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no cause for this hypertension: urinary output of catecholamines was normal; the arteriography demonstrated renal arteries of normal calibre; the nephrogram showed no parenchymous anomaly. Nevertheless, with a normal saltfree diet she demonstrated low serum potassium (between 2.7 and 3.1/l) (2.7 and 3.1 mEq/l). She was prescribed 300 mg of spironolactone, 20 mg of furosemide, 750 mg of methyldopa per day, and strict bed rest. But the salt-free diet was not strictly followed. The patient was readmitted one month later with persistent hypertension

at 210/160 mm Hg, standing and lying down. Renal function rapidly deteriorated. The dosage was augmented combining diuretics (spironolactone and furosemide) with methyldopa and a blocking drug. This was not effective. Diazoxide was administered for a short time bringing her blood pressure up to 130/80 mm Hg lying down and 100/70 mm Hg standing. Retinal haemorrhaging receded but azotemia reached 1.80 g/l. After eight days her blood pressure rose to 180/140 mm Hg accompanied by heart failure and severe oliguria. Even though large doses of furosemide were administered diuresis was less than 100 ml/24 h and dialysis was necessary twice a week.

A bilateral nephrectomy was performed on 20 January 1971. The microscopic appearance of the kidney tissue was typical of malignant nephro-angiosclerosis. After 10 days her blood pressure stabilized at 150/100 mm Hg. Her general condition rapidly improved and the patient was able to return home 12 days after surgery, without medication, but she had to follow a diet containing less than 2 g of Na Cl in 24 hours. When examined six weeks later her general condition was excellent with blood pressure at 140/80 mm Hg before dialysis. In November 1972 she underwent a renal graft and after 10 months her blood pressure still remained normal.



Renal vascular disease: fibrinoid necrosis.

Discussion

This case fits into the framework of hypertension due to oestroprogesterone. Authors differ about the frequency of this hypertension. Where there has been a marked increase in systolic arterial pressure it has usually remained within normal limits.⁴ These hypertensions are usually benign and recovery is rapid after the contraceptive has been discontinued.⁵ Nevertheless, when oestro-progesterones are prescribed blood pressure should be carefully watched and if arterial hypertension arises, this method of contraception should be discontinued, except in special cases.

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Department of Nephrology and Metabolic Disorders, Edouard Herriot Hospital, Lyon 69003, France

- P ZECH, MD, physician
- G RIFLE, MD, physician A LINDNER, MD, physician
- N SASSARD, мр. physician N BLANC-BRUNAT, мр. physician (sponsored by INSERM)
- J TRAEGER, MD, physician

Ocular reaction to propranolol and resolution on continued treatment with a different beta-blocking drug

A hypertensive patient treated for 20 months with propranolol developed ocular symptoms and signs similar to those described after practolol. Changing to oxprenolol was associated with remission of the ocular reaction.

Case report

In June 1973 a 48-year-old woman presented with asymptomatic, stable, uncomplicated hypertension (blood pressure 170-190/120-130 mm Hg). She had no history of allergy, rashes, or ocular disease. Treatment with propranolol twice daily, 40 mg for four weeks, 80 mg for four weeks, and finally 160 mg reduced the blood pressure to 155/105 mm Hg. This dose was con-tinued without incident until April 1975, when the patient complained of dryness of the mouth associated with a prickling sensation of the eyes, particularly the left. She had no rash or other evidence of systemic disease. Schirmer's test showed a slight diminution of tear production in the right eye and a definite diminution in the left eye. Slit-lamp examination showed generalised hyperaemia of her conjunctivae with foci of new vascular loops in the depths of the lower fornix and areas of superficial whitening obscuring underlying vessels (fig).

Because of the effective control of her blood pressure with propranolol, and in view of the reported absence of cross-reactions,¹ she was transferred to treatment with oxprenolol in a twice daily dose of 80 mg, increased after one month to 160 mg. At this dosage her blood pressure was maintained at 155-165/100-105 mm Hg at rest. After six weeks' treatment on oxprenolol and methyl cellulose eye drops 0.5% four times daily, in both eyes, her dry mouth and prickling sensation in the eyes diminished leaving only an occasional slight burning sensation in the left eye. Further ocular examination showed regression of the conjunctival vascular loops and pale areas although tear secretion by Schirmer's test was unchanged.



Foci of new vessels (smaller arrow) and superficial pale areas (larger arrow) in lower fornix of left conjunctival sac.

Discussion

Many cases of oculocutaneous reactions to practolol have been reported,^{1 2} but only rarely in patients during treatment with other beta-adrenoceptor antagonists.³ Although rashes have been reported during treatment with propranolol,3 this patient appears to be the first one described with an ocular reaction. The lesions observed were similar to, but milder than, those observed after practolol² and receded when the patient was transferred to oxprenolol. This absence of cross sensitivity between different beta-adrenoceptor antagonists confirms the experience of others.1 Nevertheless, in view of the circumstantial nature of the evidence in most reports, claims of specific reactions to beta-blocking drugs other than practolol must be viewed with reserve.

Generally it is important to emphasise that the development of this syndrome is extremely rare, given the very many patients under treatment with these drugs. The efficacy of beta-receptor antagonists in the treatment of ischaemic heart disease, hypertension, and dysrhythmias is beyond question and undoubtedly also they are

among the most significant advances in the medical treatment of these conditions in the history of medicine. These facts must be weighed against the extremely rare instances of these relatively minor epidermal reactions. Rashes are not infrequent in hypertensive patients taking methyldopa, while 1 in 5 patients treated with the latter develop a positive Coombs test and 1 in 500 go on to develop haemolytic anaemia.5 Such features have occasioned less comment than the development of this rare and usually reversible oculocutaneous syndrome in patients on treatment with beta-blocking drugs.

We are appreciative of the professional advice offered in this case by Mr B Martin.

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Departments of Surgery (Ophthalmology) and Medicine (Cardioascular Unit), University of Leeds, General Infirmary, Leeds LS1 3EX

- R BEVIS CUBEY, FRCS, DO, tutor in ophthalmology
- S H TAYLOR, BSC, FRCP, consultant physician and senior lecturer in medicine

Ischaemic necrosis of lesser curve after proximal gastric vagotomy

Avascular necrosis of the lesser curve of the stomach is a potentially fatal complication of proximal gastric vagotomy.^{1.5} After an uneventful operation the patient usually shows some evidence of delayed gastric emptying or of "ileus." On the third to sixth postoperative day collapse occurs due to peritonitis from a free perforation of the stomach. In patients who have survived this disaster the perforation was closed soon after its development.¹ ⁵ We report here a different presentation of this dangerous complication.

Case report

A 50-year-old hypertensive man progressed normally after an uneventful proximal gastric vagotomy for chronic duodenal ulceration until the fourth postoperative day, when he had a hypotensive episode. The next day he collapsed and passed a large melaena stool. A haemoglobin level of 7.4 g/dl (7.4 g/100ml) suggested that part of the bleeding had occurred the previous day. Treatment with 6 units of blood over 24 hours restored the central venous pressure to normal and the haemoglobin to 10 g/dl. Two days' later however, a further haematemesis occurred, so the abdomen was immediately reopened. The stomach and entire small bowel were distended with blood. A congested area high on the lesser curve of the stomach developed at a touch into a free perforation of the stomach out of which blood clots extruded. It seemed logical to complete the disruption of the lesser curve, and this was done easily with the side of the finger to reveal a large oval ulcer $(6 \times 2 \text{ cm})$ on the lesser curve in the middle of which a small artery was bleeding fiercely. In contrast, the stomach mucosa was purple except in the fundus where it was a normal pink colour. A high partial gastrectomy was performed without using clamps (but with very little bleeding) by cutting the stomach at the junction of viable and ischaemic mucosa. The patient made a good recovery and 5 months later was well apart from a limited appetite and a dumping tendency.

Discussion

Halvorsen et al³ noted that the lesser curve of the stomach is a potentially ischaemic area, the implication being that sloughing of the lesser curve is a specific hazard of proximal gastric vagotomy. In our case the slough was plugged by lesser omentum. An eroded artery in the omentum became the source of bleeding. Surgeons who adopted proximal gastric vagotomy for uncomplicated duodenal ulceration believed it to be free from both serious and minor complications. Unfortunately, this view must be qualified. In

Johnston's survey⁴ sloughing of the lesser curve occurred in 7 out of 4857 operations, an incidence of recognised cases of 1.4 per 103. Assuming that the occurrence of sloughing follows a binominal distribution 99% confidence limits for the true incidence would be 3.6 per 10⁵ operations at the lowest, and 2.8 per 10³ operations at the greatest. If the incidence should prove to be 2-3 per 10³ operations the complication will have to be taken seriously. Our case shows that sloughing of the lesser curve of the stomach may present as haemorrhage as well as perforation.

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Surgical Unit, University College Hospital Medical School, London WC1E 6JJ

FIONNA P MOORE, MB, BS, house surgeon J H WYLLIE, MD, FRCS, reader in surgery

Minimising blood loss in caesarean section

For surgeons working in hospitals patronized by the first among the martyrs of childbirth in India-the rural wife-emergency caesarean section on an exhausted, shocked, and exsanguinated woman is a common task, with or without the complication of rupture of the gravid uterus. Of the latter, one was seen every other day in the first 10 days of 1975-and three on the 10th. Blood is not always available for transfusion and is always expensive (50 rupees, £3, or \$5 per 330 ml), so that the prevention of undue bleeding is simply lifesaving.

Method

As soon as the child is born (or in accidental or unavoidable antepartum haemorrhage, fetal death, or rupture of the uterus before delivery) a straight intestinal clamp, six inches (15 cm) long with longitudinal or diagonal striations of the blades, is applied along each side of the uterus. The clamp encloses the whole length of the broad ligaments, including the infundibulopelvic ligaments, and cuts off, gently and temporarily, the entire blood supply of the viscus. It is then possible to open and close the uterus. Also an inspection to decide whether to repair or remove the uterus can be made more easily. If the clamps are applied before delivering a live child, speed will offset the effect of suppressing the placental circulation. After closing the uterine wall and before stitching the peritoneal flap the release of the intestinal clamps will show any inefficient haemostasis, and allow the venous engorgement of the broad ligament.

It is, of course, necessary to make an incision that is longer than usual and it should be midline and subumbilical to allow the clamps to be applied on the unemptied uterus. This will make the manoeuvre unacceptable where the bare abdomen, free of blemish or bulge, is popularly displayed. In those cases the transverse, suprapubic incision is imperative. But in India unsightly abdominal striae and wide separation of the recti are universal in the parous. A transverse scar is not appreciated because the hair of the mons veneris, which should mark it, is usually plucked or close shaved, and the vertical scar blends into the linea nigra.

Comment

My method is, recognizably, inspired by Victor Bonney, who used to advise occlusion of the uterine vessels by sponge forceps during extended myomectomy. Should it become popular, a modification by shortening the shanks of the intestinal clamps would make the instruments more handy.

Nazareth Hospital, Mokameh, Bihar, India MEAVE KENNY, MD, FRCOG, obstetrician-in-charge