SHORT REPORTS

Snare polypectomy by sigmoid-rectal intussusception

Appreciable bleeding or perforation after colonoscopic polypectomy is relatively unusual. Nevertheless, either may occur with broad-based sessile polyps owing to insufficient electrocoagulation of the base resulting from excessive current application, excessive snare-wire tightening, or a combination of both. If a polyp base is over about 2 cm in diameter the endoscopist has either to remove it "piecemeal" in several sessions or refer the patient for abdominal surgery.

Sigmoid colon polyps have been excised after deliberate intussusception to the anus using a conventional sigmoidoscope and surgical forceps.¹ On two occasions recently we have successfully used this approach at colonoscopy to remove polyps which would have been dangerous or impossible to remove endoscopically.

Case reports

Case 1—A 63-year-old man was admitted for colonoscopic snare removal of a sigmoid colon polyp diagnosed radiologically. He gave an eight-year history of recurrent colicky lower abdominal pain. On five occasions he had noticed a "lump" which had prolapsed to the anus but could be reduced by lying supine. Colonoscopy was performed with an Olympus CFMB-3 colonoscope. A 2.5-cm, broad-based polyp in the proximal sigmoid colon was snared and polypectomy attempted. The patient began to have pain from

Diagram of broad-based mid-sigmoid colon polyp before and after snare loop intussusception.

peritoneal irritation before visible electrocoagulation occurred. It was judged dangerous to continue and that the shape of the polyp was unsuitable for complete piecemeal removal. It was resnared with a handleless snare wire.² This was left in situ tightened on to the base of the polyp, and the colonoscope was withdrawn. The next day under general anaesthesia the polyp was intussuscepted to the anus by gentle traction on the snare wire and locally excised. The wound was sutured with absorbable sutures. The polyp histologically was a benign tubular adenoma and was completely excised. The patient was discharged two days later.

Case 2—A 65-year-old man was admitted for colonoscopic removal of three polyps shown radiologically. Snare polypectomy of two 1-cm polyps in the mid-transverse colon was uneventful. The third polyp in the mid-sigmoid colon was broad based and 2.5 cm in diameter. Snare polypectomy was judged to be difficult and hazardous because of the configuration of the polyp base. The handleless snare loop was closed loosely at the base, the colonoscope withdrawn, and an artery forceps used to hold the loop closed. In the operating theatre and under general anaesthesia the polyp was intussuscepted to the anal verge by gentle traction on the snare wire. The base was grasped with sponge forceps, transfixed with a stay suture, and the polyp excised with scissors. The pedicle was then sutured with absorbable sutures. The polyp was a benign tubular adenoma, completely removed. The patient was discharged the next day.

Comment

Snare intussusception in these patients minimised the risks of bleeding and perforation, the latter possibility being signalled in the first case by pain from peritoneal heating. Probably the first patient had been having episodes of spontaneous sigmoid-rectal intussusception, with the polyp at the apex of the intussuscepted loop,³ and this facilitated delivery per anus. The second case, however, shows that the technique is feasible even without such a history. Traction to the rectum should be enough to allow controlled surgical removal of large polyps with an operating proctoscope or rectal retractors.

In a few patients with broad-based polyps in the sigmoid colon the intussusception technique should be considered as a way of avoiding the risks of endoscopic polypectomy or the need to undertake abdominal surgery.

¹ Parks, A G, *Ergebnisse Der Angiologie*, p 338. New York, F K Schattauer, Verlag-Stuttgart, 1976.

² Shinya, H, and Wolff, W, *Hospital Practice*, 1975, 10, 71.

³ Goligher, J C, *Surgery of the Anus, Rectum and Colon*, p 312. London, Baillière Tindall, 1975.

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St Mark's Hospital, City Road, London EC1V 2PS

P E GILLESPIE, MB, FRACP, clinical fellow

R J NICHOLLS, MB, FRCS, senior surgical registrar

J P S THOMSON, MS, FRCS, consultant surgeon

C B WILLIAMS, BM, MRCP, consultant physician

Pulmonary oedema after propranolol therapy in two cases of phaeochromocytoma

There is little recorded evidence to support the assertion that beta-adrenergic blockade without concomitant alpha blockade may be harmful in patients with phaeochromocytoma.¹ Indeed, a recent case report suggested that beta blockade (without alpha blockade) was life-saving in a patient with adrenergic crisis due to phaeochromocytoma.² We describe two cases of phaeochromocytoma in which life-threatening pulmonary oedema followed soon after the introduction of beta-blocker treatment.

Case reports

Case 1—A 39-year-old man was referred to outpatients with hypertension. He complained of abdominal pain, headaches, night sweats, easy fatigability, and "shakiness." Treatment with propranolol 40 mg twice daily and chlorothiazide had been started eight days previously. The important clinical findings were obesity and a blood pressure of 160/110 mm Hg. Later the same day he collapsed with left-sided chest pain, dyspnoea, and haemoptysis. When examined in hospital one hour later he was ashen grey, his pulse rate was 120/min, and blood pressure 110 mm Hg systolic. He was in left ventricular failure with pronounced peripheral vasoconstriction. Electrocardiography showed ST segment elevation in anteroseptal leads. Over several days T wave inversion evolved in a similar distribution. Serum creatine phosphokinase and serum lactic dehydrogenase concentrations were slightly raised.

After admission he improved rapidly. His cardiac failure resolved without specific treatment and his blood pressure remained stable around 120/70 mm Hg. Urinary vanillylmandelic acid concentrations were consistently and urinary catecholamine concentrations intermittently raised. Right adrenal angiography showed a round mass in the gland. After preoperative treatment with phenoxybenzamine followed by propranolol a large, partly haemorrhagic phaeochromocytoma was removed. Subsequently he remained normotensive and urinary catecholamine and vanillylmandelic acid concentrations were normal.

Case 2—A 32-year-old woman had had pre-eclampsia when aged 22 and a left nephrectomy for hydronephrosis when aged 27. At that time her blood

pressure was 145/105 mm Hg. One year later she complained of episodic headaches, chest pain, palpitations, and blanching of the hands. Urinary catecholamine and vanillylmandelic acid concentrations were normal. For the next three years she had recurrent abdominal pains and episodes of anxiety associated with pallor. In January 1977 she was given propranolol 40 mg twice daily for hypertension. Several weeks later she attended another hospital with acute pulmonary oedema. Her presenting blood pressure was 130/90 mm Hg. She also had symptoms similar to the previous paroxysms. Urinary catecholamine concentrations were high.

When first seen at the Royal Melbourne Hospital, her blood pressure was 110/80 mm Hg. This rose to 190/130 mm Hg on firm palpation of the right loin. She was placed on both alpha- and beta-adrenergic blocking agents. Arteriography showed a rounded mass medial to the right kidney. At exploration a pheochromocytoma 5 cm in diameter was excised. Although her blood pressure was unstable intraoperatively her postoperative course was relatively uneventful. She has since remained normotensive on no medication.

Comment

The pulmonary oedema in these patients shortly after starting treatment with propranolol may have resulted from interruption of the beta-adrenergic drive to the heart associated with vasoconstriction mediated by unopposed alpha-adrenergic stimulation. The ECG and enzyme evidence in case 1 suggests that myocardial necrosis, a recognised complication of catecholamine excess, may have contributed to the acute pulmonary oedema. Neither patient's blood pressure was raised when they reached hospital, although this may have reflected their impaired cardiac output. While a causal relationship between beta-adrenergic blockade and precipitation of pulmonary oedema cannot be proved in either case, the sequence of events is suggestive in both. The events described support the contention that beta-adrenergic blockers should not be used before alpha-adrenergic blockade in patients suspected of having pheochromocytomas.

¹ Melman, K L, in *Textbook of Endocrinology*, ed R H Williams, 5th edn, p 313. Philadelphia, W B Saunders and Co, 1974.

² Thomas, D J B, et al, *British Medical Journal*, 1977, **1**, 688.

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Department of Endocrinology, Royal Melbourne Hospital, Melbourne, Australia

J D WARK, MB, BS, research fellow in endocrinology

R G LARKINS, PHD, FRACP, physician to endocrine laboratory

Rheumatoid arthritis: C-reactive protein and erythrocyte sedimentation rate during initial treatment

Patients with rheumatoid arthritis (RA) are generally treated first with aspirin or other non-steroid anti-inflammatory drugs. The usual view, to which we subscribed when suggesting a systematic approach to treatment,¹ is that many patients will not need additional drug treatment and that a period of observation will identify them. We now re-examine this view.

Patients, methods, and results

There were 76 patients, 52 women and 24 men, with definite or classical RA (American Rheumatism Association criteria). Their mean age was 55.2 and mean duration of RA 5.6 years; 57 had positive Rose-Waaler tests. They were all newly referred patients who were being treated with aspirin, indomethacin, or ibuprofen. No patient had had gold, corticosteroids, penicillamine, antimalarial, or cytotoxic drugs. Serum C-reactive protein concentration (CRP) was measured by radial immunodiffusion using commercially available standards (Hoechst), which gives values about half those previously reported by us.² Erythrocyte sedimentation rate (ESR) was measured by the Westergren method. Student's *t* tests for paired variants were applied to the results.

At the first consultation serum CRP ranged from 0-328 mg/l, mean 63 mg/l; ESR from 5-161 mm in 1 h, mean 57.7 mm in 1 h. After 12 weeks

Relation between serum CRP at first attendance and 12 weeks later. Hatched area denotes values falling to <20 mg/l.

mean serum CRP had fallen by 8.5 ± 5.0 mg/l and mean ESR by 3.8 ± 2.7 mm in 1 h. The differences were not significant. The differences between the initial values for serum CRP and values at 12 weeks in individual patients show (fig) that when CRP was initially above 100 mg/l it had usually fallen; with lower initial values serum CRP rose or fell in roughly equal numbers of patients. There were only 10 patients whose serum CRP fell from above 20 mg/l to that figure or below. The results for ESR were similar, though the tendency for higher initial values to fall was less obvious than with CRP. There were only eight patients whose ESR fell from above 30 mm in 1 h to below that figure.

Comment

In 76 patients with RA, probably representative of any group of newly referred patients being treated with aspirin-type drugs only, there were no significant changes in mean serum CRP or ESR in the first 12 weeks. The result was unexpected. Although neither aspirin nor indomethacin influence these measurements³ we had assumed that spontaneous remissions in a proportion of patients, perhaps one-third, would have a demonstrable effect on the group as a whole. If we are correct in asserting that serum CRP and ESR levels reflect the progress of RA,² and that values for serum CRP above 20 mg/l or for ESR above 30 mm in 1 h indicate detectable radiographic deterioration,⁴ we must assume that most of the patients referred to us had progressive disease when first seen and that the position had not significantly altered after 12 weeks. Thus the policy of using aspirin or other non-steroid anti-inflammatory drugs to relieve symptoms and allowing three months to observe progress did identify about 10% of patients satisfactorily controlled. For most of them, however, it only unnecessarily delayed consideration of more active treatment. We suggest that treatment with drugs such as gold, capable of lowering acute-phase protein levels⁵ and thus likely to alter the course of the disease by reducing the rate of radiological progression, should usually be begun soon after the first consultation if serum CRP and ESR values are substantially raised.

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¹ Constable, T J, et al, *Lancet*, 1975, **1**, 1176.

² McConkey, B, Crockson, R A, and Crockson, A P, *Quarterly Journal of Medicine*, 1972, **41**, 115.

³ McConkey, B, et al, *Quarterly Journal of Medicine*, 1973, **42**, 785.

⁴ Amos, R A, et al, *British Medical Journal*, 1977, **1**, 195.

⁵ McConkey, B, et al, *Rheumatology and Rehabilitation*, 1976, **15**, 230.

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Dudley Road Hospital, Birmingham, and Department of Experimental Pathology, University of Birmingham

R S AMOS, BSC, MRCP, Sheldon clinical research fellow

R A CROCKSON, FIMLS, technical superintendent

A P CROCKSON, FIMLS, senior chief technician

L WALSH, chief technician

B McCONKEY, DM, FRCP, consultant physician