Esophageal Disease as a Cause of Severe Retrosternal Chest Pain*

Robert Roberts, M.D., Robert D. Henderson, M.D., and E. Douglas Wigle, M.D.

During one six-month period 11 patients were referred with a diagnosis of coronary artery disease, because of recurrent episodes of severe, prolonged retrosternal chest pain necessitating from one to seven hospital admissions per patient for "suspect myocardial infarction." In no instance was this diagnosis proved by electrocardiogram or serum enzyme changes, but 7 of the 11 patients had abnormal resting electrocardiograms. Selective coronary arteriograms were normal in 10 patients and revealed nonobstructive coronary artery disease in the 11th patient. Esophageal studies revealed hiatus hernia in 9 and mild to severe disordered motored activity of the esophagus in all 11. Acid perfusion into the esophagus reproduced the chest pain in nine patients and in the other two, the hiatus hernia was incarcerated. On direct questioning, all patients indicated that the pain was worsened by lying down and bending over, and in eight patients there was a history of pharyngoesophageal or gastroesophageal dysphagia. In this day when the problem of chest pain with normal coronary arteries is very topical, our report emphasizes the need to consider symptomatic esophageal disease in the differential diagnosis of this problem.

Esophageal disease has long been known to cause severe retrosternal pain and to be an important consideration in the differential diagnosis of this symptom.1 During the six-month period January to June 1971, 11 patients were referred to the Cardiovascular Unit, Toronto General Hospital with a diagnosis of coronary artery disease, based on a history of one or more episodes of prolonged severe retrosternal chest pain. Each patient had been admitted to other hospitals from one to seven times for suspected myocardial infarction. In the investigation of these patients, selective coronary cineangiograms were normal in 10 and revealed minor nonobstructive lesions in the 11th patient. Esophageal studies (barium swallow, motility studies and acid perfusion into the esophagus) revealed a hiatus hernia, moderate-to-severe disordered motor activity of the esophagus, or both, in every patient. Acid perfusion into the esophagus reproduced the chest pain complaint in nine patients, and the hiatus hernia was incarcerated in the other two patients. Six patients underwent transthoracic repair of their hiatus hernia and five received medical therapy.

On reviewing the English literature, we were unable to find a similar series in which the patient underwent both selective coronary cineangiography and detailed esophageal investigation. In this era in which ischemic chest pain with normal coronary arteriograms is becoming an important problem,2,4 it seems appropriate to reemphasize esophageal disease as a cause of severe retrosternal pain, possibly leading to a mistaken diagnosis of myocardial infarction.

PATIENTS STUDIED

The study involved 11 patients referred to our cardiovascular unit for assessment of coronary artery disease between January and June 1971. The group consisted of seven women and four men ranging in age from 41 to 57 years, with average an age of 50 years. Each patient was questioned independently and examined by all of us. Most patients had been seen by at least two physicians prior to referral.

METHODS

Investigation included a complete history and physical examination, routine blood studies and serum electrolyte determinations, urinalysis, glucose tolerance test, measurement of serum cholesterol and triglyceride levels and a lipoprotein electrophoresis, to ascertain if any of these were abnormal. Each patient also had a resting and exercise electrocardiogram as well as a chest x-ray film, cervical and thoracic spine x-ray films and an oral cholecystogram.

Electrocardiograms were obtained after exercise by having the patients do a double Master two-step, with ECG's recorded immediately and at three, six and ten minutes afterward. The appearance of 0.5 mm or more of flat ST segment depression was judged a positive response.

*From the Divisions of Cardiology and Thoracic Surgery, and the Departments of Medicine and Surgery, Toronto General Hospital and the University of Toronto, Toronto, Ontario. Supported in part by the Ontario Heart Foundation.

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Reprint requests: Dr. Wigle, Toronto General Hospital, Toronto, Ontario, Canada
Table 1—Symptoms in 11 Patients with Retrosternal Pain Due to Esophageal Disease*

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Patients with Symptom, No.</th>
</tr>
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<tbody>
<tr>
<td>Nature of Chest Pain</td>
<td></td>
</tr>
<tr>
<td>Location: retrosternal</td>
<td>11</td>
</tr>
<tr>
<td>Quality: tight, gripping</td>
<td>9</td>
</tr>
<tr>
<td>Radiation: one or both arms</td>
<td>7</td>
</tr>
<tr>
<td>back</td>
<td>5</td>
</tr>
<tr>
<td>epigastrium</td>
<td>2</td>
</tr>
<tr>
<td>Precipitating Factors:</td>
<td></td>
</tr>
<tr>
<td>Postural movements</td>
<td>11</td>
</tr>
<tr>
<td>Exertion</td>
<td>7</td>
</tr>
<tr>
<td>Recumbent position (night)</td>
<td>6</td>
</tr>
<tr>
<td>Eating</td>
<td>3</td>
</tr>
<tr>
<td>Pain Relieved by</td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>8</td>
</tr>
<tr>
<td>Antacids</td>
<td>8</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>7</td>
</tr>
<tr>
<td>Other Symptoms</td>
<td></td>
</tr>
<tr>
<td>Dysphagia</td>
<td>8</td>
</tr>
<tr>
<td>Heartburn</td>
<td>8</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td>5</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>4</td>
</tr>
<tr>
<td>Water brash</td>
<td>3</td>
</tr>
</tbody>
</table>

*All patients had previously been admitted to the hospital and treated as “suspect myocardial infarction” because of prolonged retrosternal pain.

Cardiac investigation included right and left heart catheterization, left ventricular cineangiocardiograph and selective coronary cineangiography performed by the Judkins technique. Cardiac output was determined by the standard Fick method.

Esophageal motility studies were made, using three PE 190 tubes with side openings 5 cm and 2.5 cm apart. The tubes were constantly perfused with water, using a modified 2202 Harvard pump at a flow rate of 3.6 ml per minute per tube. Statham P23 Db strain gauges were used as sensing devices, with a Honeywell 1508 ultraviolet visicorder as the recording device.

Each subject had a standard motility study, by placing the tubes in the stomach, then withdrawing the tubes at 0.5 cm-intervals through the esophagus.

Following the motility study, 0.1 normal hydrochloric acid was perfused at 3.6 ml/min, with constant pH monitoring. This was continued for a period of 30 minutes. At intervals, acid perfusion was stopped and the pH altered to near normal by water perfusion. Also at intervals a 30-ml bolus of water or of 0.1 normal hydrochloric acid was injected. The subjects’ pain response and the manometric trace were constantly monitored during this period. The subject at no time was aware of the solutions being infused. If the patient complained of pain similar in nature and radiation to the presenting pain, the response to acid perfusion was considered positive.

RESULTS

Table 1 lists the characteristics of the chest pain and other symptoms. All patients had previously been admitted to various hospitals from one to seven times with a suspected diagnosis of myocardial infarction because of prolonged retrosternal pain. In no case was the diagnosis confirmed by pathologic Q waves in the electrocardiogram or elevation of serum enzyme levels. The pain was typically described as “tight or gripping” and radiated to one or both arms in seven patients, to the back in five patients and to the epigastrium in two. On direct questioning, there was a history of precipitation of chest pain by lying down or bending over in all patients, and in seven the pain at times could be provoked with exertion and relieved by rest. In six patients the pain occurred at night in the recumbent position. Eating produced the pain in only three patients. The pain was relieved by taking antacids in eight patients and by sublingual nitroglycerin therapy in seven. In five patients the relief of pain by use of nitroglycerin was not immediate, but occurred within 10 to 20 minutes.

Eight patients complained of either pharyngoesophageal or gastroesophageal dysphagia or heartburn. Nausea and vomiting, dyspnea or water brash occurred less frequently.

Physical examination was essentially normal in all cases. Results of routine blood determinations were normal; no patient had diabetes, but one patient had type-4 hyperlipoproteinemia.

Eight of the 11 patients had abnormal resting electrocardiograms. Nonpathologic Q waves (less than 0.04 second in duration) were present in leads 2, 3, and aVF in two patients. Six patients had nonspecific ST-T changes. Electrocardiograms showed a normal response after exercise in all cases.

Right and left heart hemodynamics were normal in all patients. Selective coronary cineangiograms were completely normal in 10 patients and revealed nonobstructive disease in the 11th patient. In this

Table 2—Results of Esophageal Investigation in 11 Patients with Retrosternal Pain Due to Esophageal Disease

<table>
<thead>
<tr>
<th>Radiologic Evidence of Hiatus Hernia</th>
<th>Disordered Motor Activity of Esophagus</th>
<th>Gastroesophageal Sphincter Tone</th>
<th>Acid Perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, No.</td>
<td>Present 9</td>
<td>Absent 2</td>
<td>Mild 2</td>
</tr>
</tbody>
</table>

*Mild disordered motor activity = 0-30% of the waves are disordered

Moderate """" = 30-60% """" """" """" """" ""

Severe """""" = 60-90% """" """" """" """"
Important to exclude noncardiac causes of chest pain that present with "anginalike pain" and normal coronary cineangiograms. Several patients have been treated surgically or medically for symptoms thought to be of esophageal origin, but a definitive diagnosis has not been made.

The results of the special esophageal studies are summarized in Table 2. Nine of the 11 patients had radiologic evidence of a hiatal hernia; all had mild- to-severe disordered motor activity of the esophagus and 2 patients had reduced gastroesophageal junctional tone. The disordered motor activity in all cases was of a secondary type, consistent with reflux esophagitis (Fig 1). In nine patients, perfusion of hydrochloric acid into the esophagus reproduced the retrosternal chest pain. The two patients with a negative acid perfusion test were considered to have incarcerated hiatal hernias as the cause of their chest pain.

Oral cholecystograms as well as cervical and thoracic spine x-ray films revealed no abnormalities to account for the retrosternal chest pain.

Six patients underwent transthoracic repair of the hiatal hernia and five received medical therapy consisting of antacids, elevation of the head of their beds, and dietary restriction. In an 18-month follow-up, the patients treated by operation have had complete abolition of both positional and exercise-induced chest pain. The patients treated medically have had moderate-to-marked symptomatic improvement.

**DISCUSSION**

With the increased availability of selective coronary cineangiography many patients are now being seen with "anginalike pain" and normal coronary arteriograms. In the management of these cases it is important to exclude noncardiac causes.

Opinion is still divided about the possibility of differentiation between chest pain of cardiac or esophageal origin. Surveys conducted concerning this problem have often been retrospective. A prospective study by Bennett and Atkinson in 1966 was less than ideal in that the definitive parameter for the presence or absence of coronary disease was the electrocardiogram. Delmonico et al in 1968 studied 12 patients with hiatus hernia in whom the diagnosis was made by endoscopy. The patients also had normal coronary arteriograms. Neither esophageal motility nor acid perfusion studies were done in this study.

Radiologically demonstrable hiatal hernias are common even in patients with cardiac disease. Thus, it is crucial to be able to demonstrate disordered motor activity of the esophagus and/or reproduction of the chest pain with acid perfusion studies before attributing the pain to esophageal origin. In the present study 11 patients were investigated because of retrosternal chest pain and were shown to have esophageal disease rather than coronary artery disease on the basis of demonstrating (1) disordered motor activity of the esophagus in all patients, (2) reproduction of the chest pain with acid perfusion into the esophagus in 9, with the remaining 2 patients having an incarcerated hiatal hernia and (3) normal selective coronary cineangiograms in 10 patients, with minor nonobstructive disease in the 11th.

The character, location and duration of the pain in these patients was indistinguishable from that associated with myocardial ischemia. Because of this feature, all patients had from one to seven previous hospital admissions for suspected myocardial infarction. It is not surprising that the nature and location of the pain is the same whether esophageal or cardiac in origin, since both organs have a similar nerve supply. It is also well known that esophageal pain can be precipitated by exertion and relieved by rest. Relief of the pain by use of nitroglycerin, long honored as one of the hallmarks of 'angina pectoris, is also frequently associated with esophageal pain as shown in the present study and by others. In view of the similarities of the pain in these two disorders, it is not surprising that separation on a clinical basis alone is controversial. Dwyer et al in treating a group of ten patients with chest pain and normal coronary arteriograms did not carry out investigations for esophageal disease. They comment that the absence of pain in relation to meals and its association with exertion is in favor of cardiac disease. In the present study, in only 3 of our patients was the pain related to meals, and in 7 of the 11 patients the pain was precipitated by exertion and relieved by rest. Radiation of pain to the back, which has been said to favor esophageal origin of chest pain. 

![Diagram](http://journal.publications.chestnet.org/pdaccess.ashx?url=/data/journals/chest/20966/ on 04/19/2017)
pain, occurred in five patients in this report. Similarly, epigastric pain was present in only two patients. Myocardial ischemic pain, not infrequently radiates to the epigastrium as well as to the back.

In spite of all the similarities in clinical presentation we feel that there are certain features present in this group of patients with esophageal disease which are seldom seen in patients with chest pain due to coronary artery disease. Firstly, the pain was precipitated by changes in posture in all cases (lying down, bending over). Secondly, dysphagia occurred in 8 of the 11 patients (sticking of food on swallowing was noted either at the upper end of the esophagus [pharyngoesophageal dysphagia] or at the lower end of the esophagus [gastroesophageal dysphagia]). In a series of 500 patients with hiatus hernia, dysphagia was noted in 78 percent. Symptons of dysphagia and pain related to postural movements are often not volunteered spontaneously by the patient and may be overlooked if not specifically mentioned by the examiner. Pain relief by antacid therapy occurred in 8 of 11 patients but was often intermittent and uncertain, making interpretation difficult. However, it is one more feature suggestive of esophageal disease but less specific than dysphagia or pain related to postural movements.

In an 18-month followup, the patients treated by operation are now asymptomatic, and the patients treated medically have had significant symptomatic improvement.

The management of the patients reported herein has not presented any particular problems other than in establishing an accurate diagnosis. However, the management of patients with chest pain who have both symptomatic esophageal disease and obstructive coronary artery disease is far more difficult. Recent experience in this center suggests that the coexistence of these two lesions is not at all uncommon.

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