Mesenteric Venous Thrombosis as Sole Complication of Decompression Sickness

STANFORD L. GERTLER, MD, JOSEPH STEIN, MD, THOMAS SIMON, MD, and KATSUMI MIYAI, MD

Mesenteric venous thrombosis is rare in healthy individuals. A variety of underlying conditions including several hypercoagulable states have been associated with mesenteric venous thrombosis (1). We present a case of a professional diver who developed extensive small bowel venous infarction after a dive. The patient had a history of phlebitis and pulmonary embolism associated with diving but no identifiable coagulopathy. In decompression sickness, a series of chemical and vascular events are initiated which produce tissue ischemia and results in venous infarction (2). This report is the first description of mesenteric venous thrombosis as the sole manifestation of decompression sickness.

CASE REPORT

A 27-year-old, white, male professional scuba diver was seen on November 25, 1981, because of abdominal pain of 36 hr duration.

The patient was in good health and working as a diver until September 1978. At that time, following a dive, he was admitted to San Diego Naval Regional Medical Center with edema, erythema, and a palpable cord in the right calf. A venogram was negative for deep venous thrombosis and his symptoms and signs resolved on treatment with aspirin, heat, and elevation. In June 1979 he presented again following a dive, complaining of pleuritic chest pain and hemoptysis. Chest x-ray showed a right lower lobe infiltrate with blunting of the right costophrenic angle. Ultrasound revealed no pleural effusion. Arterial blood gases on room air were pH 7.48, P^aO_2 85, and P^aCO_2 34. Perfusion scan with ^{99}Tc-labeled macroaggregated albumin showed multiple defects, one of which was unmatched on ^{133}Xe ventilation scan. He was treated with intravenous heparin infusion for two days in hospital followed by coumadin for 6 months, without recurrence of his symptoms.

Two days prior to admission he made a dive in cold water using surface supplied air to a depth between 40 and 50 feet for 160 min. Twelve hours later he noted the gradual onset of diffuse, colicky, periumbilical pain. The pain was unaffected by bowel movements and positional change. He vomited clear material and had 3–4 loose bowel movements without blood. There was no pruritus, rash, paresthesias, arthralgias, dyspnea, or changes in his mental status.

On presentation to the San Diego Veterans Administration Medical Center, his temperature was 97.6° F, respirations were 22/min, blood pressure 150/80 mm Hg and pulse 60/min. He was in distress with severe abdominal pain. Examination of the abdomen showed normal bowel sounds, hyperresonance, moderate diffuse tenderness, guarding in all quadrants, but no rebound tenderness. Stool was brown and Heme-occult test negative. White blood cell count 11,100/mm^3, and the hematocrit was 46%. Urinalysis showed rare white blood cells. Electrolytes, coagulation studies, amylase, and liver tests were normal. Chest x-ray was normal and abdominal x-ray series showed small air fluid levels without dilated bowel loops, pneumatosis coli, or thumbprinting. Because of the possibility of decompression sickness causing his severe abdominal pain, he underwent recompression in a United States Navy double-lock chamber at 60 feet equivalent seawater pressure. He was treated with intravenous fluids. After 50 min his abdominal pain worsened and recompression was stopped. He was admitted to the San Diego Veterans Administration Medical Center and fluids were given.

Diarrhea persisted and on the second hospital day he vomited 500 cc of grossly bloody material. Upper gastrointestinal endoscopy showed minimal patchy erythema without an active bleeding lesion. The white blood cell

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From the Departments of Medicine and Pathology, University of California, San Diego, California.

Dr. Gertler is a NIH Research Trainee in Gastroenterology. Present address: Kaiser-Permanente Hospital, 441 N. Lakeview, Anaheim, California.

Dr. Stein's present address is: Division of Cardiology, Brigham and Women's Hospital, 75 Francis Street, Boston, Massachusetts.

Dr. Simon's present address is: University of Colorado Health Science Center, 4200 East Ninth Avenue, Denver, Colorado.

Address for reprint requests: Dr. Stanford L. Gertler, Division of Gastroenterology, University of California, San Diego, Medical Center, 225 Dickinson Street, San Diego, California 92103.

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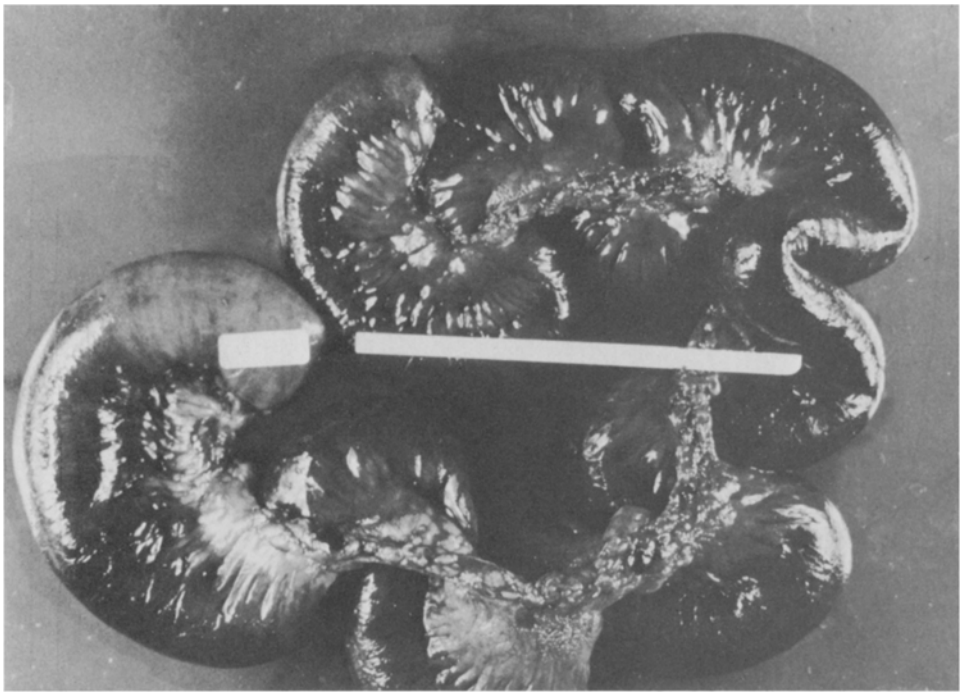

Fig 1. The resected bowel and mesentery were dark red in color. The intestinal wall and mesentery are markedly thickened due to edema and hemorrhage.

count was 20,500/mm³ with a shift to the left, and the hematocrit was 39.2%. Serum was amylase was normal. Intravenous pyelogram revealed no calculi. Exploratory laparotomy was performed because of the persistence of severe abdominal symptoms. Findings included approximately 1000 cc of bloody ascites and 130 cm of infarcted small bowel, beginning just proximal to the ligament of Tietz. The infarcted segment was resected, and after surgery he was given intravenous heparin, with an uneventful recovery.

Pathology. Pathologic examination revealed no air bubbles in the bowel on submersion. The affected segment of the bowel was dark red in color with marked thickening of the wall due to edema and hemorrhage (Figure 1). The mesentery was also thickened with edema and hemorrhage. On sectioning the mesentery, thrombi protruded from some of the transected veins. Mesentric arteries were patent. Microscopically, the affected regions of the bowel showed hemorrhage and partial necrosis of the mucosa. There was severe edema of the submucosa with marked engorgement of veins and foci of hemorrhage. Some of the submucosal veins contained fresh thrombi. Severe venous congestion and focal hemorrhage were also present in the muscularis propria and serosa. There was little or no inflammatory reaction (Figures 2 and 3).

Coagulation Studies. At the time of admission, the prothrombin time, partial thromboplastin time, and platelet count were normal. Subsequent evaluation included a normal antithrombin III level and activity, and a normal sucrose hemolysis test. The patient's plasma was also analyzed for protein C. Protein C is a vitamin K-dependent serine protease and is a potent *in vitro* anticoagulant and an *in vivo* profibrinolytic agent (3, 4). Since the patient was taking coumadin at the time of the assay, a ratio of protein C to factor X was performed. Levels of both proteins are decreased by coumadin, but the ratio remains at unity in normal controls as it was in our patient (4).

DISCUSSION

Mesenteric venous thrombosis of the small intestine developed in a professional diver. The patient presented with severe abdominal pain and diarrhea and then developed massive upper gastrointestinal bleeding. The writhing pain suffered by the patient in the absence of peritoneal findings on examination was suggestive of acute mesenteric insufficiency. Rapid emptying of the bowel is characteristic of superior mesenteric artery embolism but vomiting or diarrhea is also seen in mesenteric venous thrombosis. Blood in the stool is typical in acute mesenteric infarction, but generally there is no massive gastrointestinal bleeding (5–7). In this case, hematemesis was due to the proximal location of the infarction.

Diving-related medical complications result from barotrauma and from the vascular and chemical

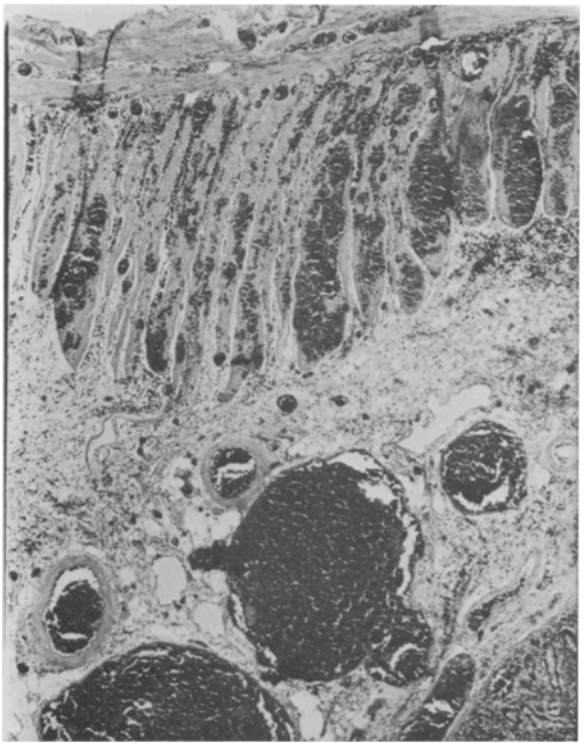

Fig 2. Micrograph showing the mucosa (upper left), submucosa, muscularis propria, and serosa. The mucosa is partially necrotic. The submucosa is thickened due to severe edema and venous congestion. Vascular congestion is also marked in the muscularis propria. Foci of hemorrhage are seen in all layers of the intestinal wall. (×145)

events involved in the pathogenesis of decompression sickness (8). Barotrauma during ascent results when compressed gas expands in enclosed spaces during reduction in environmental barometric pressure and may lead to pneumothorax or air embolism. In a similar manner, abdominal pain, belching, and passage of flatus during ascent is thought to be due to aerophagia and gaseous distension as the barometric pressure decreases (9). Gastric rupture as a manifestation of barotrauma has been recently reported (10). Alternatively, mesenteric ischemia due to obstruction of flow might produce abdominal pain. Vascular obstruction due to bubbles is felt to account for neurologic damage and pulmonary compromise associated with decompression sickness, but it has not been suggested as a cause of abdominal pain (10). We will review the pathophysiology of decompression sickness to examine factors which predispose to tissue ischemia and the relationship to our patient's mesenteric venous thrombosis.

During a scuba dive, the pressure of nitrogen increases in the inhaled air during descent. This results in a time-dependent solution of nitrogen in body fluids and tissues. Nitrogen can come out of solution and form bubbles during ascent if barometric pressure decreases too rapidly. Bubbles form predominately in the venous circulation, although in overwhelming decompression sickness bubbles may also be formed in the arterial circulation (11). The bubbles coalesce causing vascular obstruction. Although bubble formation is felt to be the central event in decompression sickness, a variety of other hematologic abnormalities have been described that produce a hypercoagulable state (2, 12, 13). Additional factors that may contribute to the pathogenesis of decompression sickness have been shown in experimental systems. These factors include interaction of the bubble surface with the cellular elements in the blood resulting in platelet microthrombi, sludging of RBC, and rouloux formation; increased vascular permeability resulting in hemoconcentration; and vascular obstruction due to fat emboli (2, 14–16).

The result of mechanical vascular obstruction due to bubbles, cellular elements, lipid aggregates, and hemoconcentration is venous obstruction and thrombosis. An important example of these vascular events is the spinal cord lesions associated with

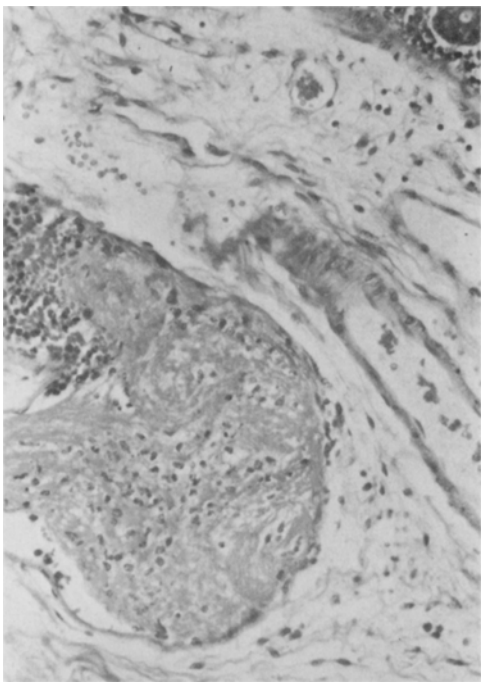

Fig 3. Fresh thrombi in one of the submucosal veins. (×540)

stasis and thrombosis of the epidural veins reported as a complication in decompression sickness (17). Despite the documentation of bubbles in the mesenteric circulation in overwhelming decompression sickness, mesenteric venous thrombosis has not been previously described. Published studies of postmorten changes in decompression sickness are limited and review of bowel findings is seldom noted (20–24). When visceral changes are reported, vascular bubbles and bowel wall congestion are described on gross examination. In contrast, careful documentation of vascular compromise is described in the brain, spinal cord, lungs, liver, spleen, kidneys, and long bones. Perhaps the extensive collateral circulation of the bowel protects it from vascular compromise in decompression sickness.

The onset of symptoms 12 hr after the dive and the lack of improvement with recompression in our patient are unusual in decompression sickness. Generally symptoms begin within 1 hr of surfacing. By 12 hr 97% of all cases have developed symptoms (25). In our patient, it is proposed that inadequate decompression during ascent from his dive resulted in asymptomatic venous bubble formation as reported in experimental animals and man (26). These bubbles triggered the hematologic events previously discussed and led to the patient's mesenteric venous thrombosis. The patient's symptoms and massive gastrointestinal hemorrhage were due to compromised bowel. The symptoms of mesenteric venous thrombosis may be of surprisingly long duration, suggesting that bowel infarction may be delayed after vascular compromise (1). In this case the period of recompression was shortened to 50 min due to his deteriorating condition. Treatment of decompression sickness consists of immediate recompression in a hyperbaric chamber with standard protocols lasting 2–6 hr. A return to hyperbaric conditions reduces bubble size, and breathing enriched oxygen produces a gradient of removal for nitrogen bubbles and alleviates tissue hypoxia. Recompression often produces rapid improvement in the symptoms of decompression sickness (25). Failure of our patient to improve with recompression also suggests that his symptoms were due to bowel ischemia or necrosis, although he may have improved with a longer period of recompression.

This unusual case emphasizes the need to consider mesenteric infarction in young patients with severe abdominal pain. Additionally, this case is a vivid example of the hematologic consequences of decompression sickness. While decompression sickness can be considered a hypercoagulable condition, our patient seems to have a unique predisposition to venous thrombosis in association with diving. He had a history of phlebitis and pulmonary embolism incurred following dives not associated with other symptoms of decompression sickness. The certainty of these diagnosis can be questioned, but it raises the possibility of an underlying thrombotic diathesis. Laboratory testing failed to detect a hypercoagulable state. Presently he is being treated with chronic coumadin therapy and has refrained from diving. No further thromboembolic episodes have occurred.

SUMMARY

A 27-year-old male commercial diver developed massive mesenteric venous thrombosis following a dive. Symptoms at presentation included abdominal pain and diarrhea. A severe upper gastrointestinal bleed developed. Exploratory laparotomy demonstrated 130 cm of infarcted small bowel. The pathophysiologic events in decompression sickness predispose to vascular obstruction and venous infarction. This patient had a past history of possible thrombophlebitis and pulmonary embolism associated with diving but no identifable coagulopathy.

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REFERENCES

1. Grendell JH, Ockner, RK: Mesenteric venous thrombosis. Gastroenterology 82:358–372, 1982
2. Philp RB: A review of blood changes associated with compression–decompression: Relationship to decompression sickness. Undersea Biomed Res 1:117–150, 1974
3. Stenflo J: A new vitamin K-dependent protein purification from bovine plasma and preliminary characterization. J Biol Chem 251:355–363, 1976
4. Griffin JH, Evatt B, Zimmerman TS, Kleiss AJ, Wideman C: Deficiency of protein C in congenital thrombotic disease. J Clin Invest 68:1370–1373, 1981
5. Donaldson JK, Stout BF: Mesenteric thrombosis. Am J Surg 29:208–217, 1935
6. Ottinger LW, Austen WG: A study of 136 patients with mesenteric infarction. Surg Gynecol Obstet 24:251–261, 1967
7. Kirscher PA: Occlusion of the mesenteric arteries and veins with infarction of the bowel. Mt Sinai J Med NY 21:307–317, 1955
8. Straus RH: Diving Medicine: Am Rev Respir Dis 119:1001–1023, 1979.

9. Lundgren CEF, Ornhagen H: Nausea and abdominal discomfort—possible relation to aerophagia during diving: an epidemiologic study. Undersea Biomed Res 2:155–160, 1975
10. Cramer FS, Heinbach RD: Stomach rupture as a result of gastrointestinal barotrauma in a scuba diver. J Trauma 22:23–240, 1982
11. Catchpole HR, Gersh I: Pathogenetic factors and pathological consequences of decompression sickness. Physiol Rev 27:360–397, 1947
12. Philp RB, Ackles KN, Inwood MJ, et al: Changes in the hemostatic system and in the blood and urine chemistry of human subjects following decompression from a hyperbaric environment. Aerosp Med 43:498–505, 1972
13. Philp RB, Schacham P, Gowdey CW: Involvement of platelets and microthrombi in experimental decompression sickness: Similarities with disseminated intravacular coagulation. Aerosp Med 42:494–502, 1971
14. Bove AA, Hollenbeck JM, Elliot DH: Changes in blood and plasma volumes in dogs during decompression sickness. Aerosp Med 435:49–55, 1974
15. Levin LL, Steward GJ, Lynch PR, Bove AA: Blood and blood vessel wall changes induced by decompression sickness in dogs. J Appl Physiol 50:944–949, 1981
16. Kitano M, Hayashi K: Acute decompression sickness—report of an autopsy case with widespread fat embolism. Acta Pathol Jpn 31:269–276, 1981
17. Hollenbeck JM, Bove AA, Elliot DH: Mechanisms underlying spinal core damage in decompression sickness. Neurology 25:308–316, 1975
18. Sillery RJ: Decompression sickness—a review of the literature and previously unreported histologic observations. Arch Pathol 66:241–246, 1958
19. Fryer DI: Pathological findings in fatal sub-atmospheric decompression sickness. Med Sci Law 30:110–123, 1962
20. Clay JR: Histopathology of experimental decompression sickness. Aerosp Med 34:1107–1110, 1963
21. Haymaker W, Johnston AD: Pathology of decompression sickness—a comparison of lesions in airmen with those in Caisson workers and divers. Milit Med 117:285, 1955
22. Waller SO: Autopsy features in scuba diving fatalities. Med J Aust 1:1106–1108, 1970
23. Rensselar HV: The pathology of the Caisson disease. Med Rec 40:141–150, 178–182, 1891
24. Brooks H: Caisson disease—the pathological anatomy and pathogenesis with an experimental study. Long Island Med J 1:14–150, 196–208, 1907
25. Rivera JC: Decompression sickness among divers: An analysis of 935 cases. Milit Med 126:314–334, 1964
26. Newman TS, Hall D, Lineweaver P: Gas phase separation during decompression in man. Undersea Biomed Res 3:121–131, 1976