Arterio-Venous Fistula:

RUPTURE OF ABDOMINAL AORTIC ANEURYSM INTO THE VENA CAVA

ONE OF THE most dramatic complications of arteriosclerotic aneurysm of the abdominal aorta is rupture into the vena cava with a resulting large central arterio-venous fistula. This condition has been reported with increasing frequency in the past several years, though the number of successful surgically treated patients to date is only three. The purpose of this communication is to record the fourth successfully treated patient and to point out that the reported cases have presented a strikingly similar, easily recognizable, clinical picture. Furthermore, early surgical correction of the arterio-venous fistula has resulted in an excellent recovery rate in the reported patients.

Case Report

L.S., a 65 year old, white male retired railroad worker was admitted to the University of Oklahoma Medical Center on 6 January 1959, complaining of back pain and swelling of the legs of two weeks duration.

The family history was not helpful. The patient had been in good general health except for mild hypertension of several years duration. There was no history of recent injury or operation.

One month before hospital admission the patient had experienced the gradual onset of severe lumbar back pain radiating to both hips. Two weeks before admission he noted swelling of the lower extremities and concomitantly became short of breath. Because of increasing severity of these complaints, the patient was referred to the University Hospital Outpatient Clinic.

On admission, the patient was a well developed, obese, white male who appeared to be acutely ill. Vital signs were: temperature

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98.6°, pulse 96/minute, respiration 30/minute, blood pressure 130/70 (both arms). There was no cyanosis. The neck veins were full. Rales were present throughout both lungs. The heart was not enlarged to percussion. There was a sinus rhythm and a soft systolic murmur was audible at the apex. The abdominal veins were dramatically engorged and filled from below. No pulsations were visible in these veins. Abdominal examination was unsatisfactory because of obesity, but no mass was palpable. There was a loud bruit, audible over the entire abdomen and back. Massive pitting edema involved the lower extremities and lower trunk. Femoral pulses were palpable, but no distal pulses were felt in the edematous lower extremities.

The pertinent initial laboratory data included: hemoglobin 11.2 gm., hematocrit 37%, WBC 10,250. Urinalysis revealed a trace of protein and an occasional white cell. An insufficient quantity was obtained on admission to measure specific gravity. Serum electrolytes were essentially within normal limits except for a BUN of 96 mgm.%

The patient was immediately admitted to the University Hospital and initially treated for congestive failure by digitalization and salt restriction. Electrocardiogram revealed no evidence of myocardial infarction. Chest x-ray revealed evidence of cardiac failure and abdominal x-ray revealed no calcification or other abnormalities. During the first 24 hours of hospitalization, the patient's condition deteriorated rapidly. The total urinary output, despite adequate oral intake was 150 cc. Re-examination of the patient thirty hours after admission revealed an increase in the intensity of the abdominal bruit. A review of the available evidence suggested the diagnosis of aortic-vena caval fistula, probably due to a ruptured abdominal aortic aneurysm. Operation was carried out as an emergency thirty-six hours after admission because of clinical deterioration. With the patient under general anesthesia, the abdomen was opened in the midline from the xiphoid process to the symphysis pubis. A large arteriosclerotic aneurysm of the
terminal aorta was found and there was a prominent thrill in the vena cava maximal at a level just above the aortic bifurcation. Because of the patient’s obesity and intense peri-aortic inflammatory reaction, dissection was unusually difficult. The iliac arteries were isolated and tapes passed around them. The aorta was then isolated beneath the renal arteries and was divided between the clamps after clamping the iliac arteries. As the aneurysm was removed, the fistula was controlled using Potts clamps on the vena cava. The fistula was approximately 3 cm. in length and was located about 2 cm. above the aortic bifurcation. The vena cava was repaired using 00000- arterial silk leaving a satisfactory lumen. A nylon bifurcation prosthesis (Tapp-Edwards) was then sutured in place and the aortic flow re-established to the iliac arteries. Appendectomy was performed before abdominal closure.

Postoperatively, the patient improved rapidly; the lungs cleared, blood pressure remained stable and urinary output increased, reaching 1000 cc. on the third postoperative day. The BUN slowly fell to normal by the seventh postoperative day. The abdominal venous distention disappeared, but edema of the lower extremities cleared more slowly and ankle edema was still present at the time of discharge. The patient’s weight on admission was 214 pounds, decreasing to 180 pounds at discharge. Chest x-ray revealed a significant decrease in the size of heart postoperatively. The patient was ambulatory and gaining strength at the time of discharge, nineteen days following operation.

Discussion

Aortic-caval fistulae have been reported occasionally for many years. Most commonly they have been caused by trauma, surgical or otherwise. More recently, a number of cases of abdominal aortic-caval fistulae caused by rupture of arteriosclerotic aneurysms have been recorded. Though the total number of cases reported is small, it seems likely that a combination of an increasing aged population and increased awareness of this condition may result in more frequent early recognition of such cases.

Rupture of an expanding arteriosclerotic aneurysm into the vena cava apparently occurs by the same process that results in rupture into the abdominal cavity, retroperitoneal space or gastrointestinal tract. Because of the nature of the aortic wall at the site of the rupture, these communications tend to be larger than those encountered in fistulae due to trauma. As Eisman has suggested, this probably explains the fact that physiologic derangement following the development of a fistula due to aneurysm is particularly severe and rapidly progressive.

The pathologic physiology of arteriovenous fistulae has received a great deal of attention and a detailed discussion is beyond the scope of this report. Clinically, however, four recently reported cases with arteriovenous fistulae due to ruptured aortic aneurysms have presented a remarkably similar picture and the case described in this report represents a fifth. This group consists only of white males past fifty years of age. All of these patients complained of abdominal discomfort of a rather non-descriptive character for at least one month prior to the onset of acute symptoms. Only one was known to have an aneurysm prior to his acute illness. Each gave a history of swelling of the lower extremities, dyspnea and weakness. Progression of symptoms necessitated hospitalization in a maximum of four weeks in all patients. Physical examination in each case revealed evidence of cardiac failure, engorged veins in the anterior abdominal wall, massive edema of the lower extremities and trunk, and a loud bruit audible over the abdomen. A pulsatile abdominal mass was not always present. X-ray examination of the abdomen was helpful when calcium was visible. Aortography and venous catheterization were diagnostic in the one case in which each was tried. This group of cases then presented a similar, distinctive clinical picture which is not closely resembled by any other entity of which we are aware. Diagnosis in our case was unduly delayed by failure to recognize the significance of this combination of characteristic findings.

Operative treatment when the diagnosis was established consisted eventually of re-
section of the aneurysm, repair of the inferior vena cava and aortic replacement using a bifurcation prosthesis in all of the patients treated surgically. The immediate result in each patient operated on was good. One patient died six months postoperatively and the case reported herein has not been followed for a significant period of time. Without operation, it seems unlikely that any of these patients would have survived more than a few days.

**Summary**

The case of a 65 year old man with an aortic caval fistula caused by rupture of an abdominal aortic aneurysm is reported.

This condition, though rare, presents an easily recognizable clinical picture consisting of a short history of swelling of the legs, dyspnea, weakness, and physical findings of heart failure, increased abdominal venous pattern, edema of the lower extremities and trunk, and a bruit audible over the abdomen.

Prompt recognition and surgical treatment has given good early results in reported cases.

**BIBLIOGRAPHY**


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AMERICAN CANCER SOCIETY institutional grant committee announces the following awards: $1,380 for the support of three Fleming scholars (highschool students) this summer at Oklahoma Medical Research foundation . . . $1,120.95 to Doctor Walter L. Honska, Jr., resident in medicine at Veterans hospital, for studies of the nature of inhibitory effect of normal human and normal rat gastric juice on rat gastric mucosa . . . $1,050 to Doctor William O. Smith, assistant professor of medicine and assistant chief, VA's radio-isotope service, for research on experimental atrophic gastritis produced in dogs by preparation of human gastric juice.