

Case Report

Fatal Blunt Pancreatic Trauma and The Vertebral Anvil Mechanism: A Single-Case Forensic Autopsy Report

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A B S T R A C T

Background. Blunt pancreatic injury is uncommon, and fatal cases after unarmed assault are rarely documented with combined autopsy, histological and toxicological data. Compression of the gland against the lumbar vertebral bodies (the “vertebral anvil effect”) has been proposed as the mechanism of transection.

Objective. To describe the autopsy findings in a fatal blunt pancreatic injury after a reported assault and to assess whether a simple mechanical estimate supports vertebral compression as the mechanism.

Methods. This single-case forensic autopsy study followed the CARE (CAsE REport) guideline. A 21-year-old man was examined by Letulle en masse evisceration at police request. Injuries were mapped, specimens were screened by chromatography and mass spectrometry, and pancreatic histology was scored semiquantitatively. Impact energy and force were estimated from assumed inputs.

Results. A 12.0 × 8.5 cm epigastric contusion and forearm abrasions were present. The pancreas was transected at the neck–body junction with main-duct disruption and a retroperitoneal hematoma of about 350 mL; a splenic laceration with about 120 mL of hemoperitoneum was also found. Toxicology was negative. Histology showed diffuse acinar necrosis, fat necrosis and neutrophilic infiltration. The estimate gave 53–212 J and 650–2,570 N per blow; the number of blows was unknown and no sourced tissue threshold was available.

Conclusion. The findings are consistent with death from traumatic pancreatic transection with hemorrhage and secondary necrotizing pancreatitis after blunt epigastric force. The mechanical estimate is illustrative only; the mechanism is inferred from the anatomy and injury pattern. One case cannot establish the vertebral anvil effect or the survival interval.

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1. Introduction

Blunt abdominal trauma caused by interpersonal violence is a recognized cause of preventable death.^{1,2} Injury to the pancreas is uncommon among blunt abdominal injuries, but it carries substantial morbidity and mortality, particularly when the main pancreatic duct is disrupted or other organs are injured.^{2,3} Diagnosis is often delayed because the gland lies deep in the retroperitoneum, abdominal wall signs may be slight, and early imaging can be unremarkable.^{3,4}

The pancreatic neck and body cross in front of the L1–L2 vertebral bodies, the aorta and the superior mesenteric vein. A localized anteroposterior force can therefore compress the gland against the spine, which is the classical explanation for transection at the neck and body and is often called the “vertebral anvil effect”.^{2,3} Once the main duct is disrupted, pancreatic enzymes can escape into the retroperitoneum and may digest parenchyma, fat and vessel walls and trigger a systemic inflammatory

response.^{5–8} Case reports describe isolated pancreatic injury after blunt trauma in adults and children.^{9–12}

Most published reports of blunt pancreatic injury are clinical or surgical case reports on diagnosis and management.^{13,14} Forensic autopsy reports of pancreatic transection after unarmed assault that combine macroscopic, histological and toxicological findings with an explicit mechanical argument are scarce.

The aim of this report is to describe the autopsy, histological and toxicological findings in one fatal case of blunt pancreatic transection after a reported assault, to state what these findings do and do not show about the cause of death and the mechanism, and to test with an explicit calculation whether a vertebral compression mechanism is mechanically plausible.

2. Methods

2.1. Case identification and setting

This is a single-case forensic autopsy study (case report) prepared according to the CARE (CAse REport) guideline.¹⁵ The autopsy was performed in November 2022 at the Installation of Forensic Medicine and Medicolegal Studies, dr. Soedono Regional General Hospital, Madiun, East Java, Indonesia. According to the police request and witness accounts, a 21-year-old male student was struck repeatedly on the abdomen by a group of 13 people in a student residential area, lost consciousness, was taken to hospital by emergency medical services and was pronounced dead shortly after admission. The number of assailants, the use of closed fists and the location are taken from the police record and are reported as stated there.

2.2. Statutory authorization, ethics and consent

The autopsy and the analyses were performed on a written request from the district police (Kepolisian Resor) of the Indonesian National Police (POLRI) under Articles 133 and 134 of the Indonesian Criminal Procedure Code (KUHP).¹⁷ The request covered external and internal examination, collection of organs and specimens, toxicology and histology to establish the cause, mechanism and manner of death. The study was approved by the CMHC Research Center, Indonesia (approval number 2022/0724) and followed the Declaration of Helsinki.¹⁸ Written informed consent for publication of the autopsy findings and photographs was obtained from the legal next of kin. Identifying details were removed from the text and the photographs.

2.3. External examination

Stature, body weight and body mass index (BMI, weight in kg divided by the square of height in m) were recorded. Livor mortis and rigor mortis (jaw, neck and limb joints) were recorded to estimate the postmortem interval (PMI), defined here as the time since death at the start of the autopsy.¹⁹ The whole body surface was examined with an ABFO No. 2 scale, and each injury was recorded by site, size (length × width, cm), pattern (bruise, confluent ecchymosis, abrasion or laceration), margins and tissue appearance.

2.4. Autopsy technique and injury grading

Internal examination used the Letulle en masse evisceration technique¹⁹ after an anterior midline incision from the chin to the pubic symphysis. The organ block (tongue, larynx, trachea, lungs, heart, esophagus, stomach, duodenum, pancreas, liver, spleen, intestines, kidneys and pelvic organs) was removed as one unit and examined on the table. The gastrocolic ligament was divided to expose the pancreas, which was examined from head to tail. Free blood in the retroperitoneum and the peritoneal cavity was aspirated and measured in calibrated cylinders. Pancreatic injury was graded with the 1990 American Association for the Surgery of Trauma Organ Injury Scale (AAST-OIS):²⁰ Grade I, minor contusion or hematoma without duct injury; Grade II, minor laceration without duct injury; Grade III, distal transection or parenchymal injury with duct injury; Grade IV, proximal transection or parenchymal injury involving the ampulla; Grade V, massive disruption of the pancreatic head. Associated injuries of the spleen, diaphragm, stomach, colon, mesentery, lungs and heart were recorded.

2.5. Histopathology

Sections were taken from the pancreatic head, neck, body and tail, peripancreatic fat, the anterior-superior left hemidiaphragm, spleen, lungs, liver, myocardium and kidneys. Blocks were fixed in 10% neutral-buffered formalin for 48 hours, processed routinely, embedded in paraffin, cut at 4 µm and stained with hematoxylin and eosin (H&E). Masson's trichrome was used to assess collagen and the von Kossa stain to identify calcium deposits within fat necrosis. Slides were examined by light microscopy (Olympus BX53) at 40× to 400× magnification. Acinar necrosis, cytoplasmic lysis, interstitial hemorrhage, vessel wall necrosis, neutrophilic infiltration, fat necrosis and ductal epithelial damage were scored on a 4-point semiquantitative scale (0, absent; 1, mild, <25% of the examined tissue; 2, moderate, 25%–50%; 3, marked, 51%–75%; 4, severe, >75%) in ten randomly selected high-power fields per block. Preservation of apical zymogen granules and basal basophilia was used as an indicator of acinar viability, and the interlobular septa were examined for vessel wall necrosis and thrombosis.

2.6. Toxicology

Specimens were collected before organ manipulation and kept under chain of custody: femoral blood (sodium fluoride preservative), central cardiac blood, vitreous humor, urine, bile, gastric contents and liver homogenate. Volatile alcohols (ethanol, methanol, isopropanol, acetone) were measured by dual-column headspace gas chromatography with flame ionization detection (HS-GC-FID). Illicit and psychotropic drugs, sedatives, opiates and synthetic cannabinoids were screened and quantified by liquid chromatography–tandem mass spectrometry (LC-MS/MS). Cyanide, organophosphates and carbamates were tested by colorimetric methods and gas chromatography–mass spectrometry (GC-MS). Results below the reporting cut-off are reported as not detected.

2.7. Mechanical estimate

A simple impulse–momentum and kinetic energy estimate was made to judge whether closed-fist blows could plausibly load the pancreas against the spine. It is an illustrative calculation from assumed inputs, not a measurement or a reconstruction. The assumed inputs were an effective striking mass (m) of 2.5–3.5 kg, an impact velocity (v) of 6.5–11.0 m/s and an impact duration (Δt) of 15–25 ms on a compliant abdominal wall. Kinetic energy per blow was calculated as $E_k = \frac{1}{2}mv^2$ and average impact force from the impulse–momentum relation as $F = mv/\Delta t$. It was assumed that 15%–25% of the energy reached the retroperitoneum, and contact pressure was calculated as $P = F/A$ for an assumed vertebral contact area (A) of 10–12 cm².

3. Results

3.1. History and external findings

The decedent was a 21-year-old male student with no documented pre-existing disease. The assault history is as given in Section 2.1. The clinical course is not available: admission findings, vital signs, resuscitation (including whether cardiopulmonary resuscitation was performed), fluids or blood products, and laboratory and imaging results are not recorded in the material available, and the times of the assault, admission and death are unknown, so the survival interval after injury is unknown.

The autopsy began about 7 hours after death. Height was 172.0 cm and weight 68.0 kg (BMI 22.99 kg/m²), with a

normal habitus and an abdominal subcutaneous fat thickness of 1.8 cm at the umbilicus. Rigor mortis was fully established in the jaw, neck and limbs, and livor mortis was purplish-red, fixed and non-blanching over the dorsal surfaces, consistent with a supine position after death. These signs correspond to an estimated PMI of 6–12 hours. The PMI is the time since death at autopsy and does not describe how long the decedent survived the injury.

External injuries were concentrated over the anterior torso and the forearms. A confluent purplish-red contusion with ecchymosis measuring 12.0 × 8.5 cm covered the epigastrium and extended into both hypochondria; its irregular, multifocal margin suggests several overlapping blunt impacts. Abraded contusions 1.5–3.5 cm in diameter lay on the extensor surfaces of both forearms and over the dorsal knuckles. The forearm lesions are consistent with defensive injury, although knuckle lesions can also arise from the decedent’s own blows. No cranial fracture, facial bone deformity or intracranial hemorrhage was recorded, and nothing suggested traumatic brain injury as a contributor to death.

3.2. Internal findings

After en masse evisceration, the pancreas was covered on both surfaces by abundant dark-red, non-clotted blood (Figure 1A; Table 1). A complete transverse transection of the parenchyma and the main pancreatic duct (duct of Wirsung) lay at the junction of the neck and body, in front of the L1–L2 vertebral bodies. The transection measured 4.5 cm across and passed through the full anteroposterior thickness of the gland (2.8 cm). Branches of the transverse

and dorsal pancreatic arteries that cross the neck were severed at the transection plane. The splenic artery and vein, which run along the superior and posterior margins of the body, were intact but showed severe adventitial contusion and perivascular suffusion. A retroperitoneal hematoma of about 350 mL filled the anterior pararenal space, extended laterally to the left renal hilum and inferiorly along the psoas sheath, and dissected into the root of the mesentery.

Associated injuries are summarized in Table 1. The spleen showed a linear laceration at the superior pole measuring 4.0 × 1.5 cm and 1.2 cm deep, with about 120 mL of perisplenic blood; no AAST grade was assigned to the splenic injury. The mesocolon showed confluent ecchymosis (8.0 × 5.0 cm), and the anterior–superior left hemidiaphragm showed a contusion with focal necrosis (5.0 × 3.5 cm). The pleural cavities contained no free fluid and there was no pneumothorax. The left lower lobe showed subpleural ecchymosis, edema and congestion. The heart weighed 380 g with biventricular dilatation, and testing under water showed no air in the four cardiac chambers. The stomach held about 150 mL of partly digested food with intact mucosa. The gallbladder and bile ducts are not described. Measured intra-abdominal blood loss was about 470 mL (350 mL retroperitoneal and 120 mL perisplenic), roughly 10% of an estimated blood volume of 4.8 L (68 kg × 70 mL/kg). It is described as moderate; the relative contributions of hemorrhage, the splenic injury and the pancreatic injury to death were not apportioned.

Table 1. Internal macroscopic findings at autopsy (Letulle en masse evisceration).

Organ / system	Gross findings	Measurements	Descriptive diagnosis
Pancreas and lesser sac	Abundant dark-red, non-clotted blood; complete transverse transection of parenchyma and main duct at the neck–body junction; severed transverse and dorsal pancreatic arterial branches; splenic vessels intact but contused	Gland 15.0 × 4.5 × 2.8 cm (dimensions to be labeled); retroperitoneal hematoma ~350 mL; weight not recorded	Traumatic pancreatic transection with hemorrhage; AAST-OIS Grade IV as reported (see text)
Spleen	Wrinkled, contracted capsule; linear parenchymal laceration at the superior pole; perisplenic blood	Laceration 4.0 × 1.5 cm, depth 1.2 cm; blood ~120 mL; weight not recorded	Splenic laceration with hemoperitoneum (AAST grade not assigned)
Stomach, colon, mesentery	Confluent mesocolic ecchymosis; vascular congestion of colon, stomach and mesentery; stomach with partly digested food, mucosa intact, no ulcer or erosion	Mesocolic ecchymosis 8.0 × 5.0 cm; gastric contents ~150 mL	Mesocolic contusion
Diaphragm	Hemorrhagic contusion with focal necrosis of the anterior–superior left hemidiaphragm	5.0 × 3.5 cm	Diaphragmatic contusion
Lungs and pleura	Left lower lobe subpleural ecchymosis, edema and congestion; no pneumothorax; pleural cavities empty	Left lung 520 g; right lung 580 g; free pleural fluid 0 mL	Pulmonary contusion and congestion (nonspecific)
Heart	Biventricular dilatation; no air in the four cardiac chambers on testing under water; coronary ostia patent	Weight 380 g (no reference range; wall thickness not recorded)	Biventricular dilatation (nonspecific)
Liver and kidneys	Liver with smooth capsule, no laceration or steatosis; kidneys with pale cortical surfaces, intact capsules, patent ureters	Liver 1,450 g; kidneys 140 g (left), 145 g (right)	No injury; renal pallor (nonspecific)

Notes: AAST-OIS, American Association for the Surgery of Trauma Organ Injury Scale. Measurements are as recorded at autopsy.

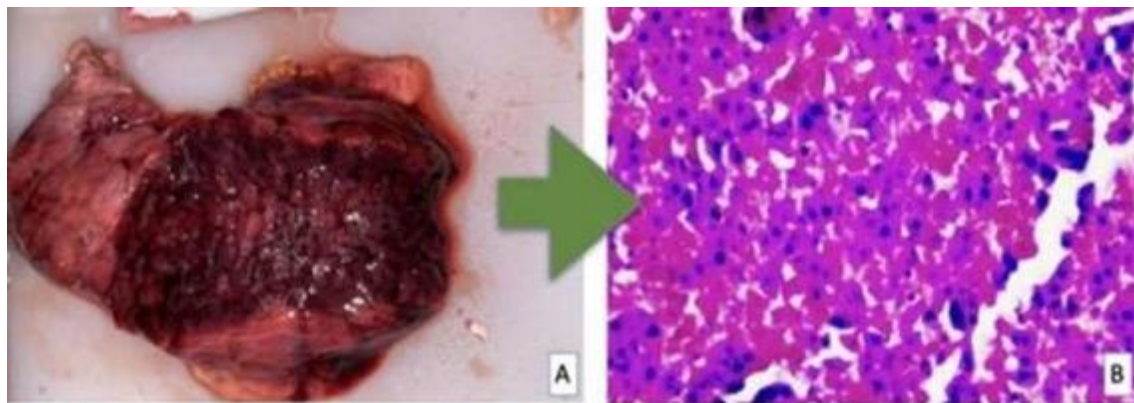


Figure 1. Pancreas at autopsy and histology. (A) Macroscopic view of the pancreas with dark-red blood over the surface and a transverse transection across the neck-body junction. (B) Hematoxylin and eosin stain, original magnification 400×: coagulative acinar necrosis, cytoplasmic lysis, loss of cell boundaries and interstitial hemorrhage.

3.3. Toxicology

No alcohol, drug or poison was detected in any specimen tested (Table 2). Ethanol was not detected (<0.010 g/dL) in femoral blood or vitreous humor, and methanol, isopropanol and acetone were not detected. LC-MS/MS was negative for amphetamine, methamphetamine, 3,4-methylenedioxymethamphetamine (MDMA), synthetic cannabinoids, delta-9-tetrahydrocannabinol (THC),

morphine, codeine, tramadol, fentanyl, methadone, cocaine, benzoylecgonine, benzodiazepines and barbiturates. Tests for cyanide, organophosphates and carbamates in gastric contents and blood were negative. A negative screen excludes the tested exogenous causes at the cut-offs used; it does not exclude biliary, metabolic or other causes of pancreatitis, and a negative blood ethanol does not exclude chronic alcohol use.

Table 2. Toxicological screening: analytes, specimens, methods, reporting cut-offs and results.

Analytes	Specimens	Method	Reporting cut-off	Result
Ethanol and volatile alcohols (methanol, isopropanol, acetone)	Femoral blood; vitreous humor	HS-GC-FID	0.010 g/dL (10 mg/dL)	Not detected (<0.010 g/dL)
Amphetamine-type stimulants (amphetamine, methamphetamine, MDMA)	Urine; cardiac blood	LC-MS/MS	10.0 ng/mL	Not detected
Cannabinoids (THC, THC-COOH, synthetic cannabinoids)	Urine; bile	LC-MS/MS	5.0 ng/mL	Not detected
Opiates and opioids (morphine, codeine, tramadol, fentanyl, methadone)	Femoral blood; urine	LC-MS/MS	10.0 ng/mL	Not detected
Cocaine and benzoylecgonine	Femoral blood; urine	LC-MS/MS	10.0 ng/mL	Not detected
Sedatives (benzodiazepines, barbiturates)	Liver homogenate; urine	LC-MS/MS	20.0 ng/mL	Not detected
Cyanide, organophosphates, carbamates	Gastric contents; cardiac blood	Colorimetric tests; GC-MS	0.050 mg/L	Not detected

Notes: GC-MS, gas chromatography-mass spectrometry; HS-GC-FID, headspace gas chromatography with flame ionization detection; LC-MS/MS, liquid chromatography-tandem mass spectrometry; MDMA, 3,4-methylenedioxymethamphetamine; THC, delta-9-tetrahydrocannabinol; THC-COOH, 11-nor-9-carboxy-THC. The submitted table gave cut-offs for the group, not for MDMA, synthetic cannabinoids, tramadol, fentanyl, methadone or barbiturates individually.

3.4. Histopathology

Pancreatic sections showed diffuse, confluent coagulative necrosis of the acinar tissue involving more than 75% of the examined fields (score 4), with loss of basophilia, cytoplasmic lysis, nuclear pyknosis, karyorrhexis and ghost-like cellular outlines (Table 3; Figure 1B). Near the transection plane the interstitium showed proteolysis and acinar dissociation, a pattern consistent with enzyme-mediated tissue digestion. Hematoxylin and eosin staining cannot show enzyme activity, and immunohistochemistry for trypsin activation was not performed.

Peripancreatic and retroperitoneal vessels showed marked hemorrhage with extravasated intact erythrocytes (score 4). Medium and small arterioles showed transmural fibrinoid necrosis with disruption of the internal elastic lamina, and small veins and capillaries contained microthrombi. Peripancreatic fat showed widespread fat necrosis with basophilic amorphous calcium deposits (fat saponification; score 4). Dense neutrophilic and monocytic

infiltrates surrounded necrotic acinar foci and ruptured vessels (score 3), and the ductal epithelium showed focal desquamation, basement-membrane rupture and periductal hemorrhage (score 4). Autolysis of the pancreas begins early after death and shares features with necrosis,²³ but intravital hemorrhage, fibrinoid vessel wall necrosis, thrombi and neutrophil infiltration are not explained by autolysis alone.²⁴ Because the survival interval is unknown, these changes are described as early injury-related changes and not as evidence of a particular interval.

The spleen showed fresh erythrocyte pooling along the trabeculae with neutrophil margination at the site of rupture. The diaphragm showed focal myofiber fragmentation, interstitial edema and coagulative necrosis next to the subphrenic space. Lung sections showed alveolar capillary congestion, intra-alveolar proteinaceous edema and leukostasis in the microcirculation, a nonspecific pattern.

Table 3. Histological scores by tissue compartment (hematoxylin and eosin).

Compartment	Findings	Score (0–4)	Descriptive interpretation
Pancreatic acinar tissue	Diffuse coagulative necrosis (>75% of examined tissue); cytoplasmic lysis; loss of basophilia; ghost outlines	4	Consistent with enzyme-mediated necrosis; autolysis not fully excluded
Peripancreatic vessels	Marked hemorrhage; erythrocyte extravasation; transmural fibrinoid necrosis with internal elastic lamina disruption; microthrombi	4	Vessel wall injury compatible with enzymatic digestion
Peripancreatic fat	Widespread fat necrosis; necrotic adipocyte ghosts; basophilic calcium deposits	4	Fat saponification
Perivascular infiltrate	Neutrophils and monocytes around necrotic foci; leukostasis	3	Consistent with an early vital reaction
Ductal epithelium	Focal desquamation; basement-membrane rupture; periductal hemorrhage	4	Duct injury at the transection plane

3.5. Mechanical estimate

With the assumed inputs (Section 2.7), kinetic energy per blow at the body surface was 53–212 J ($E_k = \frac{1}{2}mv^2$; $0.5 \times 2.5 \text{ kg} \times (6.5 \text{ m/s})^2 = 52.8 \text{ J}$ and $0.5 \times 3.5 \text{ kg} \times (11.0 \text{ m/s})^2 = 211.8 \text{ J}$), and the average impact force was 650–2,570 N ($F = mv/\Delta t$; $2.5 \text{ kg} \times 6.5 \text{ m/s} / 0.025 \text{ s} = 650 \text{ N}$ and $3.5 \text{ kg} \times 11.0 \text{ m/s} / 0.015 \text{ s} = 2,567 \text{ N}$). If 15%–25% of the energy reached the retroperitoneum, 8–53 J per blow was available there. Over the assumed contact area of 10–12 cm², the average force corresponds to a contact pressure of

0.54–2.57 MPa. The values in the submitted text (50–180 J, 450–900 N, 0.5–0.9 MPa and a cumulative 180–320 J) do not follow from these inputs and are not used. The number of blows was not recorded, so no cumulative energy can be derived, and no sourced failure threshold for pancreatic parenchyma is available for comparison. The calculation therefore shows only that a single blow could produce contact pressures of the order of 1 MPa over the vertebral contact area (Figure 2); it does not show that the threshold for pancreatic transection was exceeded.

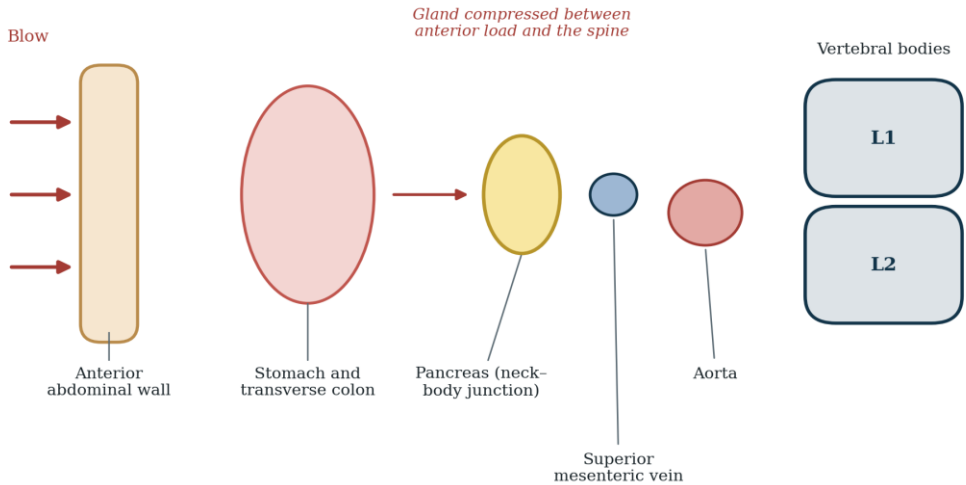


Figure 2. Proposed vertebral compression mechanism (schematic). Sagittal view, not to scale: a blow from the front drives the abdominal wall, stomach and transverse colon backward and compresses the pancreatic neck against the superior mesenteric vein, aorta and the L1–L2 vertebral bodies.

4. Discussion

4.1. Principal finding

In a young man after reported repeated blows to the abdomen, autopsy showed a complete pancreatic transection at the neck–body junction with main-duct disruption, retroperitoneal and perisplenic hemorrhage, an epigastric contusion, negative toxicology and diffuse pancreatic necrosis on histology. These findings are consistent with death from traumatic pancreatic transection with hemorrhage and secondary necrotizing pancreatitis, which is the diagnosis used in this report in place of the term “severe hemorrhagic pancreatitis” used in the submitted version. The proposed mechanism, compression of the gland against the spine, is inferred from the position of the transection and the overlying contusion; the mechanical estimate does not demonstrate it. The survival interval is unknown, and the relative weight of

hemorrhage and autodigestion in causing death cannot be stated.

4.2. Mechanism

The pancreatic neck and body lie in front of the L1–L2 vertebral bodies, the aorta and the superior mesenteric vein, and an anteroposterior force can compress the gland against these structures (Figure 2).^{2,3} In this case the 12.0 × 8.5 cm epigastric contusion, the position of the transection and the associated injuries of the spleen, diaphragm and mesocolon fit a localized anteroposterior force to the upper abdomen. The mechanical estimate in Section 3.5 does not add to this inference: its inputs are assumptions, the number of blows is unknown, and the energy reaching the retroperitoneum (8–53 J per blow) cannot be compared with a sourced tissue threshold. Adding the energies of separate blows and comparing the sum with a single-event threshold is not an accepted way to predict rupture, and it was not done here. The direct anatomical evidence of compression, hemorrhage or an

imprint on the prevertebral tissue and the anterior surface of L1–L2, and the state of the aorta, vena cava and superior mesenteric vein, is not reported.

After ductal disruption, activated pancreatic enzymes may digest parenchyma, fat and vessel walls.^{5–7} The histological pattern (acinar necrosis, fat necrosis with calcium deposits, fibrinoid arterial necrosis and neutrophil infiltration) is compatible with this process, and a systemic inflammatory response and organ dysfunction, as defined by consensus criteria,^{8,25} may have followed. Histology cannot show enzyme activity or the order of events, however. Hypocalcemia from fat saponification, lactic acidosis, hyperkalemia, kinin release and acute kidney injury may have contributed to death, but none of these was measured (no calcium, lactate, potassium, blood pressure or vitreous chemistry), so they are hypotheses and not findings. Pancreatic autolysis shares features with necrosis;²³ the vital-reaction features and an interval of about 7 hours before autopsy support but do not prove an antemortem process, and no immunohistochemical marker of vitality was used.

4.3. Comparison with published cases

Published reports of blunt pancreatic injury from low-energy blows in adults and children describe injury at the neck and body and delayed diagnosis because early signs are slight.^{4,9–12} In these clinical reports the patients lived long enough for imaging or surgery,^{13,14} whereas the present decedent died after an unknown interval and was examined at autopsy; the clinical reports therefore describe course and treatment, not the gross and microscopic findings given here. Autopsy series of blunt abdominal trauma describe the distribution of injuries by organ,¹ but we did not find one that matched the combination of pancreatic transection, histological scoring and toxicology in this case. Retropancreatic fat has been examined as a modifier of pancreatic injury severity on CT, a possibility this case cannot address (BMI 22.99 kg/m²; abdominal subcutaneous fat 1.8 cm).

4.4. Medicolegal implications

The findings support, but do not decide, the following forensic opinion: the cause of death is traumatic pancreatic transection with hemorrhage and secondary necrotizing pancreatitis, and the injury pattern is consistent with blunt force to the epigastrium as reported by the police. The manner of death rests on all the evidence available to the investigating authorities and the court. Negative toxicology excludes the tested toxic causes but not biliary, metabolic or other causes, so the gallbladder and bile ducts should be examined and reported in similar cases. The en masse technique allowed the pancreas, the hematoma and the splenic pedicle to be examined together, but one case cannot show that it is superior to the organ-by-organ (Virchow) technique;¹⁹ that would need a comparative series. The number of assailants and the contribution of any one of them cannot be derived from the autopsy, and questions of criminal liability belong to the court. For practice, we suggest that autopsy reports of blunt abdominal trauma record the position of any pancreatic transection relative to the superior mesenteric vein and the AAST-OIS edition used, the prevertebral findings, the survival interval and clinical course when known, and the histological scoring procedure with observers and agreement.

4.5. Limitations

This is a single case, so no generalization is possible. The survival interval, clinical course, resuscitation, number of

blows and scene details are unknown or come from secondary sources. The mechanical estimate rests on assumed inputs, was not measured or validated, and was not compared with a sourced tissue threshold. Postmortem imaging, immunohistochemistry, postmortem biochemistry and serial serum enzymes were not available, and the gallbladder, biliary tract and prevertebral findings are not reported. Histology was scored semiquantitatively on one scale without a stated agreement statistic, and autolysis cannot be fully excluded.

5. Conclusion

This single-case forensic autopsy documents a complete pancreatic transection at the neck–body junction with retroperitoneal and perisplenic hemorrhage and pancreatic necrosis after a reported blunt epigastric assault. The findings are consistent with death from traumatic pancreatic transection with hemorrhage and secondary necrotizing pancreatitis. Vertebral compression is a plausible mechanism, inferred from the injury pattern and not shown by the mechanical estimate. The survival interval and the relative contribution of hemorrhage and autodigestion remain unknown. Autopsy reports of similar cases should record the transection site relative to the superior mesenteric vein, the prevertebral findings, the survival interval and the histological scoring procedure.

Declarations

Use of generative artificial intelligence. None. The authors declare that no generative artificial intelligence tool was used in the conception, research, analysis, drafting, revision or preparation of this manuscript.

Author contributions. VAL: Conceptualization, Data curation, Investigation, Methodology, Project administration, Resources, Writing – original draft. SAP: Formal analysis, Investigation, Validation, Visualization. DM: Formal analysis, Methodology, Supervision, Writing – review & editing. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Conflict of interest. None. The authors declare no competing financial or non-financial interests.

Ethical approval. Approved by the CMHC Research Center, Indonesia (approval number 2022/0724), in accordance with the Declaration of Helsinki.

Consent for publication. Written informed consent for publication of the autopsy findings and photographs was obtained from the legal next of kin (Section 2.2).

Data availability. The raw histological scores and toxicology records are available from the corresponding author on reasonable request, subject to legal and ethical restrictions.

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