



G83 Severe Hemorrhagic Pancreatitis in Forensic Autopsies: Report of Four Unequivocal Cases and Two Equivocal Cases

Wendy M. Gunther, MD, and Babatunde L. Stokes, MD*, Office of the Chief Medical Examiner, Tidewater District, 830 Southampton Avenue, Suite 100, Norfolk, VA 23510-1046

After attending this presentation, attendees will recognize factors from history, autopsy, and histology which may assist in identifying severe or hemorrhagic pancreatitis as a cause of death in forensic cases.

This presentation will impact the forensic science community providing case presentations showing how severe pancreatitis may be identified as a cause of death in forensic autopsies; differentiating unequivocal hemorrhagic pancreatitis from postmortem autolysis; and, providing consideration for difficult cases in which equivocal pancreatitis is a “red herring” which is not substantiated as the cause of death.

Pancreatitis is a natural disease which varies in severity from mild to fatal. Severe hemorrhagic pancreatitis may present at forensic autopsy, where it may represent the cause of death. Pancreatitis may be related to gallstones, to the abuse of alcohol, and to some prescription drugs. With this, toxicology is important in fully examining a death from pancreatitis. Pancreatitis may also represent a confounding or “red herring” contributor to death; it may also be suspected when it is not present.

A series of four forensic cases of fatal hemorrhagic pancreatitis from the Tidewater experience illustrates these findings, contrasted with cases which show how suspected pancreatitis (whether present or not) may present pitfalls to the medical examiner when assigning the cause of death.

Acute pancreatitis can arise from a variety of etiologic factors, but in most cases the specific cause is unknown. In some instances chronic alcoholism or toxicity from some other agent, such as glucocorticoids, thiazide diuretics, or acetaminophen, can bring on an acute attack of pancreatitis. In about half the patients a mechanical obstruction of the biliary tract is present, usually because of gallstones in the bile ducts. Viral infections also can cause an acute inflammation of the pancreas. Acute hemorrhagic pancreatitis is an acute inflammation of the pancreas accompanied by purulent peritoneal fluid, the formation of necrotic areas on the surface of the pancreas and in the omentum, and, frequently, also accompanied by hemorrhages into the substance of the gland. Some of the postmortem findings are discussed and potential pitfalls in diagnosing acute hemorrhagic pancreatitis on postmortem examination.

Case 1: EL, a 32-year-old male with a history of hypertension and chronic alcohol use complained of recent onset abdominal pain associated with nausea and vomiting was found unresponsive in bed. He was pronounced dead in the emergency department. Medications included hydrocodone, cetirizine, sildenafil citrate, and metoprolol. Internal examination revealed red discoloration and hemorrhage into an enlarged and partially necrotic pancreas, distended pancreatic duct with inspissated material, 200cc of purulent peritoneal fluid, hepatomegaly (4,000g), and cardiomegaly (438g). Histology revealed extensive acute inflammation with patchy necrosis superimposed on diffuse fibrosis, inspissated material in the ducts, and peripancreatic fat necrosis. The liver showed marked steatosis. The cause of death was due to hemorrhagic pancreatitis due to acute and chronic alcohol use.

Case 2: EW, a 42-year-old male with history of chronic alcohol and recurrent chronic abdominal pain unrelieved by over the counter stomach relief medications became incoherent before having a witnessed collapse. He died in the emergency department. On internal examination, the pancreas has multifocal areas of hemorrhage, partially necrotic, a pseudocyst in the head, inspissated material in the ducts, and peripancreatic fat necrosis and saponification. There is 200cc of purulent peritoneal fluid, marked fatty change of the liver, and mild splenomegaly (184g). Histology reveals extensive mixed inflammation, necrosis, pseudocyst, severe fibrosis (especially in the tail), and hemorrhage of the pancreas. The liver shows marked steatosis and regenerating nodules with severe chronic inflammation. The cause of death is hemorrhagic pancreatitis due to chronic alcoholism.

Case 3: SF, a 66-year-old female with a history of chronic alcohol use and gallstones was hospitalized for a subdural hematoma associated with an unwitnessed fall. She died approximately two months later while in a rehabilitation center. On internal examination, the head and body of the pancreas were firm with inspissated material in the main duct and the tail of the pancreas contained an abscess surrounded by necrosis. There was also six liters of ascites, severe jaundice, cardiomegaly (500mg), splenomegaly (327g), gallstones, and a fatty liver. Histology showed broad tract of fibrosis in the pancreas with amorphous cellular material in the ducts and advanced necrosis with saponification. The cause of death was attributed to multi-organ failure due to acute pancreatitis.

Case 4: MC, a 55-year-old male with a history of gastroesophageal reflux disease, hyperlipidemia, and hypertension complains of abdominal pain for a day associated with vomiting and diarrhea before being found dead on the living room floor in his home by his mother. Medications include fenofibrate. Internal examination shows severe hemorrhage into the pancreas with focal areas of necrosis, moderate amount of retroperitoneal hemorrhage, 400cc of serosanguinous fluid in peritoneal cavity, 100c of serosanguinous fluid in the right pleural cavity, and peripancreatic necrosis. Histology reveals diffuse fibrosis of the pancreas with multifocal areas of hemorrhage. Toxicology is currently pending. The cause of death was attributed to hemorrhagic pancreatitis. (however, this is a recent case that hasn't been officially signed out yet).



Pathology Biology Section - 2012

Case 5: LW, a 33-year-old female with a history of pancreatitis, hypertension, gout, foot pain treated with tramadol and recent shortness of breath was found unresponsive at home and died in the emergency department despite resuscitation. Her medications included tramadol, temazepam, and zolpidem. On internal examination, the pancreas was unremarkable but there was hepatosplenomegaly (1854 and 217g), cardiomegaly (495g), pulmonary edema (1579g combined weight), small left lower lobe pulmonary embolus, and a deep vein thrombosis in the right lower extremity. Histology showed a small focus of bronchopneumonia. When toxicology showed elevated levels of hydrocodone (0.07mg/L) and tramadol (5.2mg/L), death was attributed to the combined respiratory depressive effects of the two drugs.

Case 6: PC, a 49-year-old institutionalized woman with a history of mental retardation, recurrent renal stones, and gastric ulcer complained for two days of abdominal pain. Her medications included valproic acid for seizure disorder. She was treated with morphine and found unresponsive in a chair hours later. On internal examination, the tail of the pancreas showed sharply demarcated patchy areas of hemorrhage, and histology revealed diffuse fibrosis of pancreas with patchy areas of necrosis and hemorrhage. When toxicology showed elevated levels of morphine (0.2mg/L) and thioridazine (0.9mg/L), death was deemed due to overdose, although abdominal pain was probably due to acute pancreatitis, which is a recognized complication of valproic acid administration.

In these six cases, hemorrhagic to purulent fluid was always present in the lesser sac when hemorrhagic pancreatitis was the unequivocal cause of death. It was not identified when pancreatitis was either absent, or not the primary cause of death. Other findings common to fatal cases included extensive neutrophilic inflammation, and extension into peripancreatic fat. The absence of these findings suggests prudence in waiting for the toxicology results to determine the cause of death.

Hemorrhagic Pancreatitis, Forensic Pathology, Cause of Death