CASE REPORTS

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Perioperative Hepatic Dysfunction in Two Patients during Elective Aortic Aneurysm Repair

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HEPATIC ischemia may occur during shock, massive hemorrhage, sepsis, or congestive heart failure.[1,2] The acute hypoxic hepatocellular necrosis that occurs presents as a syndrome referred to as acute ischemic hepatitis (AIH),[2] shock liver,[3] or acute hepatic infarction.[4]

We describe two patients who underwent elective aortic aneurysm repair and in whom, perioperatively, disseminated intravascular coagulation (DIC) and liver dysfunction developed, associated with an increase in serum concentrations of aspartate aminotransferase (AST) and lactic dehydrogenase (LDH), as is usually seen in AIH.[2,4,5] Although DIC is known to be a complication of aortic aneurysm repair, hepatic dysfunction consistent with AIH has never been clearly described in this clinical setting. In several case reports, increases in serum hepatic transaminases were described in patients who underwent aortic aneurysm repair,[6,7] but these enzyme increases were not clearly associated with ischemic hepatitis; rather, they were considered as part of multisystem organ failure.

Case Reports

Patient 1

A 65-yr-old woman was admitted at our institution for elective repair of a thoracoabdominal aneurysm that extended from the left subclavian artery to above the renal arteries with occlusion of the celiac axis and superior mesenteric artery. Preoperative laboratory results included a hemoglobin of 11.9 g/dl, hematocrit of 35%, platelet count of 225,000/mm^3, prothrombin time (PT) of 11.9 s, and partial thromboplastin time (PTT) of 26.9 s. The creatinine was 0.9 mg/dl, blood urea nitrogen was 12 mg/dl, and blood glucose was 99 mg/dl. Serum transaminases were LDH 175 IU/l (normal, 100–220), AST was 25 IU/l (normal, 7–40), and alkaline phosphatase was 96 IU/l (normal, 20–120); total bilirubin was 0.4 mg/dl (normal, 0–1.5). An electrocardiogram showed Q waves in the II, III, and VF leads that had also been evident on an electrocardiogram obtained 6 months earlier. An echocardiogram indicated hypokinesia of the distal segment of the interventricular septum and the inferior myocardial wall.

After right radial arterial and intravenous catheters were secured, a lumbar subarachnoid catheter (L_{4-5}) for cerebrospinal fluid drainage and a thoracic (T_{8-9}) epidural catheter for postoperative pain management were inserted. A pulmonary artery catheter was introduced through the left internal jugular vein. Anesthesia was induced with sodium thiopental, midazolam, fentanyl, and vecuronium. After the trachea was intubated with a left-sided double-lumen tube, anesthesia was maintained with O_2, isoflurane, and fentanyl. After anesthetic induction, additional intravenous access was established (14-G and 8.5-French catheters in the right and left antecubital veins). The cerebrospinal fluid was drained continuously to maintain a cerebrospinal fluid pressure of 10–13 mmHg.

The anesthesia course was uneventful for the first 4 h of surgery. During the preclamp period, only crystalloid and colloid solutions were administered. Mannitol was infused (25 g) 30 min before cross clamping of the aorta. The aorta was cross clamped just distal to the origin of the left subclavian artery, and 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ sodium nitroprusside was used to blunt the hemodynamic response to cross clamping. Also, 100 mEq/h sodium bicarbonate was given immediately after cross clamping. Before cross clamping, the arterial pressure was 128/80 mmHg; after after cross clamping, it increased to 160/95 mmHg, and decreased to 135/80 mmHg after intravenous administration of 80 μg nitroglycerin. Surgical repair of the aneurysm involved creating bypasses from the aortic graft to both the celiac axis and superior mesenteric artery, as well as reimplantation of the intercostal arteries (T_4, T_7, T_8, and L_2). Approximately 45 min after aortic cross clamping, the patient's systolic blood pressure decreased to 85 mmHg; for this reason, more crystalloid and colloid solutions were infused. Afterward, a repeat hematocrit was 18%, and blood replacement was begun. Despite administration of blood and colloids, maintaining an adequate blood pressure was difficult; therefore, norepinephrine and dopamine at 0.1 $\mu g \cdot kg \cdot^{-1} min^{-1}$ and 10 $\mu g \cdot kg \cdot^{-1}$, respectively, were administered, and the blood pressure increased to 120/55 mmHg and was maintained. After 1 h and 35 min of aortic cross-clamp time, the proximal aortic clamp was released, and blood flow was restored to the liver and both kidneys. Despite rapid intravascular volume replacement, the blood pressure decreased briefly to 90/50 mmHg while pulmonary artery and central venous pressures increased to 40/22 mmHg and 20 mmHg, respectively. Cardiac out-

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put decreased from 3.5 l/min to 2.9 l/min, and 500 mg calcium chloride was given intravenously. In addition, norepinephrine and dopamine infusions were increased to 0.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$, respectively, which was sufficient to maintain the blood pressure at around 120/85 mmHg for the rest of the surgery. Acute onset of diffuse microvascular bleeding was noted soon after the proximal aortic cross clamp was released. Blood for coagulation tests was drawn, but fresh-frozen plasma, platelets, and packed red blood cells were infused with a rapid infusion device even before the test results were available. The coagulation tests (available 45 min later) were PT 29 s, PTT greater than 180 s, fibrinogen 55 mg/dl (normal, 200–400 mg/dl), and platelet count 60,000/mm^3. Serum transaminases were LDH 696 IU/l and AST 165 IU/l, whereas alkaline phosphatase was 35 IU/l.

After 2 h and 10 min of cross-clamp time, the distal aortic cross clamp was released, and blood flow to the legs was established. At that time, hypotension (systolic blood pressure of 80 mmHg) and a 30-s episode of ventricular tachycardia occurred, for which the patient was treated successfully with 100 mg of lidocaine and 50 mEq of bicarbonate. Arterial blood gas measurements immediately after treatment were: *p*H 7.18, Pa_{CO_2} 33 mmHg, Pa_{O_2} 138 mmHg (on FI_{O_2} 1.0); serum bicarbonate was 18 mmol/l, base deficit −8 mmol/l, and potassium 4.7 mmol/l. Throughout the remainder of surgery, bleeding was diffuse, and we continued to replace blood and blood products. The estimated blood loss during the surgery was more than 20 l. Blood and blood products replacement during the entire procedure consisted of 36 units of fresh frozen plasma, 22 units of packed red cells, and 11 units of platelets. During surgery, we salvaged 16.8 l of blood; of this, 4.3 l was processed for reinfusion, but only 2.5 l was transfused intraoperatively. The remaining 1.8 l was sent to the blood bank for washing before later reinfusion. (Although the question of whether salvaged blood can cause coagulopathy is still controversial, we were concerned that transfusion of a large amount of salvaged blood, because it contains high titers of fibrin degradation products,[8] could worsen the existing severe DIC.) At the end of surgery, the PT was 17.6 s, PTT was 45.7 s, fibrinogen was 135 mg/dl, platelet count was 280,000/mm^3, and D-dimer was between 2,000 and 4,000 ng/mL (normal, <250); the hemoglobin was 6 g/dl, hematocrit was 18%, creatinine was 1.4 mg/dl, lactic acid was 11.7 mmol/l (normal, 0.5–2.2), and serum glucose was 295 mg/dl. Serum transaminases were LDH 2,244 IU/l, AST 792 IU/l, and alkaline phosphatase was 63 IU/l. Serum ammonia and bilirubin concentrations were 38 mmol/l (normal, 11–35 mmol/l) and 1.4 mg/dl, respectively.

In the intensive care unit, 6 units of fresh frozen plasma, 5 units of packed red blood cells, and 20 units of cryoprecipitate were administered to the patient overnight. Her immediate postoperative pulmonary status was consistent with pulmonary edema, which required continuous mechanical ventilation of her lungs as well as a furosemide intravenous drip at 5 mg/h. The patient's hemodynamic parameters remained stable throughout the entire postoperative period, but her liver function continued to deteriorate, and, on postoperative day 2, the LDH was 5,280 IU/l (LDH_5 isoenzyme fraction was 68%) and AST was 4,950 IU/l. Bilirubin and alkaline phosphatase were 3.1 mg/dl and 149 IU/l, respectively. Also during postoperative day 2, urine output declined and the patient required hemodialysis. During the first postoperative week, the platelet count was between 56,000/mm^3 and 150,000/mm^3, PT was between 21.6 and 16 s, PTT was less than 29.5 s, D-dimer was greater than 8,000 ng/ml, and fibrinogen was greater than 200 mg/dl. The patient's neurologic status was grossly intact; she followed commands, moved all extremities, and showed no signs of hepatic encephalopathy. By postoperative day 7, urine output had improved and serum transaminases had fully normalized (fig. 1, upper panel). On postoperative day 7, respiratory function was normal, and we were able to extubate the trachea. The patient was discharged home on postoperative day 26 in good general condition and, although she required no further dialysis, the creatinine remained elevated (3.3 mg/dl).

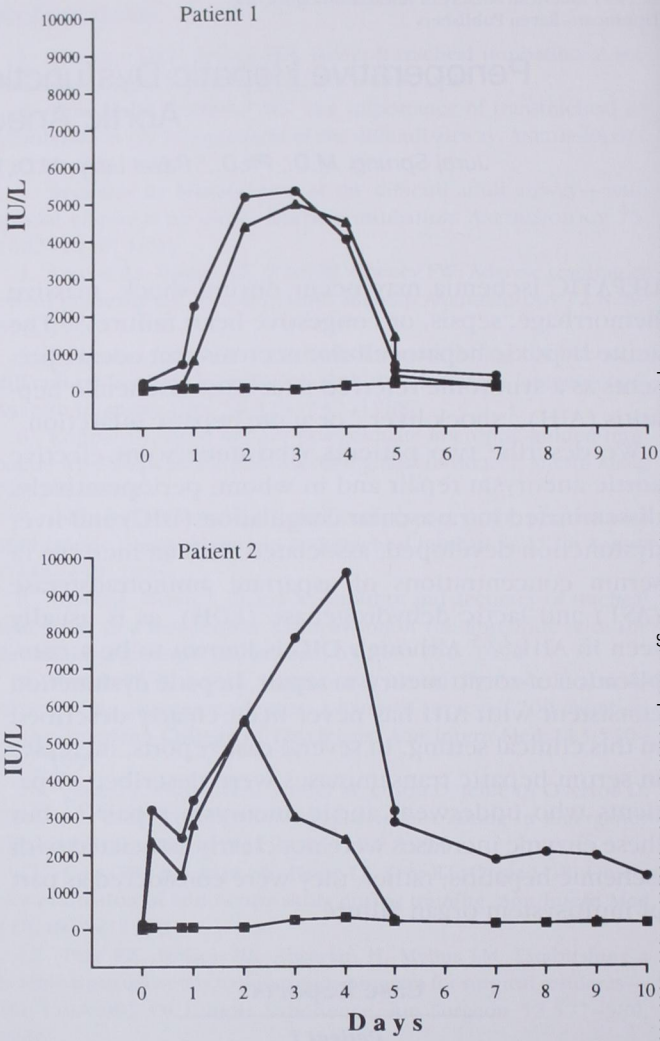

Fig. 1. Perioperative changes in serum transaminases and alkaline phosphatase in two patients who underwent aortic aneurysm repair and developed acute ischemic hepatitis. ● = lactic dehydrogenase; ▲ = aspartate aminotransferase; ■ = alkaline phosphatase.

Patient 2

A 57-yr-old man presented with lower back pain and was diagnosed as having a 6-cm infrarenal abdominal aortic aneurysm. His previous medical history was significant for coronary artery disease that required coronary artery bypass grafting 7 months earlier. Preoperative

laboratory results included a hemoglobin of 13.5 g/dl, hematocrit of 39%, platelet count of 222,000/mm^3, PT of 12.1 s, and PTT of 25.4 s. The creatinine was 1.0 mg/dl, blood urea nitrogen was 20 mg/dl, and serum glucose was 90 mg/dl. Total bilirubin was 0.4 mg/dl, alkaline phosphatase was 56 IU/l, LDH was 139 IU/l, and AST 18 was IU/l.

After intravenous and radial artery cannulae were secured, anesthesia was induced with fentanyl, isoflurane, and pancuronium and maintained with isoflurane and 1:1 mixture of O_2 and N_2O. A central venous catheter was introduced through the right internal jugular vein, and dopamine was infused at 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The blood pressure before anesthesia induction was 100/60 mmHg, and the heart rate was 70 beats/min; both remained near these values throughout surgery. As a reaction to aortic cross clamping, blood pressure increased to 150/90 mmHg. The aorta was cross clamped above the renal arteries for 1 h and 40 min and was repaired with an aortobiliac graft. In addition, a left aortorenal graft was performed. An estimated blood loss of 1.8 l was replaced with crystalloid and colloid solutions and 850 ml of salvaged blood. At the end of the operation, one small-bowel loop appeared dusky; however, the mesenteric vessels at its surface were pink, pulsations were good in the mesentery below the bowel, and peristalsis was visible. Therefore, the bowel color was attributed to mild bowel injury caused by compression with retractors.

The patient was transferred to the intensive care unit in hemodynamically stable condition with good urinary output. His immediate postoperative hemoglobin was 11 g/dl, the hematocrit was 33%, and the platelet count was 95,000/mm^3. Four hours later, the patient became hypotensive and required fluid resuscitation with 2 l of colloids, 2 units of fresh frozen plasma, and 4 units of packed red blood cells, after which the hemoglobin was 7.0 g/dl, hematocrit was 21%, PT was 22.4 s, PTT was 55.9 s, platelet count was 31,000/mm^3, fibrinogen was 143 mg/dl, and the fibrin split products were greater than 40 μg/ml (normal, 0 μg/ml). Metabolic acidosis was present, with a serum lactic acid concentration of 20.8 mEq/l; arterial blood gas measurements were *p*H 7.17, Pa_{O_2} was 67 mmHg (on $F_{I_{O_2}}$ of 0.5), and Pa_{CO_2} was 50 mmHg; the base deficit was −9 mmol/l, and serum bicarbonate was 19 mmol/l. Serum transaminases were LDH 3,201 IU/l (LDH_5 isoenzyme fraction was 66%) and AST 2,343 IU/l; alkaline phosphatase was 56 IU/l. The coagulopathy was treated with 5 units of fresh frozen plasma, 48 units of platelets, and 20 units of cryoprecipitate, as well as 7 units of packed red blood cells.

Because abdominal bleeding was considered as a possible cause of the low hematocrit, the patient underwent exploratory surgery. Laparotomy disclosed continuous oozing from soft abdominal tissues without evidence of macrovascular bleeding; approximately 1.5 l of blood was in the abdomen. Despite continuous intraoperative replacement of blood and blood products, the coagulopathy persisted, with a PT of 22.3 s, PTT of 33.7 s, platelet count of 70,000/mm^3, fibrinogen of 200 mg/dl, fibrin split products of greater than 40 μg/ml, and a factor VIII procoagulant protein (factor VIII:C) activity of 258% (normal, 50–150%). Additional intraoperative blood loss was assessed at 800 ml, which was replaced with 700 ml of whole blood; the coagulopathy was treated with an additional 24 units of platelets, 20 units of cryoprecipitate, and 4 units of fresh frozen plasma. Serum transaminases continued to increase postoperatively: LDH was 9,640 IU/l (LDH_5 isoenzyme fraction was 62%), AST was 2,409 IU/l, and alkaline phosphatase was 294 IU/l. Bilirubin was 5.6 mg/dl and increased at a slower rate than did the serum transaminases. The serum ammonia concentration was 168 mmol/l. During postoperative day 2, urine production decreased and creatinine increased; by postoperative day 3, the creatinine was 6.8 mg/dl and hemodialysis was indicated. However, liver function further deteriorated, and by postoperative day 7, bilirubin had increased to 20.8 mg/dl and the serum enzymes had decreased: LDH was 1845 IU/l, AST was 141 IU/l, and alkaline phosphatase was 144 IU/l (fig. 1, lower panel). A liver biopsy specimen showed extensive confluent coagulative necrosis of liver cells characterized by the presence of the ghost outline of nonviable hepatocytes. Hepatic intravascular thrombosis was not seen. The histopathology was most consistent with ischemic liver necrosis. Liver, renal, and respiratory function further deteriorated, and on postoperative day 10, all supportive measures were withdrawn, and the patient died.

Discussion

Acute ischemic hepatitis is caused by poor hepatic perfusion and most often is associated with acute heart or circulatory failure.[1,2] Acute ischemic hepatitis may also be caused by regional impedance of blood flow to the liver, specifically after celiac axis occlusion during pancreaticoduodenectomy.[9] Although the definitive diagnosis of AIH is established by histologic evidence of acute centrilobular hepatocyte necrosis in patients with an acute increase in the serum concentration of hepatic enzymes,[3] liver biopsy increases the risk of bleeding, especially if the patient already has a coagulopathy.[4] Therefore, it is acceptable to make a diagnosis of AIH based solely on clinical and biochemical criteria, including (1) presence of a clinical setting in which AIH may occur (hypotension or low cardiac output); (2) typical increase in serum transaminases, specifically an acute increase in hepatic LDH (isoenzyme LDH_5) and AST to more than 1,000 IU/l; and (3) a brief elevation of these serum transaminases that lasts from 3 to 11 days.[2,4,5] The serum bilirubin increases marginally, rarely to more than four times normal. Cholestasis is not a feature of AIH; therefore, alkaline phosphatase concentrations greater than twice normal are not seen. Prothrombin time is usually slightly high, but this increase does not correlate with the serum transaminase concentrations.[3,4] Mild-to-moderate renal failure often accompanies AIH, probably as part of the same hemodynamic disturbance that caused the AIH.[2]

Liver tissue oxygenation depends on both the saturated hepatic arterial blood and less saturated portal venous blood. The hepatic artery supplies 20–35% of the blood directed to the liver,[10] and interruption of this flow can cause liver ischemia. Both of our patients developed hepatic dysfunction after prolonged aortic cross clamping. In addition to a large aortic aneurysm, patient 1 had extensive atherosclerotic changes in the

celiac axis and superior mesenteric artery that may have chronically compromised the arterial blood flow and oxygen delivery to the liver. In addition, we cannot exclude the possibility that the aggressive administration of vasopressors in this patient contributed to the liver ischemia, because intrahepatic portal flow is regulated by the tone of postsinusoidal sphincters, which respond to α-agonists.[10] In patient 2, the aortic cross clamp was positioned just distal to the origin of the superior mesenteric artery and, at the end of surgery, the viability of a small-intestine loop was questionable; therefore, we believe that the clamp placed near the celiac axis somehow impeded the circulation to the liver, leading to immediate postoperative manifestation of acute liver failure. In addition, placement of abdominal retractors may have impeded the portal venous inflow, which supplies 65–80% of the blood sent to the liver.[10] Although normothermic total liver ischemia of up to 60 min is a safe technique for hepatic resections in healthy and cirrhotic livers,[11] both of our patients had undergone considerably longer ischemic periods. In summary, mechanical obstruction of arterial (cross clamp) and venous (surgical retractors) flows to the liver, superimposed on already compromised oxygenation caused by chronically (atherosclerotic lesions) or acutely (hypotension or vasopressors) decreased hepatic blood flow, possibly resulted in ischemia-induced liver dysfunction.

The clinical events during aortic aneurysm repair in our patients, and the consequent changes in the liver function tests (fig. 1), are consistent with the diagnosis of AIH. In patient 1, the biochemical signs of liver dysfunction were discovered by the end of surgery, indicating that the liver, excluded from the circulation for 95 min, suffered severe intraoperative ischemia. In our patient 2, the ischemic mechanism is less clear, but the biochemical signs of liver dysfunction, as well as coagulopathy, were diagnosed immediately postoperatively. In this patient, the high concentration of plasma factor VIII:C indicates that the liver failure was a dominant pathology. Factor VIII, synthesized in liver and endothelial cells, increases during hepatic failure.[12] This increase may be a result of poor clearance, caused by suppressed hepatic reticuloendothelial function, continuous extrahepatic synthesis of factor VIII, or reduced hepatic synthesis of protein C, a potent inhibitor of activated factor VIII.[12] At the same time, however, factor VIII:C would have been reduced if the dominant pathology were a consumptive coagulopathy, such as DIC.[13] Postoperatively, in both patients, the PT, rather than PTT, increased. This increase in PT also indicates a primary liver injury because, in DIC, the PTT is a more sensitive test of altered coagulation than PT and is the first to be prolonged.[13] Increased concentrations of D-dimer and fibrin split products, which were detected in our patients, are not unique indicators of DIC, and they increase in both liver[14] and acute renal failure.[15]

The clinical picture in our two patients was characterized with liver dysfunction and coagulopathy. Disseminated intravascular coagulation or subclinical forms of DIC are recognized as complications in patients with aortic aneurysms,[16–19] but AIH has never been clearly described during aortic aneurysm repair. The pathogenesis of DIC during aortic aneurysm repair has not been fully elucidated, but liver ischemia has been implicated as a possible cause. The hepatic reticuloendothelial system plays a major role in clearing from the blood activated coagulation factors, thromboplastic material, and fibrin split products released into the blood from ischemic tissue beds[20,21]; thus, liver dysfunction can have an important role in disturbing this balance. Hickman and Potter[5] proposed that even a small increase in serum AST resulting from circulatory failure may reflect a decreased capacity for hepatic clearance. If the clearance of thromboplastic material released during aortic surgery is slowed because the liver is ischemic, conditions favorable for coagulopathy may be created. In patient 1, the DIC became clinically evident immediately after the aortic clamps were released, indicating that activated coagulation factors were acutely released from ischemic tissues, exceeding the clearing capabilities of the ischemic liver. Cohen *et al.*[22] induced moderate DIC in dogs by applying the supraceliac aortic clamp for 60 min, and induced severe DIC when the aorta was clamped for 90 min, but the authors did not perform liver function tests. Dinbar *et al.*,[23] however, found, in monkeys, that liver ischemia induced decreases of 10 and 63% in the platelet count at 1 and 4 h, respectively, after 60 min of liver ischemia. They demonstrated that consumption coagulopathy intensified after revascularization of the liver,[23] which is consistent with the finding of intensive bleeding in our first patient after release of the aortic cross clamp.

Mortality in patients with AIH is high. Hickman and Potter[5] described 29 patients with circulatory failure who developed AIH, with an overall mortality rate of 58.6%. Gibson and Dudley[4] reported 47% mortality in 17 patients with AIH associated with cardiac disease.

In both studies, no patient died from AIH; rather, the death was attributed to underlying diseases, which indicates that an excessive rise in serum LDH and AST concentrations resulting from hepatic ischemia is a sign of poor prognosis. Therefore, the mortality from AIH largely reflects the severity of the associated systemic illness. The AIH may be either self limited, resulting in fast recovery (patient 1), or may progress to multisystem organ failure resulting in death (patient 2). The "normalization" of serum transaminases in patient 2 (fig. 1, lower panel) reflects massive liver necrosis that was confirmed by a biopsy specimen.

In conclusion, patients undergoing aortic aneurysm repair perioperatively may develop liver ischemia, which can result in a clinical picture consistent with AIH. We believe that the clinical course and biochemical changes in our patients may be explained by interrupted or decreased blood supply to the liver (supraceliac aortic cross clamping, surgical compression of the celiac axis and portal venous inflow, use of vasopressors, or intraoperative hypotension). During aortic aneurysm repair, a mild form of AIH is probably more common than is generally believed; when it is mild, AIH is not diagnosed as a distinct condition, but when severe, it is usually included in a broad diagnosis of multisystem organ failure.

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References

1. Kisloff B, Schaffer G: Fulminant hepatic failure secondary to congestive heart failure. Am J Dig Dis 21:895–900, 1976

2. Gitlin N, Serio KM: Ischemic hepatitis: Widening horizons. Am J Gastroenterol 87:831–836, 1992

3. Rawson JS, Achord J: Shock liver. South Med J 78:1421–1425, 1985

4. Gibson PR, Dudley FJ: Ischemic hepatitis: Clinical features, diagnosis and prognosis. Aust NZ J Med 14:822–825, 1984

5. Hickman PE, Potter JM: Mortality associated with ischaemic hepatitis. Aust NZ J Med 20:32–34, 1990

6. Scott J, Humphreys DR: Dissecting aortic aneurysm and disseminated intravascular coagulation. Br Med J 1:24, 1977

7. Micallef-Eynaud PD, Ludlam CA: Aortic aneurysms and consumptive coagulopathy. Blood Coagul Fibrinolysis 2:477–481, 1991

8. Griffith LD, Billman GF, Daily PO, Lane TA: Apparent coagulopathy caused by infusion of shed mediastinal blood and its prevention by washing of the infusate. Ann Thorac Surg 47:400–406, 1989

9. Bull DA, Hunter GC, Crabtree TG, Bernhard VM, Putnam CW: Hepatic ischemia, caused by celiac axis compression, complicating pancreaticoduodenectomy. Ann Surg 217:244–247, 1993

10. Kang Y: Anesthesia for liver transplantation. Anesth Clin N Am 7:551–580, 1989

11. Huguet C, Gavelli A, Chieco PA, Bona S, Harb J, Joseph JM, Jobard J, Gramaglia M, Lasserre M: Liver ischemia for hepatic resection: Where is the limit? Surgery 111:251–259, 1992

12. Langley PG, Hughes RD, Williams R: Increased factor VIII complex in fulminant hepatic failure. Thromb Haemost 54:693–696, 1985

13. Schmaier AH: Disseminated intravascular coagulation: Pathogenesis and management. Intensive Care Med 6:209–228, 1991

14. Carr JM: Disseminated intravascular coagulation in cirrhosis. Hepatology 10:103–110, 1989

15. Gordge MP, Faint RW, Rylance PB, Ireland H, Lane DA, Neild GH: Plasma D dimer: A useful marker of fibrin breakdown in renal failure. Thromb Haemost 61:522–525, 1989

16. Fine NL, Applebaum J, Elguezabal A, Castleman L: Multiple coagulation defects in association with dissecting aneurysm. Arch Intern Med 119:522–526, 1967

17. Collins GJ Jr, Rich NM, Scialla S, Andersen CA, McDonald PT: Pitfalls in peripheral vascular surgery: Disseminated intravascular coagulation. Am J Surg 134:375–380, 1977

18. Fisher DF Jr, Yawn DH, Crawford ES: Preoperative disseminated intravascular coagulation associated with aortic aneurysms: A prospective study of 76 cases. Arch Surg 118:1252–1255, 1983

19. Mulcare RJ, Royster TS, Phillips LL: Intravascular coagulation in surgical procedures on the abdominal aorta. Surg Gynecol Obstet 143:730–734, 1976

20. Fletcher AP, Biederman O, Moore D, Alkjaersig N, Sherry S: Abnormal plasminogen-plasmin system activity (fibrinolysis) in patients with hepatic cirrhosis: Its cause and consequences. J Clin Invest 43:681–695, 1964

21. Deykin D: The role of the liver in serum-induced hypercoagulability. J Clin Invest 45:256–263, 1966

22. Cohen JR, Angus L, Asher A, Chang JB, Wise L: Disseminated intravascular coagulation as a result of supraceliac clamping: Implications for thoracoabdominal aneurysm repair. Ann Vasc Surg 1:552–557, 1987

23. Dinbar A, Rangel DM, Fonkalsrud EW: Effects of hepatic ischemia on coagulation in primates: Application to liver transplantation. Surgery 68:269–276, 1970