

Clinical-Pathological Conference

Hemoptysis and Abdominal Pain in a 74 Year Old Man

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Clinical Presentation

A 74 year old male was admitted to the hospital surgical service because of abdominal pain.

The patient was well until 20 years earlier when he had a gastrectomy for ulcer disease. He apparently did well until recently when over the past 3 months he complained of symptoms reported by the house staff to be consistent with a "dumping syndrome." Evaluation at another hospital showed a normal upper GI series, barium enema and oral gall bladder study. A celiac angiogram revealed atherosclerotic vascular disease. An ESR was 130. Still suffering, the patient presented with epigastric pain without radiation and a 25 pound weight loss.

The past medical history was notable for an episode of coughing blood 3 years prior to admission, but work-up at another hospital revealed no diagnosis. The patient had a history of smoking and a positive PPD for many years. He denied any history of tuberculosis, liver disease, recent GI bleeding, or alcohol abuse.

On physical examination the patient appeared well but complained of abdominal pain. His temperature was 98 with a pulse of 60, respiratory rate 20, and blood pressure 130/70. No rash or lymphadenopathy was observed. The lungs were clear. The heart showed a grade II/VI systolic ejection murmur at the base, but no gallops were noted. The abdomen was distended with hyperactive bowel sounds. There was pain on palpation in the suprapubic region with a normal liver span and no palpable spleen. No popliteal or femoral pulses were noted. The neurological examination was not remarkable.

The urine was positive for heme without cells present. The hematocrit was 35%; the white cell count was 8600. The urea nitrogen was 23 mg/dl, glucose 114 mg/dl, sodium 139 mEq/liter, potassium 3.7 mEq/liter, chloride 101 mEq/liter and bicarbonate 23 mEq/liter. The electrocardiogram showed non-specific ST and T wave changes. X-ray films of the chest showed a slightly enlarged heart and no evidence of aneurysm. There was possible calcification noted in the lateral projection. The abdominal film was unremarkable. Prothrombin time was normal, as was the serum amylase. A VDRL was non-reactive.

The day after admission an upper GI series revealed a Billroth II without edema or ulcers. Scans of the pancreas and liver scan were also normal. The patient remained NPO on IV fluids, receiving demerol for pain. On the ward

the patient had daily episodes of hemoptysis at a rate of no more than 50 ml per day. On day four he was transferred to the pulmonary unit where he coughed up 250-500 ml of blood. He was intubated at the time and transferred to the respiratory intensive care unit. Blood pressure was 20/palp and the central venous pressure was 0. The patient received blood transfusions, fluids, sedation and assisted ventilation. An arterial blood gas revealed: pH 7.38, pCO₂ 75 mm Hg, and an O₂ saturation of 92 per cent. Physical examination at this time revealed decreased breath sounds at the right base, active bowel sounds and a guaiac negative stool. X-ray film of the chest showed infiltrates in RUL, RML, RLL, and LLL. The next day there was improvement with some clearing of the lungs on chest film. Hematocrit was 28 per cent, LFTs were normal and ESR was 53. An SPEP was normal. The fibrinogen was 479 mg/dl, Ca 7.9 mEq/liter, PO₄ 2.2 mg/dl, alkaline phosphatase 90. The patient extubated himself but continued to do well while coughing up clots of blood over the next 48 hours. His pO₂ rose to 64 mm Hg. Treatment was pursued with sedation, ampicillin and observation. On day 8 he underwent bronchoscopy, but no lesions or active bleeding were seen. Expectored sputum continued to contain blood. A lung scan was consistent with emphysematous change in the LLL. Differential vital capacity showed 60 per cent right lung and 40 per cent left lung. The patient remained stable until day 14 when he began coughing or vomiting bright red blood and became hypotensive. He was intubated but no blood was found. The nasogastric aspirate showed blood in the stomach. Gastric lavage and transfusion were begun, followed by placement of a Sengstaken tube to control bleeding. At this time pH was 6.87, pCO₂ 20 mm Hg, O₂ saturation 80 per cent. He received four units of plasmanate and seven units of blood, but remained hypotensive. EKG remained unchanged. The patient was taken to the operating room where he died during surgery.

Clinical Discussion

This patient presented status post a Billroth II gastrectomy with symptoms of a "dumping syndrome," a 25 pound weight loss and three months of epigastric pain. We are told of a negative GI workup, with a celiac angiogram revealing atherosclerosis. Admission physical examination was notable for mild systolic hypertension with a widened pulse pressure, a II/VI systolic ejection murmur, tenderness in the suprapubic region and absent femoral pulses. Laboratory data are remarkable for a mild normochromic anemia, normal electrolytes, cardiomegaly, heme positive urine, normal prothrombin time and abdominal film, and a nonreactive VDRL. The patient's hospital course was punctuated initially by daily episodes of minimal hemoptysis for three days, followed by a significant episode of greater than 1000 ml blood loss. Let us begin by discussing hemoptysis and its causes. Hem-

optysis, or the coughing up of blood, varies from the production of blood-tinged sputum to only blood. In approximately half of patients with hemoptysis, the standard chest film shows no evidence of gross abnormality and minimal changes. Whether the hemoptysis is scanty or massive is probably of minimal diagnostic value. Hemoptysis can be divided into several major etiological categories for the purpose of discussion. They include infections, neoplasia, autoimmune disorders and vascular abnormalities.

Infections causing bronchitis, bronchiectasis, pneumonia, lung abscess, pulmonary mycosis and tuberculosis, are the most common causes of hemoptysis. Although this patient was a cigarette smoker, his chest film showed no infiltrate and he was afebrile with a normal WBC. We are given no information regarding his sputum. There is no history of alcohol use, heavy sedation, vomiting or seizures that would predispose to aspiration. He had not received chronic antibiotic or immunosuppressive therapy, which would favor the development of infection. He had no history of travel in the tropics or exposure to the oriental lung fluke *Paragonimus westermani*, which can cause hemoptysis.

If a neoplasm were the source of hemoptysis, it should have been detected by X-ray, bronchoscopy, or cytology. In bronchogenic carcinoma, bleeding is often a late symptom, and is usually not profuse. Abnormalities on chest film include segmental or lobar consolidation, atelectasis, a hilar mass, cavitation, a solitary circumscribed nodule, or a large peripheral mass lesion. None of these was present on the film. Occasionally, the standard postero-anterior and lateral chest films may be negative. The lesion is visible bronchoscopically in 70 per cent of cases. Cytological studies have been shown to be positive for tumor cells in 75-90 per cent of cases (Smiddy et al., 1973). This patient had a negative bronchoscopy, negative acid-fast bacillus cultures and negative cytological studies.

Of the immunologic disorders associated with hemoptysis, Goodpasture's syndrome should be mentioned. It may present without evidence of azotemia, but the patients almost invariably have anemia, hematuria, and proteinuria (Bierne et al., 1968). Those were not prominent in this case. The initial chest film and the patient's age further make that diagnosis unlikely. Pulmonary vasculitis does not occur in classic polyarteritis nodosa, but it does occur and is associated with necrotizing pulmonary granulomas in allergic angiitis and granulomatosis as well as in Wegner's granulomatosis. The last two conditions are associated with single or multiple consolidations, often nodular, and sometimes cavitated. There is no radiographic evidence to support those diagnoses in this patient. Furthermore, in Wegner's granulomatosis, severe intranasal inflammation is often present, and rapidly progressive renal failure is common (Fauli et al., 1973). Other possible conditions are pulmonary infiltration with eosin-

ophilia, pulmonary hemosiderosis, systemic lupus erythematosus, and pulmonary alveolar proteinosis. None of those is supported by the available data.

Vascular disorders that can cause hemoptysis include primary pulmonary hypertension, pulmonary arterio-venous fistulas, pulmonary infarction, and mitral stenosis. There was no evidence for any of these entities.

Occasionally, an aortic aneurysm penetrates into the tracheobronchial tree causing death by exsanguination and asphyxiation (Sabetly et al., 1966). However, the chest film showed no evidence of aneurysm, and preterminal intubation showed no evidence of blood. However, hemoptysis may precede fatal hemorrhage by several weeks (Sabetly et al., 1966). This is a little known complication of descending thoracic aneurysms that have become adherent to the adjacent lung (Billig, 1977).

Finally, as in any case of unexplained bleeding, clotting disorders must be considered. None was evident in this patient. Also, trauma or a foreign body can result in hemoptysis, but we have no history to support either in this case. This analysis then brings up an important consideration: Whether in fact the blood was indeed coming from the respiratory tract.

As a distinguishing feature, blood that originates in the airways is usually bright red, mixed with frothy sputum, has an alkaline pH, and contains alveolar macrophages that are laden with hemosiderin. In contrast, blood that originates from the GI tract is usually dark, has an acid pH, contains food particles, and occurs in a patient with a long history of gastric complaints. Of this information, only the history of abdominal complaints was available from the house staff. For that reason, we shall return to the initial complaint of epigastric pain accompanied by a dumping syndrome.

Ulcers at or adjacent to an anastomosis between stomach and small bowel are due to multiple causes. They include retained gastric antrum, continued gastrin production, and incomplete vagotomy. The frequency of marginal ulcers after a Billroth II is 5-10 per cent, although the figure rises to 30-50 per cent in the presence of a retained antrum. Marginal ulcers are refractory to medical therapy, frequently bleed and occasionally perforate. Appropriate treatment is surgical. Despite the negative upper GI series, one should keep these facts in mind. Since exsanguination was the cause of death of this patient, and since he did manage to have a Sengstaken tube placed antemortem, one must consider the possibility of esophageal varices. However, the negative liver scan and upper GI series, normal prothrombin time and liver function chemistries make that diagnosis improbable.

We return to the protocol and note the following data: ESR 130; a celiac angiogram revealing atherosclerosis, and

physical data including a distended abdomen with suprapubic pain on palpation. Those prompt a discussion of aortic aneurysms and their potential effects relative to this patient, namely terminal exsanguination. Aneurysms may occur in any artery, but they are most common and significant in the aorta. Aortic aneurysms produce serious clinical disease and often cause death by rupture. Aneurysms may be classified by indicating, when known, the specific nature of the vascular damage that leads to dilatation. The three most important causes are atherosclerosis, syphilis and cystic medionecrosis. These disorders are responsible for almost all aortic aneurysms regardless of location.

Approximately 75 per cent of all atherosclerotic aneurysms are confined to the abdominal aorta. Many aneurysms that are asymptomatic are discovered on either routine physical or X-ray examination (Billig, 1977). When symptomatic, aneurysms may cause a sense of fullness in the epigastrium; pain when present is usually in the hypogastrium and lower back. A pulsating expansile mass with thrill and murmur may be present; tenderness over the mass may indicate imminent or actual rupture. Owing to difficulty in separating the abdominal aorta from surrounding structures by palpation, one tends to overestimate the size of an aneurysm. Occlusive arterial disease is sometimes detected in the femoral and more distal pulses in the legs (Gore and Hirst, 1973). Both the presence of systemic hypertension and a history of smoking have been clearly documented to be more common in patients with abdominal aortic aneurysm, although whether they play a significant enhancing role has not been established (Billig, 1977).

Rupture into the GI tract results in hematemesis and melena of varying degrees with attendant findings of blood loss. The development of aortoduodenal fistulas in unoperated abdominal aortic aneurysms in 37 patients was reviewed by Rottino (Rottino, 1943). He showed that the enlarging arterial wall erodes the overlying duodenum resulting finally in massive GI hemorrhage often preceded by a smaller bleed. This patient may well have had just such a herald bleed, prior to exsanguination. In spite of this patient's GI surgery, the third portion of his duodenum (the most common site of aortoduodenal fistulas) remained in place. This must be kept in mind when considering the cause of this patient's death.

The other 25 per cent of atherosclerotic aneurysms involve the thoracic aorta (Billig, 1977; Dillon et al., 1967). Dilation may occur anywhere along the thoracic aorta, in the ascending segment, the arch, or the descending portion. The last two sites are most common. Sometimes, the entire aorta is ectatic, with localized aneurysms at many sites. Aneurysms of the descending thoracic aorta not infrequently extend into the abdominal aorta, creating what is in essence a thoraco-abdominal aneurysm. Signs and

symptoms of thoracic aneurysms are related to their size and location and are caused primarily by impingement on adjacent structures (Joyce et al., 1964). They, too, may be asymptomatic, but more often cause chest pain, cough, dyspnea, dysphagia, stridor and hoarseness by compression of the recurrent laryngeal nerve. Tracheal deviation, wheezing, cough, dyspnea, intrapulmonary hemorrhage and hemoptysis are the direct result of compression of the tracheobronchial tree and contiguous lung, especially the left main stem bronchus, by aneurysms of the descending thoracic aorta (Halpert et al., 1962; Joyce et al., 1964). Hemoptysis, sometimes intermittent for several weeks, may result from erosion of the aneurysm into pulmonary parenchyma. Most thoracic aneurysms, particularly those that are symptomatic, are visible on a chest film. However, some, particularly those that are saccular, may be present and even rupture without causing symptoms or being visible on a chest film (Billig, 1977). Rupture of a thoracic aneurysm may occur into the pleural or pericardial cavities or any surrounding structures including the esophagus. Rupture into the respiratory tract may occur at one of three sites: The trachea, the left main stem bronchus, or the left lung (Joyce et al., 1964).

We shall now consider the etiology of aortic disease. The pathogenesis of syphilitic aortitis has been recognized for decades and its pathologic features well documented. Although declining in incidence, it still accounts for one of the largest single groups of aortic inflammatory lesions. In recent years a diagnosis of syphilitic cardiovascular disease is rarely made. Even in the presence of positive serologic tests and overt clinical manifestations, physicians are often reluctant to diagnose syphilis. The fact remains that antibiotic coverage has not eradicated the disease. Old untreated cases still come to light, and tertiary lesions persist.

Syphilitic aortic aneurysms occur anywhere along the aorta, but are most commonly in the ascending aorta. They are usually saccular (hence small and likely to be missed on chest film), but may be fusiform. The development of such aneurysms is based upon the medial destruction characteristic of luetic aortitis. Their signs and symptoms are not unlike those of atherosclerotic origin, and may be completely asymptomatic. On physical examination the presence of precordial pain, hypertension, cardiac enlargement, or congestive heart failure is of little assistance. Accentuation of the aortic second sound and a systolic aortic or apical murmur are helpful in uncomplicated cases. Aortic insufficiency without other stigmata of rheumatic heart disease should be considered as suggestive (Heggveit, 1964).

X-ray studies of the chest may provide valuable clues by showing extensive calcification in the wall of the ascending aorta, especially if an aneurysm is present. This is true in both young and older patients. The calcium deposition

is not caused by syphilis alone, but by atherosclerosis superimposed on the process. Ordinarily atherosclerosis spares the ascending aorta, but syphilis predisposes to calcification. Aneurysm of the aorta is a late complication of aortitis usually occurring from 15–30 years after the initial infection. Fifty per cent occur in the ascending portion, 30 per cent in the transverse arch, 15 per cent in the descending and 5 per cent in the abdominal portions. Syphilitic aortic aneurysms tend eventually to rupture into the right or left pleural space, the trachea, the mediastinum or the esophagus (Heggtveit, 1964). In a series of 100 cases of syphilitic aortitis evaluated at autopsy at Kings County Hospital, N.Y., between 1950 and 1960, only 17 per cent were clinically established antemortem.

In patients over 65 a negative VDRL is an unreliable marker of disease in the presence of aortitis. In fact, one study (Heggtveit, 1964) of 100 cases of syphilitic aortitis revealed the VDRL to be weakly positive or negative in more than 50 per cent of patients. Hence the need for an FTA to exclude reliably the diagnosis in a 74 year old patient. One might also note the lack of aortic insufficiency, which is in fact the most frequent complication of syphilitic aortitis. One should note further that a blood pressure of 160/70 was obtained on admission, demonstrating a mild increase in pulse pressure. Additionally, the murmur of aortic insufficiency, if subtle, can be missed.

The third common cause of aortic aneurysm is cystic medial necrosis. This process is most often the result of chronic stress against the aortic wall, as in longstanding hypertension. It is also seen in hereditary connective tissue disorders such as Marfan's syndrome and Ehlers–Danlos syndrome. A combination of bicusped aortic valve, cystic medial necrosis and aortic root dissection has been described. Clinical manifestations of that process most commonly center around resultant aortic dissection, for which degeneration of the aortic media is believed to be the pathological prerequisite. Pain is found in 90 per cent of cases, although painless dissection does occur (Cohen and Littman, 1964). Those physical findings most typically associated with dissection are pulse deficits, aortic insufficiency, and neurological symptoms (Segarra, 1958). This patient had pulse deficits, and perhaps aortic insufficiency, albeit mild, but had no evidence of cerebral vascular accident, ischemic neuropathy, paraparesis, or change in mental status. In addition, the EKG, which frequently shows left ventricular hypertrophy from preexisting hypertension, showed only non-specific ST and T wave changes. Chest X-ray findings of a wide aortic contour were also not described.

To mention other causes of aortic disease, tuberculous aortitis becomes manifest only after the onset of its major complications. Giant cell arteritis may affect the aorta in about 15 per cent of cases. Rarely, the aortic wall is weakened by this granulomatous process leading to localized

aneurysm formation, aortic annular dilatation and aortic regurgitation. Giant cell arteritis is a disease of the elderly that affects both sexes equally. The illness has rarely been diagnosed before the age of 50, and usually affects those above 60. The symptomatic involvement of arteries is frequently preceded by systemic symptoms, including fever, sweats, malaise, fatigue, anorexia and weight loss. A very high sedimentation rate is a *sine qua non* for this disease and is a valuable guide to the activity of the process. A moderate normochromic anemia is the rule. Biopsy is required for diagnosis, but aortography can differentiate any arteritis from atherosclerotic disease. Unfortunately, such a study was not done. The possibility of aneurysm, constitutional symptoms, an elevated ESR, age and anemia, are all consistent with a diagnosis of giant cell arteritis.

Lastly, Takayasu's arteritis is worth mentioning to complete the discussion. This disease entity has been described by a variety of terms that reflect some of its many features, such as aortic arch syndrome, pulseless disease, reversed coarctation, occlusive thromboaropathy, young female arteritis, as well as Takayasu's arteritis. The disease has an overwhelming predilection for young females and thus would seem unlikely in this case.

To review, it seems likely that there was inflammatory and aneurysmal disease of the aorta, possibly atherosclerotic, syphilitic, giant cell, or non-specific. This in turn led to a fistula with the GI tract with eventual exsanguination. Because aortoenteric fistulas are relatively uncommon, they are not often considered in the differential diagnosis of gastrointestinal hemorrhage. Delayed treatment will allow most aortoenteric fistulas to progress to massive hemorrhage with a mortality of 40–75 per cent (Florendo et al., 1979). The term, herald bleed, has been used to describe the initial hemorrhage of aortoenteric fistulas. Characteristically, the hemorrhage is severe, but often stops spontaneously, to be followed by further episodes of hemorrhage that may be fatal. The most common site of communication between the aorta and the gastrointestinal tract is in the third portion of the duodenum (Barrett, 1949; Rottino, 1943) which in spite of this patient's surgery was still in place and may well have emptied into the gastric remnant. The esophagus is the second most common site, with all other portions of the GI tract being equally rare sites (Rottino, 1943; Sinar, 1977).

One may assume that this patient went to surgery with the surgeon's intent of performing an exploratory laparotomy and/or thoracotomy. At that time a diffusely atherosclerotic aorta was noted with a communication between the thoracic aorta and the esophagus. That could account for the massive terminal upper GI bleed. In terms of the patient's earlier episodes of so-called hemoptysis, one may suspect that the aneurysm was leaking into the proximal portion of the esophagus, thereby accounting for the negative bronchoscopy. The clinical presentation

suggests a panaortitis, most likely syphilitic, though a giant cell or non-specific aortitis cannot be excluded.

Pathology

The patient was taken to the operating room where a gastrostomy revealed active bleeding from the esopha-

gus, and subsequently a thoracotomy demonstrated a fistula between the esophagus and thoracic aorta. The patient was placed on partial cardiopulmonary bypass, and the affected segment of aorta was resected and replaced with a dacron graft. While the surgical procedure was progressing, the patient arrested, and could not be resuscitated.



Figure 1. The aorta has been opened from the normal aortic valve (AV) to the descending thoracic segment. Note the interposed dacron cloth prosthesis distal to the aortic arch vessels. The aortic intima has extensive superficial pearly-grey plaques with focal ulceration and

calcification, giving the vessel a tree-bark appearance. The opened esophagus has a large ulceration (arrows) corresponding to the resected aorta, which represents one end of the fistulous tract extending from the aorta.

The post-mortem examination performed approximately 8 hours after death demonstrated the cause of this patient's clinical symptoms. Upon opening the aorta, the surgical resection site, repaired with a 10 cm segment of woven vascular prosthesis interposed distal to the aortic arch, was visible (Figure 1). That portion of the vessel corresponded to a fistulous tract opening into the esophagus, which was densely adherent to the adventitia and the surrounding mediastinal tissues. The thoracic aorta was diffusely involved by superficial scarring giving a "tree-bark" appearance, with multiple calcified and ulcerating pearly-grey plaques. This process began just above the aortic valve, which by gross examination was uninvolved. The abdominal aorta and iliac vessels had moderate atherosclerotic alterations, but were less severely involved than the thoracic aorta.

The major challenge posed by this case is the nature of the vascular disease that caused an aorto-esophageal fistula. The gross pathology is highly suggestive of syphilitic aortitis, but other processes can mimic these features. We should certainly feel more confident about the

diagnosis of syphilis if the aortic valve was involved; however, the lack of commissural separation and cuspal scarring causing aortic insufficiency does not rule out this condition. Similarly, ankylosing spondylitis and rheumatoid arthritis, which can cause an identical aortitis, also frequently involve the commissures and valve. In this case, the clinical data are inconsistent with those diagnoses.

Can we consider any of the other conditions mentioned by Dr. Matos, particularly medial degeneration of the idiopathic or Marfan's type, Takayasu's arteritis, or giant cell aortitis? Both on the clinical grounds discussed, as well as the pathological features, I do not believe the findings are consistent with such diagnoses. Thus, we are left with differentiating between aortic atherosclerosis versus aortic syphilis. Although the localization and fistula formation favor syphilitic aortitis, an atherosclerotic aneurysm can have the identical features. In fact, 8 years ago I performed an autopsy on a patient with an aorto-esophageal fistula secondary to atherosclerotic disease. Discrimination between these two conditions may depend on the

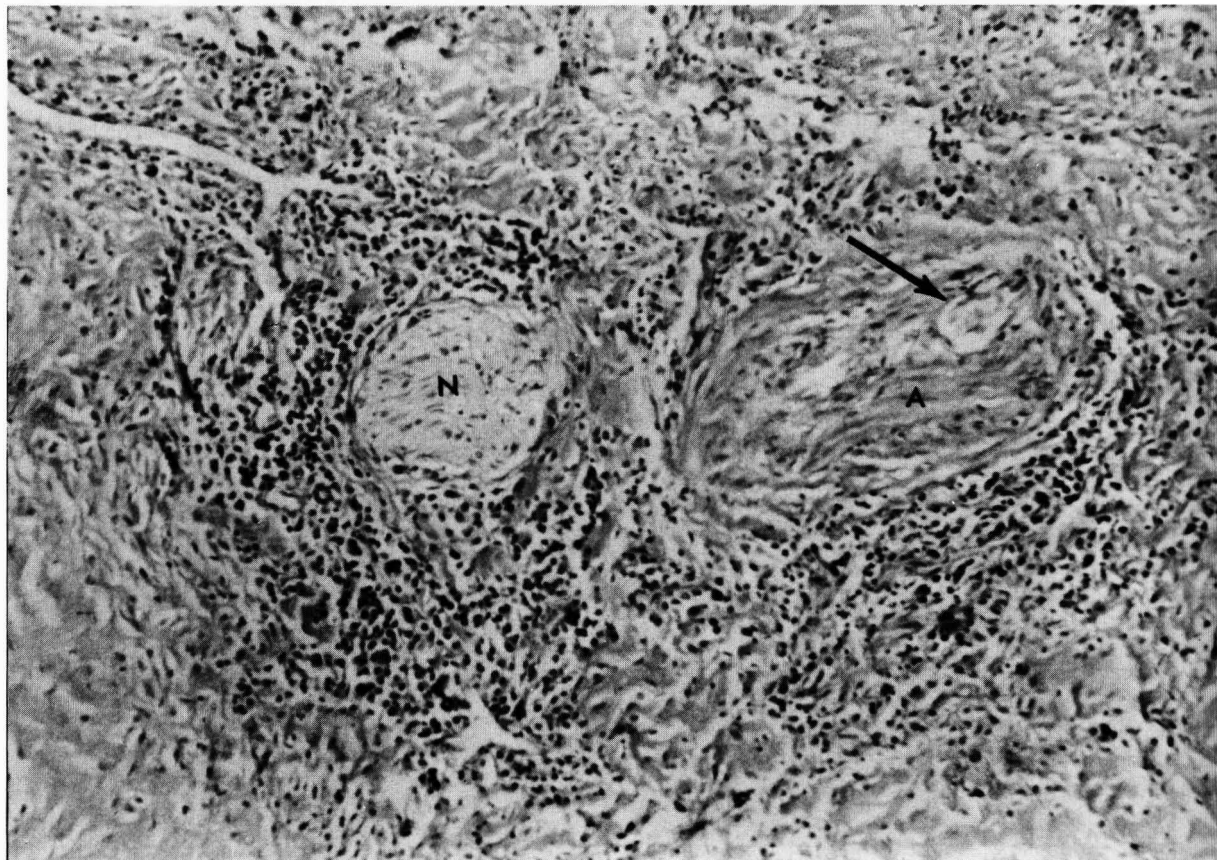


Figure 2. The aortic adventitia is diffusely thickened by scar tissue. A nerve (N) and a muscular artery (A) are surrounded by a diffuse infiltration of inflammatory cells, including many plasmacytes. The ar-

terial lumen (arrow) is almost completely obliterated by connective tissue and proliferated intima (endarteritis obliterans). H&E, X 150

histological analysis of the involved tissue. That was true in the case under discussion.

Sections of the aorta revealed extensive destruction of the wall, with replacement of the medial elastic fibers and smooth muscle by collagen. It is this focal scarring caused by an endarteritis of the vasa vasorum that gives rise to the puckering of the intima, more descriptively known as tree-barking or pig-skinning. The loss of elastic tissue diminishes the tensile strength of the wall and eventually leads to ectasia and finally true aneurysm formation. Characteristically, the vascular scarring is associated with marked narrowing of the vasa vasorum (endarteritis obliterans), and a cuffing of the vessels with large numbers of inflammatory cells, particularly plasma cells. Although inflammation may be observed in the adventitia of aortas affected by atherosclerosis, it is rare to see a predominance of plasma cells. Plasmacytic cuffing and endarteritis associated with marked scarring of the adventitia can be seen in Figure 2. These features are highly consistent with syphilitic aortitis, but in the absence of treponemal organisms or antigens the diagnosis cannot be made with 100 per cent certainty. Staining for organisms was negative, as is true in virtually all tertiary cases, and immunofluorescent procedures were not performed.

Sections of the esophagus, through the fistulous tract, revealed that the aortoenteric connection was relatively long-standing. Thus, it was lined by organizing granulation tissue and blood clot consistent with the clinical course and the multiple episodes of hematemesis. This chronicity is typical of aneurysm rupture, regardless of etiology; aneurysms frequently leak slowly days or weeks before a catastrophic hemorrhage. The explanation for enteric fistula formation, rather than free aortic rupture, is the extensive adventitial inflammation associated with luetic aortitis. The adherence of the aorta to the closely apposed esophagus permitted a tract to develop between the two structures with the progressive weakening of the aortic wall. Within the fistula we discovered embolized atherosclerotic debris, partially digested food particles, and free blood clot associated with the terminal hemorrhage. The final event was massive hematemesis and aspiration of blood into the lungs.

Final Diagnoses

1. Syphilitic aortitis with aneurysm formation and aorto-esophageal fistula
2. Massive hematemesis with pulmonary aspiration of blood

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