Coeliac artery compression syndrome: can we believe our ears?

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SUMMARY. Two patients with coeliac artery compression syndrome' are described.

Symptoms were vague and nonspecific, and the diagnosis was made by finding an epigastric bruit on auscultation of the abdomen and confirming the presence of a vascular lesion by angiography. Both patients were cured by surgical decompression of the coeliac trunk. Auscultation of the abdomen can be a useful part of physical examination in general practice.

Introduction

COELIAC artery compression syndrome refers to the association of rather vague and nonspecific abdominal symptoms, which may be severe, with the presence of proven compression of the coeliac axis. This compression is usually caused by the median arcuate ligament of the diaphragm but can also be caused by strands of the coeliac plexus.

There is no consensus in favour of the existence of such a syndrome, as the generally held view is that the equivalent of more than one of the visceral arteries must be occluded before ischaemia of the gut may be produced. There is therefore some controversy about the idea that compression of a single vessel, such as the coeliac axis, may produce symptoms in an otherwise healthy person.

I wish to record two cases where bizarre and even disabling symptoms were associated with an epigastric bruit as the only positive physical finding. Both patients underwent angiography, which demonstrated compression of the coeliac artery, and subsequent surgical decompression with complete relief of all symptoms.

This paper is also designed to encourage physicians to auscultate the abdomen as part of a physical examination.

Case reports

First patient

Mrs A. was seen in August 1974. She complained of a sensation in the epigastrium, variously described as "gurgling" and "buzzing", present for several months, and aching mid-epigastric pain radiating across the right lower chest. The mid-epigastric pain was intermittent and was noticeably worse when she lay on her righthand side. In addition, she had lost 4·5 kg (10 lbs) in weight in the previous four months and had short periods of diarrhoea several times a month during that time. During these episodes she would have up to 15 loose watery stools daily. An x-ray series of investigations in April showed a pattern of malabsorption.

Pertinent medical history showed that she was allergic to milk and had had her gallbladder removed four years previously. There was a family history of thyrotoxicosis and pernicious anaemia.

Physical examination was unremarkable except that the patient looked rather frail for her 39 years and a loud epigastric systolic bruit, grade 4/5, was audible on auscultation of the abdomen in the supine position.

The patient was referred to a cardiovascular surgeon for consultation and subsequently had angiography performed using retrograde femoral technique. This showed significant narrowing of the coeliac artery at its origin. The area of stenosis was 1·2 cm long and the lumen was 4 mm in diameter.

A diagnosis of coeliac artery compression syndrome was made, but before surgical decompression could be arranged, the patient developed a relatively acute attack of chest pain and an ECG showed left bundle branch block. A normal ECG had been recorded before her cholecystectomy in 1970. She was referred for specialist cardiological assessment and a diagnosis of healed cardiomyopathy was made. At this time arrangements were made for surgical treatment of the coeliac artery compression and at operation a significant band about the coeliac artery was divided and a coeliac ganglion obliterated.

Postoperatively, the patient continued to have

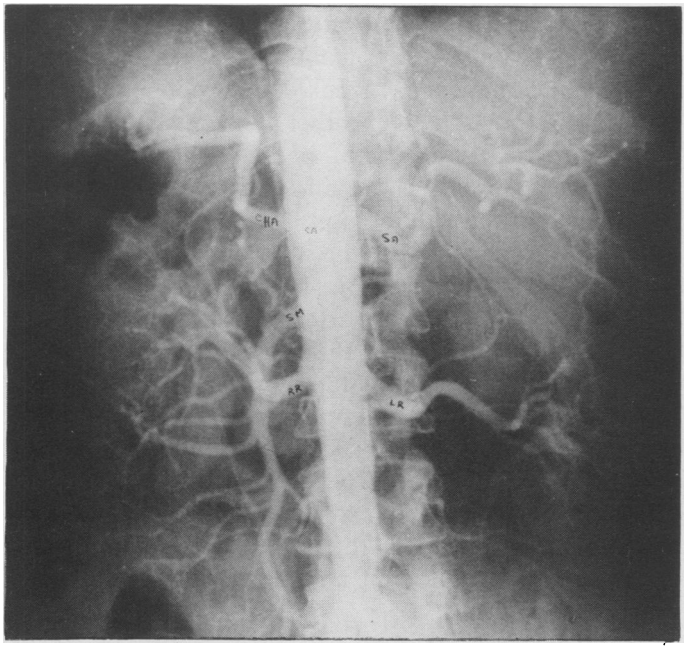

Figure 1. *Anterior/posterior view; retrograde femoral aortogram. SA = splenic artery; LR = left renal artery; RR = right renal artery; SM = superior mesenteric artery; CHA = common hepatic artery; CA = coeliac artery.*

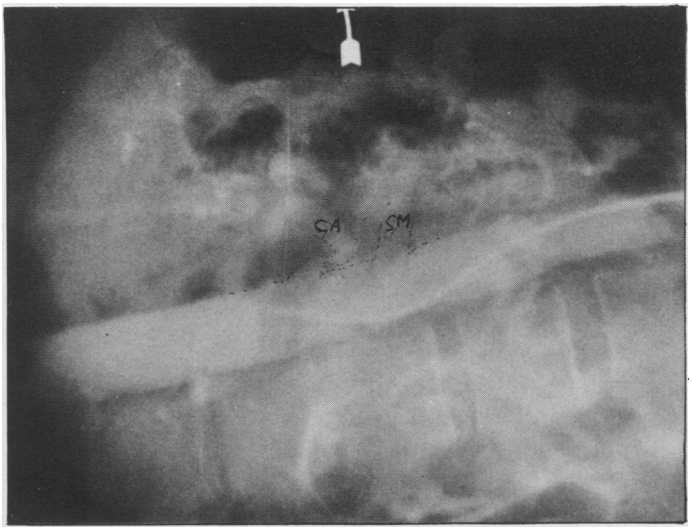

Figure 2. *Lateral view; retrograde femoral aortogram showing aorta at level of coeliac artery origin with tip of catheter visible proximal to coeliac origin. The indentation on the superior aspect of the coeliac artery has been outlined for clarity. CA = coeliac artery; SM = superior mesenteric artery.*

troublesome diarrhoea and, after experimenting with various medications, put herself on a gluten-free diet, with rapid resolution of the problem.

By September 1976 the patient was able to say that she felt 100 per cent fit and was swimming four times per week without distress. She was also able to eat a normal diet without difficulty.

Second patient

The second patient, Mrs B., was seen in January 1976. She was 44 years old and had a 12-year history of chronic abdominal pain. The nature of the pain had remained constant during that time. She had undergone partial gastrectomy in 1966, because of a peptic ulcer, and following this procedure the pain went away for several months, only to return. The pain was described as "crampy", it was felt in the epigastrium, and it came on soon after eating. The larger the meal, the more severe the pain. The pain was relieved by vomiting. The patient also had intermittent bouts of diarrhoea mixed with constipation. She had found that she lost weight if she did not eat frequent small meals. The pain was sufficiently severe that, when first seen, she was taking antidepressants, anticholinergics, analgesics, and barbiturates.

The only positive physical findings were that she was a miserable-looking person who had a loud epigastric bruit, grade 4/5, audible on auscultation of the abdomen in the supine position. She had severe social problems related to an alcoholic husband and was chronically depressed.

A consultation was obtained with a vascular surgeon, who felt that her symptoms warranted investigation by angiography. Selective coeliac artery angiogram was carried out by retrograde femoral technique and the result are shown. Figure 1 demonstrates the aorta and its branches at the level of the coeliac origin and Figure 2 shows a notch on the superior margin of the coeliac artery approximately 5 mm distal to its origin.

Surgical treatment was not offered to this patient immediately because of her chronic anxiety state and the difficulty of determining the full extent of the psychological component of her distress. She was given antacids, anticholinergics, and a peptic ulcer diet combined with psychotherapy and suitable psychotropic drugs. This was all to no avail and the decision to treat the coeliac compression was made. At operation, the median arcuate ligament of the diaphragm was found to be compressing the superior aspect of the coeliac artery and this was incised.

The postoperative course was stormy with the development of a wound infection and marked depression and anxiety. However, gradual improvement took place, the wound healed, the psychological disturbance settled down, and the patient recently described herself as "feeling better than she has done for years". Her social problems remain, she is still liable to periods of tension and depression, but she has no abdominal pain whatsoever and eats a full diet without discomfort. She has gained weight and she finds that she is better equipped to cope with her difficulties now that she does not have her stomach problems.

Discussion

Coeliac artery stenosis, as a cause of epigastric

murmur, was first described by Marable and his colleagues (1966) and in 1968 Marable and other colleagues described a series of patients, all with epigastric bruits, whom they considered to have symptoms caused by this stenosis. Other authorities (Snyder *et al.*, 1967; Lord *et al.*, 1968) took the same view.

Ranged against them are the views of those who maintain that more than one visceral artery must be blocked before ischaemia will develop (Rob, 1967; Dick *et al.*, 1967), and those who have shown that the typical appearance of coeliac artery stenosis on angiography may be found in people who are symptom-free (Sutton, 1967; Drapanas and Bron, 1966).

Also, the fact that the symptoms are rather vague and sound psychogenic no doubt influences these judgements.

The syndrome has been summarized (Edwards *et al.*, 1970) and consists of the following symptoms and signs:

Symptoms

1. Epigastric pain or discomfort related more to posture and activity than to food.
2. Symptoms often relieved by recumbency.
3. Psychological element often suspected.

Signs

1. Predominant in young women.
2. Usually affects those leptosomatic in habitus.
3. There is an epigastric bruit which is altered by position and respiration.
4. Lateral aortography shows anterior compression of the coeliac axis.

Using these criteria, the patients described above would not have been considered for arteriography, and I suppose this reflects the enigmatic nature of the syndrome.

Other work on this subject has concentrated on studying the bruit itself to determine whether the quality of the bruit has any particular significance. Watson and fellow workers (1973) studied a group of psychiatric patients in 1970 and found the total prevalence of an epigastric bruit to be 31 per cent. They also found that when they extended their study to include a number of patients referred for gastroenterological consultation, the prevalence fell to 27 per cent. They concluded that the bruit was more likely to be of significance if it was loud, began in early or mid-systole and lasted into early diastole. They gave the mean maximum duration of the murmur heard in patients with coeliac artery compression as 0·5 seconds.

In the face of these conflicting opinions, it seems to be eminently reasonable to conclude that, for some reason as yet unknown, compression of the coeliac artery may cause symptoms in some people and not in others. Perhaps the difference lies in the amount of ganglion material involved in the compression, or perhaps it depends on the amount of movement of the coeliac trunk on inspiration and expiration. Perhaps, in some people, the coeliac trunk comes off the aorta at a more acute angle than in others. There is little doubt that there is a category of patient who has chronic abdominal pain, in whom an epigastric bruit may be found, in whom arteriography will show compression of the coeliac artery, and who will obtain dramatic relief from surgical decompression. The problem is to identify such patients in whom this arteriography is justified and then to make a decision on the merits of surgery.

My own view is that as family physicians we should be listening for abdominal bruits every time we examine an abdomen, and attempt long-term follow-up of any person who has a bruit. There is probably more useful information to be obtained about the significance of such a bruit by observing the progress, over many years, of patients who have an audible epigastric bruit but have no symptoms.

Since seeing these patients and becoming aware of this problem, I have been listening for abdominal bruits as a matter of routine in all abdominal examinations. My conclusions have not been recorded in any systematic fashion, but I am in no doubt that a high percentage of well people have an audible epigastric bruit if the abdomen is auscultated in the supine position using a conventional stethoscope. The bruit is most often heard in slim young females and can be accentuated by pressure from the stethoscope. The murmur is always systolic and is best heard in expiration. It is sharply localized and is best heard in the midline, 3 cm below the xyphoid process.

Particularly in all cases of obscure abdominal pain, auscultation of the abdomen should be carried out and emphasis placed on the value of an epigastric bruit as a diagnostic sign.

References

Dick, A. P., Graff, R., Gregg, D. M., Peters, N. & Sarner, M. (1967). *Gut*, **8**, 206-220.

Drapanas, T. & Bron, K. M. (1966). *Annals of Surgery*, **164**, 1085-1088.

Edwards, A. J., Hamilton, J. D., Nichol, W. D., Taylor, G. W. & Dawson, A. M. (1970) *British Medical Journal*, **1**, 342-345.

Lord, R. S., Stoney, R. J. & Wylie, E. J. (1968). *Lancet*, **ii**, 795-798.

Marable, S. A., Kaplan, M. F., Beman, F. M. *et al.* (1968). *American Journal of Surgery*, **115**, 97-102.

Marable, S. A., Molnar, W. & Beman, F. M. (1966). *American Journal of Surgery*, **111**, 493-495.

Rob, C. (1967). *American Journal of Surgery*, **114**, 363-367.

Snyder, M. A., Mahoney, E. B. & Rob, C. G. (1967). *Surgery*, **61**, 372-376.

Sutton, R. A. (1967). *Proceedings of the Royal Society of Medicine*, **60**, 139-141.

Watson, W. C., Williams, P. B. & Duffy, G. (1973). *Annals of Internal Medicine*, **79**, 211-215.

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