Abdominal Aortic Aneurysm with Rupture into the Inferior Vena Cava

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A patient is presented with an arteriosclerotic abdominal aneurysm that ruptured into the inferior vena cava. Clinically, the picture was that of a peripheral arteriovenous communication with heart failure of the “high-output” type. Intestinal bleeding occurred as a result of venous engorgement and hemorrhagic colitis.

ANEURYSM of the abdominal aorta is a rare finding. Osler\(^4\) reported 11 instances of abdominal aneurysm out of 2200 necropsies performed at the Johns Hopkins Hospital. Later, at the same institution, he found 16 cases of abdominal aortic aneurysm among 18,000 necropsies. The first case, according to Osler, was reported by Vesalius in 1567.

Rupture is the characteristic course of the disease. Most often the rupture occurs into the retroperitoneal tissues, the next in frequency being into the peritoneal cavity; a considerable number of instances of rupture into the gastrointestinal tract have been reported as well. Of course rupture may occur into any structure in the abdominal cavity but, as far as we have been able to determine, there has never been a reported case of abdominal aortic aneurysm with spontaneous rupture into the inferior vena cava. It is because of the rarity of this condition that this report is being made.

CASE REPORT

W. B. H., a 69 year old white retired attorney was seen on January 10, 1951, with the chief complaint of generalized aching and lower abdominal cramps. The pain was said to start in the left lower quadrant and to radiate across the lower abdomen to the right lower quadrant. This pain would persist for only a few seconds, and would return every 5 to 10 minutes. Anorexia and some nausea, but no vomiting or diarrhea, were found. The past history was irrelevant except that diverticulosis of the colon had been found when x-ray studies had been made almost 10 years previously.

Physical examination at this time revealed the blood pressure to be 150/90, with no other abnormal findings. After three days in bed he developed lower abdominal tenderness to deep palpation with some abdominal distention, and was expelling large amounts of gas both by mouth and by rectum. At this time it was thought that the patient had diverticulitis with partial intestinal obstruction. He was hospitalized and x-ray studies were made. These revealed numerous distended loops of small bowel. There was a moderate amount of gas within the colon, with a distended loop in the left upper quadrant. There was a rounded area of calcified density in the right upper quadrant which suggested a gallstone. No fluid level was seen. No other masses or soft tissue shadows were visible. X-ray findings were thought to be compatible with intestinal obstruction. He was treated with penicillin, streptomycin, and sulfathalidine. He improved on this regime but continued to have some lower abdominal cramping. On the third hospital day he was discovered to have an abdominal mass about the size of an orange. This was situated a little more to the right of the midline than to the left. During the next few hours the mass increased in size and a continuous murmur over it was noted. The next morning an associated continuous thrill was found over the mass. It was thought at this time that the mass represented aneurysmal dilatation with an arteriovenous communication, most likely between the aorta and the inferior vena cava. The blood pressure was 140/40 and there was a typical Corrigan pulse.

Four days later bleeding from the rectum occurred but this promptly subsided following the administration of morphine. During the remaining six weeks of his life there was constant severe anorexia, and, after taking food, nausea would become evident. In addition, he developed progressive right-sided heart failure which responded poorly to digitalis and mercury. All the typical signs of a high cardiac output were present during this time. The loud continuous murmur and the pronounced thrill continued to be present over the abdominal mass. He became progressively more edematous in spite of therapy, and it finally became necessary to make puncture holes in his legs to allow the fluid to drain. This procedure was associated with a 30 pound weight loss. However, he continued to lose strength as the result of starvation.
Seven weeks after the onset of illness, bleeding from the rectum recurred and the patient expired.

**Final Clinical Diagnosis.** Diverticulitis, with inflammation extending to the wall of an already arteriosclerotic aorta, with consequent dilatation and dissection, and early rupture into the inferior vena cava. It was thought that death was probably brought about by rupture of the aneurysm into the gastrointestinal tract.

**Autopsy Findings.** (The postmortem examination was performed by Drs. Roger Baker and Raymond Stokes, of the Pathology Department, Medical College of Alabama.)

The aneurysm measured 13 cm. across. There was an opening 1 cm. in diameter with rolled edges between the aneurysm and the inferior vena cava (fig. 1). "Apparently this arteriovenous fistula caused great pressure in the venous side of the circulation and one notes that the right side of the heart is greatly dilated." The left side of the heart was also dilated, "but there was no edema and not much congestion in the pulmonary circuit, which one would expect if the pressure were exerted back through the lungs from the left side of the heart." Microscopic section of the aneurysm indicated that it was arteriosclerotic in nature.

Sections of the right coronary artery revealed severe atherosclerosis and old fibrous thickening with recanalization. There was one point of hemorrhage into the wall of this artery. Much fibrosis was noted in the heart muscle but no evidence of a recent infarction.

There were peptic ulcers in the duodenum and stomach which revealed characteristic findings denoting that they had been present for a long while. Agonal perforation of the duodenal ulcer had occurred. There was tremendous venous distention of that portion of the lower bowel drained by the inferior vena cava, and hemorrhagic colitis. "We interpret the bleeding from the lower bowel as probably a matter of bleeding from the hemorrhagic mucosa of the large bowel." Chronic diverticulitis was also present.

**Discussion**

Abdominal aortic aneurysm is found in approximately 0.09 per cent to 0.3 per cent of all cases coming to autopsy. It is less common than aneurysm of the thoracic aorta. This difference is decreasing. This declining ratio is best explained by the fact that: (1) thoracic aneurysms are usually syphilitic, and syphilis is becoming less frequent as a consequence of more effective treatment, and (2) abdominal aneurysms are usually arteriosclerotic, and arteriosclerosis is increasing because people are living longer.

Abdominal aortic aneurysms are caused primarily by syphilis and arteriosclerosis. Those due to syphilis occur in people of 50 years of age or less, the majority appearing in the fourth and fifth decades; and those of arteriosclerotic origin in people 50 years of age or over, the majority appearing in the sixth and seventh decades.

Men are much more likely to have abdominal aortic aneurysms, the majority of the syphilitic variety occurring in male Negroes.

Syphilitic aneurysms usually originate at or above the renal arteries, and are of the saccular variety, while those of arteriosclerotic origin ordinarily arise below the renal arteries and are usually of the fusiform variety.
Syphilitic aneurysms produce symptoms early. Because of vertebral erosion, pain is usually the first symptom. It is usually constant and boring in character. It may throb and become much more severe at night. If pressure is produced on nerve roots, characteristic radicular pain occurs. Pressure on contiguous structures, especially the gastrointestinal tract, may produce anorexia, nausea and vomiting, or renal colic if the ureter is obstructed or a kidney displaced.

Arteriosclerotic aneurysms, due to their more anterior location, either remain asymptomatic entirely (80 per cent of one series) or produce symptoms only at a late stage. The classic and usually the first symptoms from arteriosclerotic abdominal aneurysms are those produced by rupture of the aneurysm.

The most likely place of rupture is into the retroperitoneal tissues. Next in frequency is rupture into the peritoneal cavity, and, rarely, into the gastrointestinal tract. Karabin9 presented an excellent report analyzing symptoms produced by retroperitoneal hemorrhage.

If rupture occurs into the gastrointestinal tract, rapid exsanguination occurs. Melena without rupture into the gastrointestinal tract may occur as a result of venous congestion secondary to obstruction or rupture into the inferior vena cava. Melena may also result from pressure on the gastrointestinal tract alone, as in the case reported by Coggesshal and Genoves.10

Diagnosis was made simple in our case because the mass developed so quickly and produced such outstanding signs. The abdominal pulsations were so great that the bed would move with each pulsation. The mass was expansible, easily felt, and the murmur was very loud. The rupture occurred early into the inferior vena cava, and classic signs of an arteriovenous communication became apparent. The murmur became continuous and machinery-like in type, and a continuous thrill was easily detected. The diastolic blood pressure dropped from 90 mm. Hg to 40 mm. Hg, and the classic pulse pressure of the "overactive" heart was produced.

Consequent to rupture of the aorta into the inferior vena cava, there was a marked increment in venous return to the heart (increased inflow load). This burden on a heart already impaired by myocardial fibrosis led to the typical picture of "high-output failure."

The intestinal bleeding in this patient was due to ulceration of the engorged venous channels of the lower bowel, which developed consequent to establishment of the arteriovenous shunt with a great rise in venous pressure.

Summary

A patient is reported with rupture of an arteriosclerotic aneurysm of the abdominal aorta into the inferior vena cava. The diagnosis can be made readily when a patient presents: (1) a pulsating vena cava; (2) a continuous murmur with systolic intensification over the mass; (3) a collapsing pulse; and (4) the typical signs of an overactive heart with increased output.

In the patient reported, rectal bleeding was due to excessive venous engorgement without rupture of the aneurysm into the alimentary tract.

REFERENCES

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