol were measured by routine clinical laboratory methods and the plasma testosterone levels by radioimmunoassay.

Serum prolactin levels were above 30 $\mu\text{g/l}$ in 20 amenorrhoeic women, of whom five were hirsute and 11 had galactorrhoea. Five out of six patients with amenorrhoea and hirsutism had raised circulating prolactin levels (see table). Of these two had galactorrhoea. Four of the six hirsute women had no galactorrhoea. Excretion of 17-ketosteroid and 17-hydroxycorticosteroid was normal in all patients. The plasma testosterone level was abnormally high in one patient (case 4) whose serum prolactin level was also raised. In a follow-up of this patient the plasma testosterone decreased to normal during oestrogen-progesterone treatment. Later, when bromocriptine was given in a daily dose of 5 mg, her serum prolactin and plasma testosterone levels were normal.

No abnormality of the pituitary fossa was seen on tomographic examination in any of the patients.

Discussion

The classic report of Forbes *et al.*³ described an association between polycystic ovaries, hirsutism, galactorrhoea, and amenorrhoea in patients with or without a pituitary tumour. We found raised serum prolactin levels in the amenorrhoea-hirsutism syndrome without any signs of a pituitary tumour or galactorrhoea. Prolactin synergizes with LH stimulating testosterone synthesis in the testis⁴ and promotes androgen formation by the human adrenal in organ culture.⁵ Both prolactin and testosterone levels were abnormally high in our case No. 4, and possibly testosterone secretion was increased because of the raised serum prolactin levels. But the testosterone levels returned to normal during oestrogen-progesterone treatment, when prolactin levels were still high, and during subsequent bromocriptine treatment both testosterone and prolactin levels were normal.

Though an interaction between prolactin and testosterone remains to be proved our results suggest an association between raised serum prolactin levels and hirsutism in amenorrhoeic patients.