California Medicine

Surgical Treatment of Ruptured Abdominal Aneurysms

Factors Influencing Outcome

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■ *The preoperative course and the postoperative results in 44 patients with ruptured abdominal aortic aneurysms were analyzed with particular emphasis on determining the reasons for the continuing high mortality rate. It was found that rupture was usually a process of staccato progression in pathologic events and a deteriorating clinical course. In cases in which the diagnosis was suspected and operation was performed soon after rupture, the risk was not significantly greater than that of elective aneurysmectomy. When operation was delayed until shock was established, the mortality rate rose precipitously.*

APPROXIMATELY HALF OF the patients operated upon for ruptured abdominal aortic aneurysm die during or soon after operation.[1] In spite of increasing experience, no significant reduction in the mortality rate has been made in the past 12 years.[1] However, during this 12-year period the mortality rate for *elective* aneurysmectomy in a series of 510 operations at the University of California Medical Center has dropped from 15 percent to less than 5 percent. To analyze the factors responsible for the high risk of emergency operations on ruptured aneurysms, the case records of patients operated upon for aortic rupture were reviewed. From this analysis it is hoped that principles in management can be formulated which will decrease the operative risk.

Clinical Material

Forty-four patients with ruptured abdominal aortic aneurysm were admitted to the University of California Medical Center, San Francisco, between July 1, 1957 and December 31, 1967. Their

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ages ranged from 52 to 77 years with a mean age of 63 years. Thirteen of the patients (30 percent) were over 70 years of age.

Pathologic Characteristics. All aneurysms involved the infra-renal abdominal aorta. Variable degrees of enlargement in the common iliac arteries were also present. The site of rupture was the aorta in 42 patients and the common iliac artery in two. Five categories of rupture, depending upon the direction and extent, could be identified. Four patients had limited extravasation of blood beneath the adventitia of the aorta (sealed). In 23 patients rupture had extended through the adventitia into the retroperitoneum (contained). In 13 patients, rupture, usually from the anterior wall of the aneurysm, produced intra-peritoneal hemorrhage (free). The aneurysm ruptured into the duodenum in two patients and into the inferior vena cava in two.

The diameter of the aortic aneurysms varied from 7 cm to 17 cm (mean 11 cm). In the two patients with rupture of the iliac portion the diameter of the associated iliac aneurysm was 6 cm. (The average external diameter of the aorta and common iliac arteries is 3 cm and 1.5 cm respectively.)

Clinical Features. Except for massive gastroduodenal hemorrhage from aorto-duodenal fistula and acute intractable congestive failure secondary to rupture into the inferior vena cava, the clinical features in patients with ruptured aneurysms were remarkably consistent and typical. Abdominal or back pain and a pulsatile abdominal mass were uniformly present.[2] Twenty-six patients (59 percent) were in shock at the time of admission to hospital. The duration of acute symptoms before admission in the 32 patients who could give a reliable history ranged from six hours to 21 days; the average was three and a half days. Faintness or temporary loss of consciousness frequently appeared at the onset of pain. Over half of the patients had early improvement in both pain and light-headedness. Subsequent recurrence or increase of pain, hours or even days after onset, was accompanied by profound and persistent fall in blood pressure.

Two-thirds of the patients were asymptomatic before rupture occurred and were unaware that an aneurysm was present. Continuous abdominal pain lasting for more than a month before rupture of the aneurysm was the complaint leading to an earlier diagnosis in the 13 patients who were known to have an aneurysm before rupture occurred.

TABLE 1.—*Causes of Operative Mortality in Ruptured Abdominal Aneurysm*

	Postoperative Day	*Cause of Death*
Intraoperative (11 deaths)		Shock
Postoperative (11 deaths)	1	Myocardial infarction
	2	Graft thrombosis
	2	Gangrene of colon
	6	Hepatic and renal failure
	7	Aspiration penumonia
	7	Renal artery thrombosis; renal failure
	8	Anastomotic leak with hemorrhage, occlusion of renal vein and renal failure
	8	Intestinal infarction, femoral artery occlusion, and renal failure
	10*	Myocardial infarction
	14	Pulmonary sepsis and renal failure
	30*	Pulmonary sepsis and pulmonary insufficiency

*No preoperative shock.

Management

All patients were taken immediately to the operating room. Only for patients without shock and with stable blood pressure was operation delayed to permit skin preparation, cross-matching and obtaining blood. In patients with shock, operation was begun after minimal skin preparation, and unmatched Type O blood was used. In these patients the average between hospital admittance and laparotomy was 50 minutes.

Proximal control of bleeding was obtained by cross-clamping the aorta above the aneurysm and below the renal arteries. This portion of the dissection, although often accomplished without direct vision, was usually facilitated by the dissection produced by the hematoma around the aorta. The remainder of the operation was performed in the usual manner for graft replacement of the abdominal aorta.

Results

Twenty-two (50 percent) of the 44 patients died during the time in hospital. Three others died subsequently from complications of operation. The causes of death are shown in Table 1. Although numerous causes are listed, at least one of three factors unique to the circumstance of aortic rup-

TABLE 2.—*Incidence of Shock and Mortality in Various Types of Rupture of Abdominal Aneurysm*

Type of Rupture	*No. of Patients*	*No. of Patients In Shock*	*No. of Deaths Postoperatively*	*Mortality (Percent)*
Sealed	4	0	0	0
Contained				
Small	7	1	1	14
Large	16	12	11	69
Free	13	11	9	69
Aorto-duodenal	2	2	1	50
Aorto-caval	2	0	0	0
Total	44	26	22	50

ture, and rarely encountered in elective aneurysmectomy, can be identified in each case. These common factors are shock, sepsis and iatrogenic injury.

Shock. Death as a result of prolonged shock resulted from failure of one or more organ systems (heart, kidneys, lungs and liver). Cardiac arrest following prolonged shock was the immediate cause of death in the 11 patients who died before leaving the operating room. Recent myocardial infarction was found at autopsy in five of these patients. Prolonged preoperative shock had been present in one of the two patients who died postoperatively from myocardial infarction. Renal failure, secondary to shock, was the cause of death in four patients who survived the immediate operation.

The vasodilatation that occurs with general anesthesia usually compounded the problem of blood pressure control. The blood pressure of those patients already in shock was further depressed. In a few patients whose blood pressures appeared to be controlled on arrival to the operating room, pressure dropped to shock levels once anesthesia was started.

The response to fluid replacement in the management of shock was the critical factor in survival. Twenty-six patients were in shock when they entered the hospital. In 21 of these patients shock was refractory to volume replacement and only two survived. Normal blood pressures were restored in five patients and three of them survived.

Sepsis. Local graft sepsis followed by anastomotic disruption led to death in three patients; two of them died six weeks postoperatively and one died nine months postoperatively. One patient had sepsis after the repair of the aortoduodenal fistula. In the second patient, one ureter had been transected and, although repaired, this was considered to be the cause of retroperitoneal infection. In the third patient, in whom retroperitoneal abscess led to anastomotic disruption nine months postoperatively, operation had been performed without sterile preparation.

Iatrogenic Factors. Technical errors, which rarely occur during elective aneurysmal operations, were the result of the need for haste and the distortion and obscuration of the normal anatomy which the aortic rupture had caused. Laceration of an iliac vein in one patient, transection of a ureter in another and thrombosis of a renal artery in a third all contributed to a fatal outcome. In two patients, gangrene of the left colon was probably the result of ligature of the inferior mesenteric artery at a level more distal to that used in an elective operation.[3]

The deaths from graft thrombosis (one patient), pulmonary sepsis (one patient) and aspiration pneumonia (one patient) were the only ones not specifically related to rupture of the aneurysm.

Nineteen patients survived the operation without late complications. No follow-up information is available on one of these patients and two have died from unrelated disease. Sixteen patients were living and well without further arterial difficulties at the time this report was written.

Discussion

The lethal influence of shock and the problem of its control in patients with aortic rupture have long been recognized.[1,4,5] In this series, when shock was overcome the mortality rate was 10 percent, but when shock was prolonged the mortality rate was 90 percent. A review of the case histories and the pathologic findings strongly suggests that most of the patients who died would have survived if the diagnosis had been made with the first onset of symptoms and operation had been performed immediately. Among the patients with various pathologic categories of rupture, those with sealed rupture and the most favorable prognosis were operated upon early. Those in prolonged shock, in whom bleeding had extended into the retroperitoneum and abdominal cavity, were generally operated upon late. The comparative mortality rates in these categories are given in Table 2.

These observations suggest that rupture often begins with self-limited extravasation into the subadventitia or peri-aortic tissue. Pain and a temporary systemic response to minor blood loss are experienced. A variable period of time may then elapse before rupture extends into the less resistant

area of the retroperitoneum.[5] It is during this period, when the patient's blood pressure is stable and only a minor distortion of peri-aortic structures has occurred, that aneurysmectomy can be performed with a risk little greater than that in operation for unruptured aneurysms. Since the aneurysm can be expected to exceed 7 cm in diameter[6] it should be readily detected by abdominal palpation. Even if the diagnosis of aortic rupture is wrong, nothing would be lost by early operation since the threat of eventual rupture is an indication for elective resection for aneurysms reaching this size.

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CLASSIFICATION OF EYE CHANGES IN GRAVES' DISEASE

"[In the new system for classifying or grading eye changes in Graves' disease approved by the American Thyroid Association], the plan is to have classes. Obviously, Class O means that nothing is wrong with the patient, even though he may be thyrotoxic. Class I is the category that used to be called mild or noninfiltrative, namely that end of the spectrum where eye changes are only cosmetic; and that means the signs are present, but none of the alarming symptoms — burning, tearing, sandiness, etc. To distinguish this, we made a separate category, even though proptosis is obviously common to both the more severe changes and this relatively mild area where the prognosis is excellent no matter what you do.

"Now from this point on we get involved with the serious kind of eye changes known in the past as the infiltrative or severe, where the patient often starts out with some tissue involvement and no proptosis, maybe nothing except a feeling of irritation. It's often called allergy by the ophthalmologist or it's mistaken for conjunctivitis or something of that sort. It isn't till later that the suspicion dawns that this is Graves' disease. You go from there to proptosis, which is a little bit more advanced and then to extraocular muscle involvement which is still more advanced. Finally the last two [classes] which are quite serious as far as sight goes — corneal involvement and then loss of vision, which means the optic nerve is getting involved.

"This system accounts for practically every symptom and sign and lends itself to a mnemonic. . . . The idea is that 'no' means no serious prognosis; 'specs' is the bad part. Going in order this is no involvement, only signs; then comes soft tissue [involvement], proptosis, extraocular muscle [involvement], cornea [involvement], and sight [loss]. With the help of this mnemonic, the system is really awfully easy to use."

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