Boerhaave's Syndrome:

The Importance of Early Diagnosis and Treatment

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Boerhaave's syndrome, spontaneous esophageal rupture, is associated with a 70% survival with surgical intervention. Mortality and morbidity are increased in direct proportion to the time between diagnosis and appropriate surgical intervention. Sepsis, hypovolemia and shock are the predominant causes of morbidity and mortality in Boerhaave's syndrome. Two cases of Boerhaave's syndrome are presented which were diagnosed rapidly, and were managed surgically, resulting in survival of the patients. A review of the literature is also presented with emphasis on the clinical and roentgenologic methods of diagnosis of spontaneous esophageal rupture. Particular attention is given to the fact that early diagnosis and treatment will unquestionably reduce the morbidity of this syndrome.

BAROGENIC rupture of the esophagus, the so-called "Boerhaave Syndrome," is the most serious and rapidly lethal perforation of the gastro-intestinal tract.[33] Most series in the literature report essentially a 100% mortality within 7 days without surgery and only a 70% overall survival with surgical intervention.

All investigators agree, however, that mortality and morbidity rates can be significantly lessened by earlier diagnosis and prompt surgical therapy within approximately 12 hours of the catastrophic event. Delay in diagnosis leads to increased mortality and morbidity because simple primary repair is either tenuous or no longer possible. It is the purpose of this report to re-emphasize the pathophysiology and clinical features of Boerhaave's syndrome since errors in diagnosis are usually caused by unawareness of the signs and symptoms of esophageal rupture or failure to consider the possibility. According to Abbott et al.,[1] the correct diagnosis was made within the first 12 hours in only 21% of the cases in their large series.

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Two recent cases of spontaneous rupture of the esophagus successfully treated at Abington Memorial Hospital in Abington, Pennsylvania, will be presented along with a review of the recent literature.

Case Reports

Case 1: H.F., a 49-year-old Caucasian man, was seen in the Emergency Room of Abington Memorial Hospital at 5:00 pm after two episodes of vomiting approximately one hour prior to admission. The second episode was followed by severe epigastric pain radiating to the midback area and associated with diaphoresis. He had a long history of alcohol abuse with a recent alcoholic debauch.

On admission his temperature was 36 C, pulse rate 116/min, respiratory rate 28/min, and blood pressure 150/70 mm Hg. He appeared acutely ill and was writhing in pain. Physical exam revealed clear breath sounds bilaterally, marked epigastric tenderness, and rigidity with rebound. Peristalsis was absent and crepitus was palpable in the neck.

Initial clinical impressions included a perforated peptic ulcer, pancreatitis, alcoholic gastritis, or a pneumothorax. The hemoglobin was 17.0 gm/100 ml; WBC was 9200 with a normal differential; and the serum amylase was 156 units. No evidence of an acute myocardial infarction was present on the initial EKG. Urinalysis was normal.

Plain films of the abdomen were normal and no pneumoperitoneum was present. Chest x-ray revealed a pneumomediastinum without pneumothorax or pleural effusion (Fig. 1). A barium swallow was then performed which demonstrated marked extravasation of contrast from the distal esophagus to the left posterolateral mediastinum (Fig. 2).

The patient underwent an emergency left thoracotomy at 9:30 pm (approximately 5½ hours after rupture) and 400 cc of serosanguinous fluid was present in the left pleural cavity with a marked pleural and mediastinal inflammatory reaction. The mediastinum was intact, but upon opening it a perforation of the lateral distal esophagus, approximately 2.5 cm in length and 1.5 cm above the hiatus, was discovered and repaired primarily with a two-layer silk closure. The mediastinum

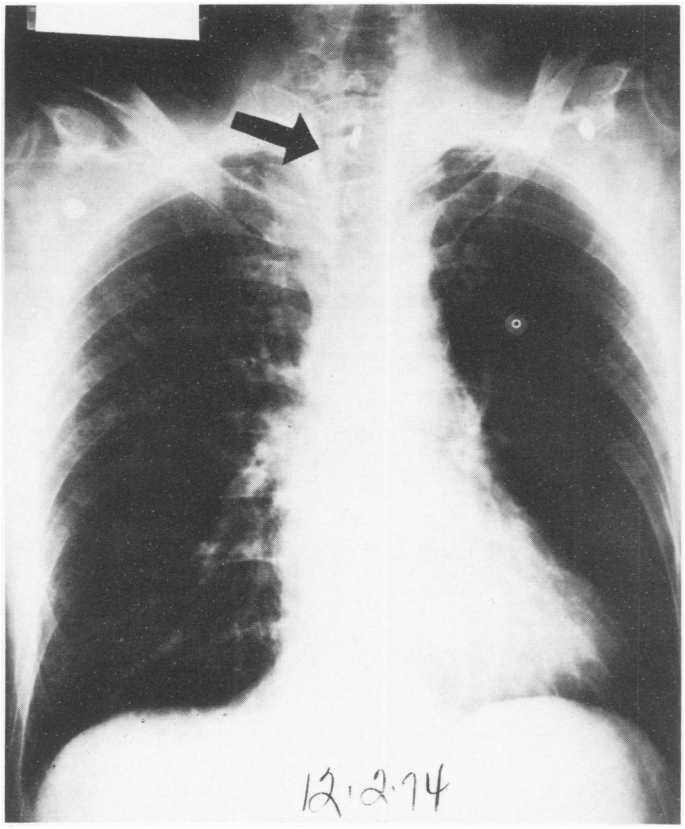

FIG. 1. Case 1. The initial chest x-ray (PA) reveals only a pneumomediastinum without evidence of a pneumothorax or left pleural effusion.

and pleural spaces were irrigated with saline and drained. A nasogastric tube was passed into the stomach intra-operatively. The patient tolerated the procedure well, except for a temperature of 40 C during the operation, secondary to the severe mediastinitis.

He was placed on therapeutic doses of antibiotics (penicillin, cephalothin and gentamicin) and had a postoperative course complicated by delirium tremens and aspiration pneumonia requiring tracheostomy and assisted ventilation. He was discharged on the twenty-eighth hospital day on a normal diet and followup upper GI x-rays were essentially normal.

Case 2: H.G., a 76-year-old Caucasian man, was seen in the Emergency Room of Abington Memorial Hospital at 7:00 pm after one episode of vomiting at 6:00 pm, followed by severe, constant left chest pain radiating to the left upper back and upper abdomen described as "stabbing." Past medical history included a previous cholecystectomy, recurrent left pyelonephritis and coronary insufficiency.

Physical exam revealed an acutely ill man with a temperature of 37.5 C, pulse rate 128/min, respiratory rate 24/min and a blood pressure of 142/70 mm Hg. The breath sounds were decreased on the left with bilateral basilar rales and dullness at the left base. The abdomen was rigid, tender in the left flank with absent peristalsis. The hemoglobin was 13.8 gm/100 ml; WBC was 19,900 with 83 neutrophils and 10 stabs with toxic granulations. Urinalysis showed many bacteria with 40–60 WBC's and serum amylase was 109 units. Plain films of the abdomen were unremarkable. Electrocardiogram showed no evidence of an acute myocardial infarction.

Chest x-ray revealed a left hydropneumothorax (Fig. 3). Gastrografin swallow demonstrated extravasation of contrast into the left pleural space.

At 10:30 pm (approximately 4½ hours after rupture), an emergency left thoracotomy was performed and 900 cc of serosanguinous fluid was aspirated. There was a perforation of the lateral wall of the distal esophagus, approximately 2 cm in length, just above the hiatus with a perforation in the mediastinal pleura also. A two-layer silk closure was performed. The mediastinal and pleural spaces were irrigated with saline and drained after a nasogastric tube was inserted. The operative procedure was tolerated well except for an episode of hypotension after induction of anesthesia believed to be secondary to an increasing tension pneumothorax. The blood pressure improved rapidly after the chest was opened and the tension pneumothorax was converted to an open pneumothorax.

His postoperative course was unremarkable except for a transient postoperative psychosis. He was discharged on the fifteenth hospital day on a full liquid diet after upper G I x-rays revealed a normal distal esophagus but a slight constriction of the midesophagus which 5 months later was proven to be a squamous cell carcinoma.

History

The clinical picture of spontaneous rupture of the esophagus was first described by Hermann Boerhaave, a distinguished eighteenth century Dutch physician, in 1724.[7] His patient, Baron van Wassenaer, died 18 hours after self-induced vomiting. In that era it was the custom of gluttons to induce vomiting with ipecac-like preparations so they could immediately eat another large meal. The baron experienced severe chest pain and dyspnea after vomiting and at autopsy Boerhaave found olive oil and roast duck in the left pleural cavity plus a transverse tear (not the usual linear rent) in the distal esophagus. Since Boerhaave's original 70-page manuscript was written, more than 300 documented cases have appeared in the world literature up to 1970.[1] The majority of these cases have appeared within the last decade as the medical profession has become more aware of the condition.

The first successful surgical therapy was accomplished by Frink in 1941 when his patient survived with drainage of the left pleural cavity alone.[15]

In 1947 Barrett is credited with the first successful repair of the condition, two hundred twenty-two years after Boerhaave's original description.[2]

Incidence and Mortality

Although the condition is not a common one, the rising trend of alcoholism may cause an increase in postemetic ruptures. Rosoff and White[31] report 16 cases from 1958–1973; 3 cases were not diagnosed until autopsy and they had an 18% operative mortality mainly due to sepsis secondary to suture line leaks.

In a 30-year review of perforations of the esophagus from multiple causes (instrumental, corrosives, trauma, etc.), Berry and Ochsner[5] in 1973 reported only one case of Boerhaave's syndrome out of 31 cases of perforation. In 1972, Keighley et al.[18] from Britain reported 12 cases over a 20-year time span.

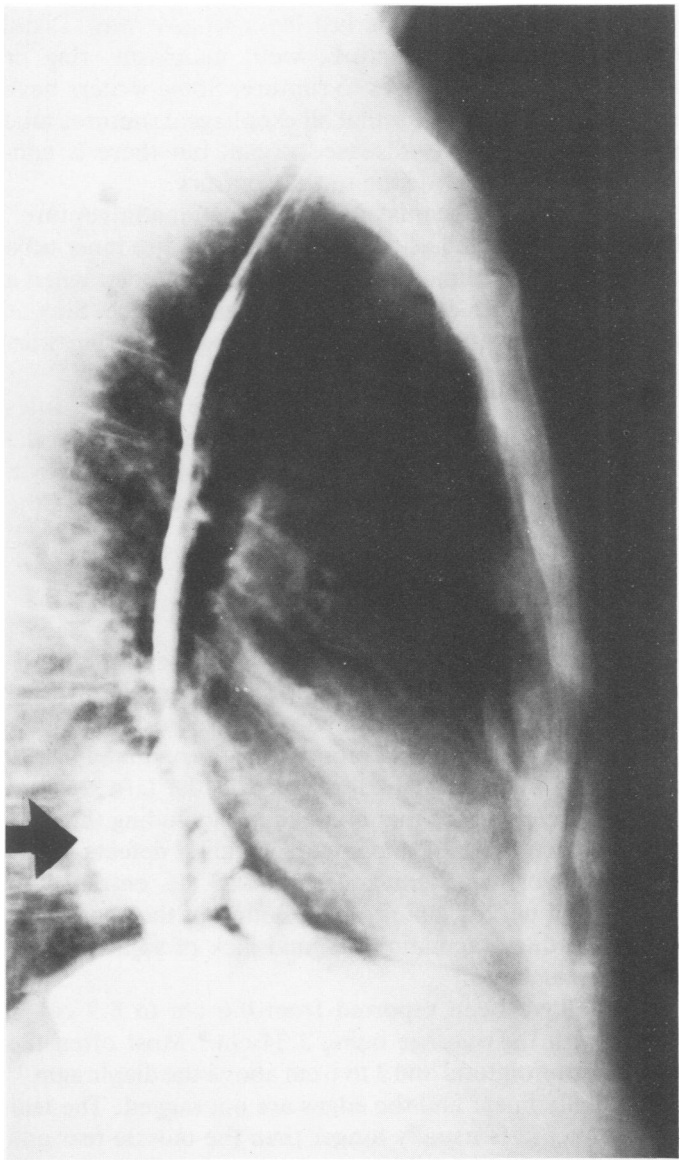

FIG. 2. The lateral x-ray of the chest represents Case 1 after a barium swallow was performed. Obvious extravasation of this contrast material is seen in the left posterior mediastinum.

Perhaps the most informative series is the oft-mentioned collection of 157 cases of Derbes and Mitchell.[11] The extreme lethality of this syndrome is documented in their analysis of 71 untreated cases. Only 35% of the untreated group survived 24 hours; 11% survived 48 hours; and all were dead at the end of one week. In fact, 25% died within the first 12 hours. The survival rate with surgery was 64%. These dismal statistics should be tempered with the realization that the potent antibiotic coverage of today was not available at the time of many of these cases. Nevertheless, it is obvious from these data that without surgical intervention rupture of the esophagus is virtually incompatible with life. Several cases have been reported of survival without surgery,[6,21,26,32,38] but in these patients the diagnosis was greatly delayed and the patient was doing fairly well. This approach is not recommended for the vast majority of these critically ill patients.

Males are afflicted more often than females in ratios reported anywhere from 5 to 1[10] to as low as 2 to 1.[34] The 40 to 60-year-old age group has the highest incidence and the disease is rare in children although 4 cases have been reported in neonates.[13,41] It is felt by some investigators that the esophagus is 13 times stronger in the neonate and 4 times stronger in children than in adults. Interestingly, perforations in neonates are usually on the right side; whereas in adults, 90% are on the left.

Etiology

Vomiting is the most frequent cause of Boerhaave's syndrome, but it is certainly not the only cause and this has led to some debate as to the name of the syndrome. It has been reported with such diverse etiologies as straining, weight lifting, severe coughing, childbirth, blunt trauma, seizures, Cushing's ulcers due to central nervous system disease, seasickness, postoperative vomiting, esophagitis with perforated ulcer, asthma, smooth

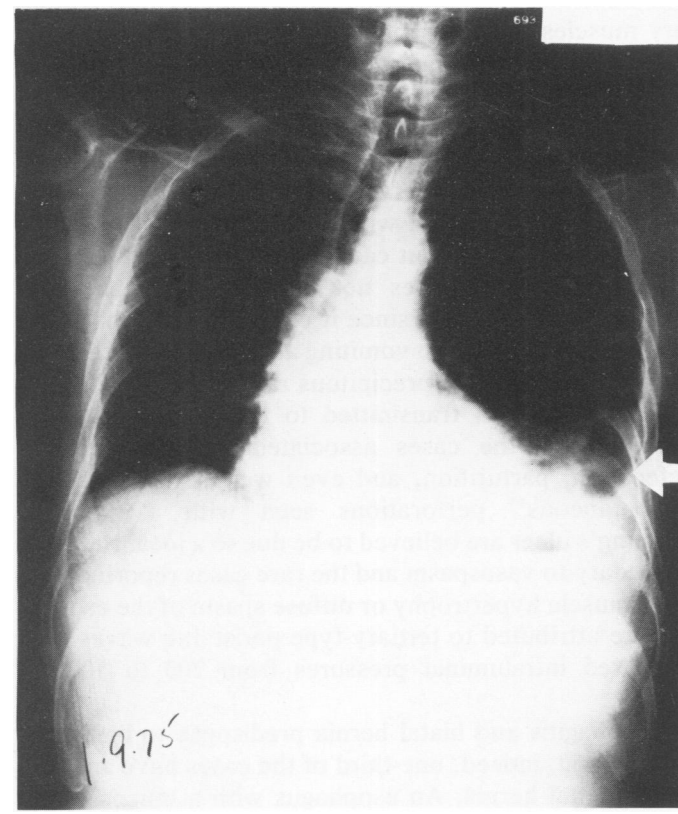

FIG. 3. This postero-anterior chest x-ray of patient number 2 represents an example of a left hydropneumothorax seen after rupture of the esophagus.

muscle hypertrophy of the esophagus, and the drug Antabuse.[14,25,42] Antabuse is a drug given to alcoholics who are attempting to reform and it causes violent vomiting if the patient consumes alcohol while on the drug. Rupture of the esophagus has occurred after the ingestion of certain mushrooms that contain a compound chemically similar to Antabuse.

It is obvious from the above listing that, although "spontaneous rupture of the esophagus" is the most common title in the literature, it is never truly "spontaneous" in that there is a precipitating factor. However, the phrase does connote that the rupture is not due to direct trauma, foreign body, or instrumentation. Abbott prefers "atraumatic panmural rupture of the esophagus" to distinguish Boerhaave's syndrome from the others.

Most frequently, the causative mechanism is a rapid rise in intraluminal pressure with sudden distention of the distal esophagus and it is for this reason that Burford[9] prefers the term "barogenic rupture." "Primary pressure rupture" has also been suggested along with "emetogenic rupture." It has been postulated that protracted vomiting leads to fatigue of the vomiting center in the sensory nucleus of the vagus nerve in the floor of the fourth ventricle.[36] This causes a discoordination of the vomiting reflex which is a complex act requiring the synchronous relaxation and contraction of many voluntary and involuntary muscles. This discoordination leads to physiologic obstruction due to a failure of relaxation of the upper or lower esophageal sphincters at the moment of greatest propulsive force. Cadaver studies have shown that it is not the total pressure exerted but the rapid rise that bursts the esophagus. In fact, pressures up to 9.63 pounds psi have been achieved with hydrostatic dilators in the distal esophagus without causing rupture.

This mechanism does not explain all instances of Boerhaave's syndrome since it can occur with only one episode of emesis or no vomiting at all. Often these cases are associated with a precipitous rise in intra-abdominal pressure which is transmitted to the esophagus. This accounts for the cases associated with straining at defecation, parturition, and even weight lifting. Those "spontaneous" perforations seen with a so-called Cushing's ulcer are believed to be due to a local ischemia secondary to vasospasm and the rare cases reported with giant muscle hypertrophy or diffuse spasm of the esophagus are attributed to tertiary-type peristaltic waves with increased intraluminal pressures from 200 to 500 cm H_2O.[18]

Esophagitis and hiatal hernia predispose to barogenic rupture and, indeed, one-third of the cases have an associated hiatal hernia. An esophagus with a mucosa damaged by esophagitis is three times easier to burst than the normal since the mucosa is the strongest layer and has been shown to be the last layer to give way. Distal obstruction due to stricture, web, neoplasm, ring or achalasia also predisposes to rupture. Some writers have even expressed the view that all esophageal ruptures take place in a previously diseased organ, but there is considerable clinical evidence to the contrary.

Even bizarre circumstances of "pneumatic rupture" have occurred, such as the child who bit a tire inner tube or the 18-year-old man who had his mouth open when a bellows filled with oxygen exploded near his face. Similar instances have occurred with blast injuries during wartime bombing attacks.

Rather than confusing the issue, the multiple titles applied to Boerhaave's syndrome serve to clarify the several different mechanisms which cause it. The eponym is still useful as a unifying factor in discussion of the clinical manifestations.

Pathophysiology

Isolated instances of spontaneous rupture of the cervical esophagus have been reported and, in one series, two out of 16 cases of Boerhaave's syndrome were right-sided, mid esophageal lacerations. In approximately 90%, the rent is in the distal esophagus on the left. Several reasons are given for this predilection including thinning of the musculature of this area, segmental defects in the circular layer, weakening of the wall by entrance of vessels and nerves, anterior angulation of the esophagus at the left diaphragmatic crus, and lack of adjacent supporting structures.[3]

Tears have been reported from 0.6 cm to 8.9 cm in length with the average being 2.24 cm.[3] Most often the tear is posterolateral and 3 to 6 cm above the diaphragm.[10] It is usually linear and the edges are not ragged. The tear in the mucosa is usually longer than the muscle tear and failure to appreciate this may lead to inaccurate repair and subsequent leakage.[19]

Massive bleeding following rupture almost never occurs as it does in the Mallory-Weiss syndrome. In fact, if massive hematemesis occurs with Boerhaave's, the clinician should be wary of a concomitant bleeding gastric or duodenal ulcer as the cause of the emesis.[10]

The cause of the serious cardiorespiratory embarrassment and shock-like condition is undoubtedly a fulminant mediastinitis secondary to the accumulation within the mediastinal and pleural spaces of corrosive gastric juices, enzymes, food and bacteria. Then follows shock, major fluid losses, and mediastinal and pleural suppuration. The mediastinal pleura usually ruptures with the initial insult, but if it does not, it is digested at a later stage by gastric contents.[6,20] If the pleura remains intact, only the mediastinal space is involved initially, but a

reactive serous effusion usually develops on the left (Case 1). Rarely, both pleural cavities may be contaminated with gastric contents.

The mechanism of injury to the esophageal wall is essentially the same in two other clinical entities—the Mallory-Weiss syndrome and intraluminal dissection of the esophagus.[20] In the relatively common Mallory-Weiss syndrome, the mucosal tear is manifested by profuse bleeding because of the rich vascular plexus that is torn with it. The remainder of the wall remains intact as it does in the rare syndrome of intraluminal dissection of the esophagus. Here there is a submucosal dissection creating a true and a false lumen analogous to dissecting aortic aneurysm. A mucosal strip separating the lumina can be shown by radiology as the so-called "stripe sign of Lowman."[23] The relevance of these two conditions to Boerhaave's syndrome lies in the fact that adequate clinical and radiological followup is required since either a Mallory-Weiss or an intraluminal dissection may go on to extraluminal perforation demanding thoracotomy. Usually, they both respond to conservative therapy alone.

Clinical Manifestations

Pain is the most striking feature in this syndrome and it is characteristically excruciating and poorly relieved by narcotics. In approximately 50% of the cases, the classic sequence of forceful vomiting, mild hematemesis and substernal chest pain was present.[34] Usually, it is a pleuritic left chest pain which may radiate to the epigastrium, substernal area or back. Four out of 12 cases in one series[19] manifested left shoulder top pain due to phrenic nerve irritation from the left hemidiaphragm. The pain may be increased by swallowing and swallowing may cause coughing if there is a pleural tear. Occasionally, the patient may collapse with the onset of the severe pain.

Vomiting usually ceases with the onset of pain or there may be no actual vomitus at all, but the patient may admit to retching. Dyspnea is usually present and respirations are typically rapid and shallow because of the severe pain and splinting. It must be remembered that a hydropneumothorax or a tension pneumothorax may be contributing significantly to the patient's respiratory impairment.

The patients may often complain of extreme thirst as is seen in hypovolemic states and they are in some degree of peripheral circulatory collapse in two-thirds of the cases. They are typically anxious, cool, clammy, slightly cyanotic, and have a tachycardia due to a combination of hypovolemia and possible bacteremia. The blood pressure may be decreased and they may be mildly febrile in the early stages or the vital signs may be within normal limits in the first crucial twelve hours particularly prior to rupture of the mediastinal pleura.

A very common clinical picture is that of an acute abdomen with marked reflex rigidity of the upper abdominal muscles, epigastric tenderness and rebound and absent peristalsis. Indeed, the most common misdiagnosis is one of perforated duodenal ulcer. A key point is that, as the disease progresses, the abdominal signs diminish in proportion to the increasing chest signs, but hopefully the clinician will not lose valuable time waiting for this progression.

Another clinical presentation is that of spontaneous pneumothorax with all the characteristic signs such as, tracheal deviation, hyperresonance, loss of retrosternal dullness, and decreased breath sounds. The clinician must be aware also that physical examination of the chest may be completely normal in the early stages of Boerhaave's syndrome.[22]

Palpable crepitus in the suprasternal notch or neck is frequently overlooked or late in appearance although it is present in roughly two-thirds of the cases. This important clinical finding is emphasized in the "Tetrad of Goth" which also includes chest pain, respiratory distress, and prostration. Hamman's sign, a "mediastinal crunch" may be heard in 20% of cases and can be mistaken for a friction rub of pericarditis.

Diagnostic Studies

The laboratory offers little help in the diagnosis of Boerhaave's syndrome and probably the most common finding, although non-specific, is a leukocytosis. Even though this is present in the majority of cases, 14 of 47 patients in Abbott's series[1] had white blood cell counts less than ten thousand, but 7 of these 14 had a leftward shift in the differential count. One-half of the patients in this group had an hematocrit greater than 50% due to hemoconcentration.

If the amylase level of the pleural effusion were to be measured, it would be very high since it is mainly swallowed salivary amylase and this can mimic the picture of acute pancreatitis which may also have a left pleural effusion. However, this fluid is rarely aspirated preoperatively and if the question did arise, one would only have to measure the serum amylase which is commonly normal in Boerhaave's syndrome.

X-ray is the most valuable diagnostic modality for this syndrome and the simple erect film of the chest which is rapidly obtainable yields the most information. The most common error in the diagnostic work-up is the failure to obtain a chest x-ray when a Boerhaave's syndrome presents as an "acute abdomen."

Virtually 80% of the cases have a left pneumothorax plus an effusion on chest x-ray.[40] At least 90% have an effusion with or without pneumothorax and it is bilateral in

7%.[42] Mediastinal air must be looked for since it is present in 66% of the cases. This may be retrocardiac and easily missed.[29] Widening of the mediastinum is occasionally seen and the so-called "V-sign of Naclerio" is another subtle x-ray finding easily overlooked.[27] It represents air in the fascial planes of the mediastinum and diaphragmatic pleura behind the heart. It is both an early and reliable finding when it occurs. Subcutaneous emphysema in the soft tissues of the neck or chest wall also may be seen on the film.

An added benefit of the erect chest film is to rule out a perforated duodenal or gastric ulcer, although one case of Boerhaave's syndrome with pneumoperitoneum and even two cases with pneumopericardium have been recorded.[24,35]

The mediastinum can also be shifted especially with a tension pneumothorax. Rarely, atelectasis alone secondary to splinting of the chest wall is the only roentgenographic finding.

An esophagogram with barium or water-soluble contrast material such as Gastrografin is readily obtainable in most emergency departments, but it must be realized that it is positive in less than 75% of the cases; therefore, a lack of extravasation does not necessarily exclude a perforation. It is in this false negative group that peroral endoscopy is useful if a high index of suspicion of esophageal disruption remains after the contrast study is reported as negative. However, endoscopy plays a very limited role in the diagnosis of Boerhaave's syndrome because it is time consuming, somewhat risky, generally unnecessary for the diagnosis, and the patients are too ill and unstable for the most part to undergo the procedure with its usual degree of safety. It is believed that the false negative esophagograms occur because the material may be too viscous (*e.g.* barium) to leak out, passage is too rapid (*e.g.* Gastrografin), or the perforation is blocked by edema, clots, or food particles. One should always obtain oblique views if the anteroposterior and lateral projections are negative to ensure that a leak is not being hidden on the film by contrast material within the lumen.

There is considerable controversy in the literature as to which is preferable—barium or water-soluble contrast. Abbott, van Heerden, and others believe barium is the material of choice, but the majority feel that it is tremendously irritating to the mediastinum and pleura and is difficult to remove at surgery.[1,36,42]

The size of the rent on x-ray can be very misleading and the clinician should not defer surgery on the grounds that the perforation is small. One "small" perforation seen on x-ray measured 8 cm in length at the thoracotomy!

One can aspirate pleural fluid to measure its amylase content or test its acidity, but this is rarely necessary. Methylene blue has also been given by mouth to see if it appears in the pleural aspirate.

Differential Diagnosis

According to Keighly, the incidence of diagnostic error can be as high as 50% as it was in his series of 12 cases.[19] Perforated peptic ulcer was the most frequent misdiagnosis but can be distinguished from Boerhaave's syndrome by a history of ulcer, pneumoperitoneum and a gradual increase in the severity of chest symptoms in most cases compared to the abdominal findings. Both perforated organs require surgery, but ulcer disease requires laparotomy while Boerhaave's is best approached transthoracically.

The second most common diagnostic error is to confuse esophageal rupture with acute myocardial infarction.[3] The hazards of an unnecessary thoracotomy in the face of an acute myocardial infarction are obvious and underscore the need for an electrocardiogram in the initial evaluation of suspected esophageal ruptures. It is also helpful in recognizing pericarditis and pulmonary embolus which can mimic Boerhaave's syndrome to some degree. Hamman's "mediastinal crunch" can sound like a pericardial friction rub and patients with Boerhaave's syndrome may have pain when sitting forward as with pericarditis. The dyspnea, tachycardia, cyanosis, chest pain, and circulatory collapse of pulmonary embolus bear striking similarity to the manifestations of esophageal rupture and represent a very dangerous pitfall. Other thoracic disease such as dissecting aortic aneurysm (usually in hypertensives and associated with loss of peripheral pulses) and spontaneous pneumothorax (rarely associated with severe pain, vomiting, or subcutaneous emphysema) should be ruled out since thoracotomy plays little role in their usual management.

Acute pancreatitis is often associated with alcohol abuse, vomiting, left pleural effusion, and chest and abdominal pain. A normal serum amylase makes pancreatitis unlikely, however, and a high serum amylase has been reported in only one case of Boerhaave's syndrome.

Other abdominal processes to be considered are biliary colic, mesenteric vascular accident, rupture of a subphrenic abscess, acute pyelonephritis, or incarcerated diaphragmatic hernia (especially with perforation). One can readily appreciate the value of a limited study of the upper gastro-intestinal tract with contrast material in the emergency room to quickly separate Boerhaave's syndrome from this array of more common disease entities.

Therapy

Almost all workers are in agreement that immediate operative intervention is the treatment of choice after vigorous volume replacement has begun. Despite the poor condition of some of these patients, the clinician is faced with virtually a 100% mortality rate with conservative therapy. Some small perforations could probably sur-

vive without surgery, but there is no reliable way to know if a given perforation is small preoperatively.

The preferred technique for most cases is a left thoracotomy in the seventh or eighth intercostal space although the transabdominal route can be used especially if intestinal obstruction or bleeding peptic ulcer was the cause of the initial vomiting.[42]

The most common repair is that advocated by Clagett and Barrett of a two-layer closure of the esophagus and drainage of the pleural space. In fact, Abbott drains the pleural space preoperatively with chest tubes and this also expands the often collapsed lung. Leakage of the esophageal closure with subsequent empyema or esophagocutaneous fistula is the most common and feared complication. The poor blood supply, absence of a protective omentum, lack of a serosal layer, and friable submucosa all lead to breakdown of the suture line in this contaminated situation. It is for this reason that many surgeons have suggested buttressing the primary repair in some way. Intercostal muscle bundles, pericardium, fundus of stomach, diaphragm pedicle flaps and even portions of lung have all been tried.[4,8,17,28,30] As recently as 1974, Rosoff and White report that 5 of their non-buttressed repairs leaked, with two deaths from sepsis.[31]

Derbes and Mitchell had esophagopleurocutaneous fistulas in 19 to 41 patients who survived 5 days after thoracotomy.[11] Abbott actually creates a "controlled fistula" by placing a soft rubber T-tube in the site of rupture and bringing the long arm out through the left pleural space, taking care to position it away from the aorta. He complements this with a gastrostomy, removes the T-tube in 21 days and manages the fistula conservatively until it closes. He rcommends this approach in late cases, malnourished alcoholics, or if a distal stricture is present.

A popular technique at present is the fundic patch of Thal,[37] using stomach to close the defect, plus pleural drainage. Woodward uses a wrap-around patch of stomach similar to a Nissen fundoplication for reflux esophagitis. The Grondahl esophagogastrostomy has also been tried but has not gained much favor.

In the event that a distal carcinoma or other obstructing lesion is found, Clagett recommends primary resection and esophagogastrectomy with drainage, but this is a formidable surgical undertaking in these poor risk patients.

The technique of exclusion and diversion is advocated by Urschel et al.[39] for most perforations but especially in late cases, large perforations, those with distal obstruction, or after failure of standard repair. Since reflux interferes with healing, they perform a cervical esophagostomy in continuity, closure and drainage of the perforation, and place an umbilical tape over a piece of Teflon felt around the esophagus above the cardia deep to the vagi. It is this last maneuver that prevents reflux and this is also complemented with gastrostomy, antibiotics, and intravenous hyperalimentation. The major criticism of this approach is that staged procedures are required.

Smith feels that gastrostomy should be used as an adjunct in all cases of Boerhaave's syndrome, but it is poor for nutritional support since feedings may reflux and increase intrathoracic contamination.[16,34] Jejunostomy or intravenous hyperalimentation are preferred for nutrition and a nasogastric tube can be used for gastric decompression. It can be safely passed during surgery or even preoperatively if caution is exercised.[6,22]

If an esophageal-cutaneous fistula does occur, it will usually close if there is no distal obstruction, local infection, foreign body, malignant change, or epithelialization of the tract. These patients can be fed orally since most food gets through or intravenous hyperalimentation can be used.

Conclusion

A surgical condition which still carries with it a 10 to 30% operative mortality and only a 70% survival with surgical therapy demands increased efforts at earlier diagnosis and more effective management.

There is no question that the most significant contributing factor to the high mortality is delay on the part of the patient and more often by the physician. It is true that alcoholics do often present later to the emergency room and that the syndrome is relatively frequent in comatose or convulsing patients. Nevertheless, a lack of consciousness of the disease, a strong tendency to diagnose peptic ulcer, pancreatitis or myocardial infarction, and a failure to recognize the importance of a left pleural effusion in a patient with a history of recent vomiting, too often leads to a delay that exceeds the critical first 12 hours of the disease.

Along with earlier recognition of esophageal rupture, the key points of surgical therapy bear re-emphasis. Wide debridement of esophageal and mediastinal tissue that is necrotic is paramount along with adequate postoperative drainage. Broad-spectrum antibiotic coverage is necessary. One must be aware of anaerobic contamination from the oropharynx and use antibiotics which have an appropriate spectrum for these anaerobic organisms. Penicillin is the most effective agent against anaerobic organisms from the oropharynx. Simple primary closure in two layers with non-absorbable suture plus drainage tubes will yield a good result in most cases, can be rapidly performed, and is within the scope of most well-trained general surgeons. If the diagnosis is delayed, however, alternate methods may have to be used.

It should be realized that shock, severe debilitation, or a moribund appearance are not contraindications to surgery but underscore the necessity and urgency of

surgical intervention. Blood pressure is often revived after the mediastinum is opened.

A constant awareness of the possibility of esophageal rupture plus the knowledge that surgery is necessary and must be prompt should improve upon the formidable mortality and morbidity of Boerhaave's syndrome.

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