Archives of Disease in Childhood, 1988, **63**, 1457–1460

Chest pain of gastrointestinal origin

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SUMMARY Twenty seven children who had been diagnosed as having idiopathic chest pain were investigated to find out if the pain was of gastrointestinal origin. The symptoms had lasted from two weeks to eight months. In 21 of the 27 children (78%) the chest pain had a gastrointestinal cause: 16 had oesophagitis, four had gastritis, and one had diffuse oesophageal spasm. All patients responded to medical treatment of their gastrointestinal symptoms, resulting in disappearance of the chest pain.

Although chest pain is not usually associated with serious disease in children, if it occurs it is a cause of great anxiety for the patient and family. In one study 16% of children with chest pain made more than one visit to the accident and emergency department.[1]

Gastrointestinal disorders have not been described as being important causes of chest pain in children.[1–3] To further our understanding of the association between gastrointestinal dysfunction and chest pain in children, we carried out oesophageal manometry, oesophagogastroduodenoscopy, and Bernstein acid perfusion tests on 27 children with idiopathic chest pain, and extended ambulatory oesophageal pH monitoring on eight of the 27 patients.

Patients and methods

Fifty one patients between 8 and 20 years of age who complained of chest pain were investigated. All patients were initially examined by general paediatricians and the diagnoses were: idiopathic chest pain (n=27), musculoskeletal disorders (n=12), costochrondritis (n=9), and oesophagitis (n=3). Previously established criteria were used to make these diagnoses.[2]

The 27 children in whom a diagnosis of idiopathic chest pain had been made were further investigated by the division of paediatric gastroenterology at New York Medical College. The three patients with symptoms of oesophagitis were not included in the study. None of the patients had the characteristic symptoms of gastro-oesophageal reflux such as heartburn, regurgitation, pain on swallowing, dysphagia, haemorrhage, or 'water brash'. The chest pain was usually localised to the left praecordium, the central or retrosternal areas, or both, and episodes of pain lasted from less than a minute to several hours (table 1).

In order to determine the cause of the pain, each patient was investigated by oesophagogastroduodenoscopy, oesophageal manometry, and a Bernstein acid perfusion test. In addition, eight patients had extended ambulatory pH monitoring.

Oesophageal manometry was carried out with a triple lumen polyvinyl catheter assembly (individual outside diameter 1·2 mm) with three pressure sensing orifices separated by 5 mm. The catheter

Table 1 *Details of patients with chest pain of gastrointestinal origin*

	No (%) of patients
Age at onset (years):	
0–7	0 (0)
8–12	6 (28)
13–17	11 (52)
17–20	4 (19)
Duration of symptoms (months):	
<1	3 (14)
1–6	15 (72)
>6	3 (14)
Site of pain:	
Left praecordium	4 (19)
Central or retrosternal	4 (19)
Right praecordium	1 (4)
Combination (left praecordium and central or retrosternal	12 (57)
Duration of attacks (minutes):	
<1	4 (19)
1–5	8 (38)
6–60	6 (28)
>60	3 (14)

was perfused with sterile water at 0·5 ml/minute by a low compliance pneumatic hydraulic capillary infusion system with a pressure response of >106·8 kPa/second. Pressures were transmitted through the fluid filled catheters to transducers connected to a three channel polygraph recording system. The station pullthrough technique was used to determine lower oesophageal sphincter location and pressure. The normal range of lower oesophageal sphincter pressure in our laboratory is 2·0 to 4·0 kPa. To measure oesophageal peristalsis, 10 swallows of 5 ml water were given at 30 second intervals.

Abnormal oesophageal contractions were defined as: non-peristaltic contractions including simultaneous onset or the peaks of waves recorded at adjacent recording sites, and non-conducted swallows that caused no oesophageal pressure wave, or a wave too weak to be transmitted to the three recording sites; or repetitive contractions with the second and third peaks of more than 10% of the primary wave amplitude; or spontaneous contractions of more than 4·00 kPa that were not generated by swallows.[4] Wave amplitude expressed in kPa was measured from the mean intraoesophageal baseline pressure to the peak of the wave. Individual wave duration in seconds was measured from the onset of the principal upstroke to the end of the wave.

The Bernstein acid perfusion test was carried out by positioning the catheter in the upper third of the oesophagus. An infusion of 0·9% sodium chloride was given at a rate of 100 drops/minute for 10 minutes. This was followed by infusion of 0·1 mol/l hydrochloric acid for up to 10 minutes. The test was negative if the patient remained asymptomatic. If severe discomfort was produced in the chest, the acid perfusion was stopped and saline reinstilled. Repeated elicitation of chest pain by acid perfusion followed by relief of symptoms on perfusion with saline indicated that the symptoms were due to the acid challenge.

Oesophageal pH monitoring was carried out by placing a pH monitoring probe in the mid-oesophagus after localisation of the lower oesophageal sphincter by oesophageal manometry.[5] Ambulatory pH monitoring was usually continued for 18 hours after placement of the probe.

Appreciable gastro-oesophageal reflux was diagnosed if there was a pH of less than 4 for 10% or more of the first two postprandial hours,[6] or if the mean duration of reflux during sleep was greater than four minutes more than two hours after feeding.[7]

Oesophagogastroduodenoscopy was carried out with an Olympus GIFXP10 gastroscope after sedation with meperidine and diazepam. Oesophagitis was diagnosed when the mucosa lost its normal vascular pattern and there was spontaneous friability, ulceration, or erythema. Gastritis was diagnosed when there was erythema, erosions, or bleeding. Oesophagitis[8 9] and gastritis[10] were graded histologically according to established criteria.

The protocol was approved by the office of research administration of New York Medical College. Informed consent was obtained from the legal guardian of each patient.

Results

In 21 of the 27 patients a gastrointestinal cause was found for the chest pain. Sixteen of the 21 (76%) had oesophagitis and four (19%) had gastritis diagnosed both macroscopically and histologically. In one patient endoscopy was normal but there was diffuse oesophageal spasm on oesophageal manometry (table 2). Five patients with oesophagitis (31%) had abnormalities of oesophageal motility. Nine of the 16 with oesophagitis (56%) had lower oesophageal sphincter pressure that were significantly less (mean 9·44, SEM 0·75) than the mean of the group of patients with gastrointestinal chest pain (mean (SEM) 16·43 (1·54), $p<0·001$). The four patients with gastritis had a mean lower oesophageal sphincter pressure significantly higher (25·00 (1·41), $p<0·01$) than the group of patients with gastrointestinal chest pain. Eight patients with oesophagitis underwent prolonged oesophageal pH monitoring and all had appreciable gastro-oesophageal reflux. Nine patients with oesophagitis had positive Bernstein acid perfusion tests (table 3).

The patients with oesophagitis and gastritis had marked decreases in the severity of their chest pain within three weeks of starting activated dimethicone 0·5 mg/kg (maximum 30 ml/dose) one and three hours after meals, or ranitidine (150 mg twice daily) (table 4). Follow up endoscopies after two months of treatment showed that the oesophagitis and gastritis had resolved. The patient with diffuse oesophageal spasm had appreciably less chest pain after treatment with dicyclomine hydrochloride; permission to carry out repeat manometry, however, was refused (table 4).

Table 2 *Aetiology of chest pain of gastrointestinal origin*

	No (%) of patients
Oesophagitis	16 (76)
Gastritis	4 (19)
Diffuse oesophageal spasm	1 (5)

Table 3 *Results in 21 patients with chest pain of gastrointestinal origin*

Case No	*Appearance at endoscopy*	*Lower oesophageal sphincter pressure (kPa)*	*Oesophageal motility*	*Bernstein acid perfusion test*	*Prolonged oesophageal pH monitoring*
1	Gastritis	2·93	Normal	Negetive	No
2	Oesophagitis	1·33	Normal	Negative	No
3	Oesophagitis	1·20	Normal	Negative	No
4	Oesophagitis	2·40	Normal	Negative	No
5	Oesophagitis	3·47	Normal	Positive	Yes
6	Oesophagitis	0·933	Normal	Positive	Yes
7	Gastritis	3·33	Normal	Negative	No
8	Gastritis	3·73	Normal	Negative	No
9	Oesophagitis	1·60	Simultaneous contractions	Negative	No
10	Gastritis	3·33	Normal	Negative	No
11	Oesophagitis	2·93	Normal	Negative	Yes
12	Oesophagitis	2·67	Normal	Positive	Yes
13	Oesophagitis	1·07	Repetitive contractions	Positive	Yes
14	Normal	2·13	Diffuse oesophageal spasm	Negative	No
15	Oesophagitis	0·800	Simultaneous contractions	Positive	Yes
16	Oesophagitis	1·33	Normal	Positive	Yes
17	Oesophagitis	1·47	Normal	Negative	Yes
18	Oesophagitis	2·53	Normal	Positive	No
19	Oesophagitis	2·40	Simultaneous contractions	Positive	No
20	Oesophagitis	2·80	Simultaneous contractions	Positive	No
21	Oesophagitis	1·60	Normal	Negative	No

Table 4 *Treatment of chest pain of gastrointestinal origin*

	Activated dimethicone No (%)	*Ranitidine No (%)*	*Dicyclomine No (%)*
Oesophagitis	10 (62)	6 (38)	0
Gastritis	1 (25)	3 (75)	0
Diffuse oesophageal spasm	0	0	1 (100)

Discussion

Chest pain in children is usually described as idiopathic.[1–3] Gastrointestinal causes are rarely diagnosed, but systematic investigation of these children for gastrointestinal disease is not usually carried out. Studies in adults indicate that over half of the patients with angina like chest pain and normal coronary arteriograms are found to have oesophageal disorders.[11] The aetiology of such chest pain can be difficult to determine, because the heart and oesophagus have similar neural pain pathways. Both the cardiac plexus and the oesophageal plexus arise from the vagus nerves and sympathetic trunks.[11] We therefore performed gastrointestinal investigations in 27 children with 'idiopathic' chest pain. Twenty one of the children had gastrointestinal causes for their complaints. Abnormal oesophageal motility, oesophagitis, and gastritis were among the causes of non-specific chest pain.

Normal oesophageal motor activity consists of an orderly peristaltic contraction that passes through the oesophagus resulting in opening, and subsequent closing of the lower oesophageal sphincter. This progressive propulsive contraction of the oesophagus occurs in response to swallowing (primary peristalsis) or distension (secondary peristalsis).[12] Abnormalities of oesophageal peristalsis have been shown to produce symptoms of chest pain; one patient had difuse oesophageal spasm, which is particularly associated with chest pain.[13] It is characterised by simultaneous contractions after at least 10% of swallows of liquid during oesophageal motility testing, intermixed with normal peristaltic contractions.[13] Other abnormalities of peristaltic contractions are also associated with chest pain.[14]

Oesophagitis was the principal cause of chest pain in our patients. The most common clinical sign is heartburn, which is usually described as an uncom-

fortable burning sensation located below the sternum. Chest pain from oesophagitis, however, may be non-specific.[15] None of our patients with idiopathic chest pain had any of the characteristic symptoms of oesophagitis, and the diagnosis was made both endoscopically and on histological examination. Of the 16 patients with oesophagitis, five had associated abnormalities of oesophageal motility. Eight children with oesophagitis had prolonged oesophageal pH monitoring, which showed appreciable gastro-oesophageal reflux in each patient. All patients responded clinically to antacids or H_2 receptor antagonists. Follow up endoscopies carried out two months after the start of treatment were normal, indicating resolution of the oesophagitis.

Gastritis and peptic ulcer disease may also present as chest pain.[15] Four of the patients studied had both endoscopic and histological evidence of gastritis. *Campylobacter pylori* has recently been described as a cause of gastritis.[16] Future investigations of idiopathic chest pain in children should therefore include biopsy and culture for this bacterium. All our patients responded to treatment with an antacid or H_2 receptor antagonist, and so it is unlikely that *C pylori* was a cause. Repeat endoscopies after two months of treatment were also normal.

Our experience indicates that gastrointestinal causes of unexplained chest pain in children should be investigated. The symptoms of gastrointestinally induced chest pain are often non-specific, and can be determined with certainty only by oesophageal manometry and fibreoptic endoscopy. A patient with idiopathic chest pain may benefit from a therapeutic trial of an antacid before extensive gastrointestinal investigation is instigated.

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Accepted 17 June 1988