

CASE REPORT

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Histologic Evidence of Repetitive Blunt Force Abdominal Trauma in Four Pediatric Fatalities

ABSTRACT: In cases of acute fatal child abuse, certain injuries, including cutaneous blunt force trauma, skull fractures, subdural hematomas, intra-abdominal hemorrhage, and retinal hemorrhages are common and well described in the pediatric and forensic literature. These gross findings at autopsy, when taken into consideration with scene investigation and interviews with caregivers, may indicate both a clear manner and cause of death. In such cases, the discovery of additional pathologic changes attributable to older abusive injuries helps support a conclusion of death due to inflicted trauma. We discuss four cases of fatal child abuse in which acute blunt force abdominal trauma was the cause of death. In each of these cases, careful examination with proper sectioning and microscopy of select abdominal tissues revealed that the acute tissue trauma was superimposed on a background of older, healing injury. This older trauma was characterized by classic histologic elements of tissue repair, including fibroblast proliferation, early scar formation, increased vascularity, and hemosiderin-laden macrophages. Iron and trichrome stains were used to confirm the presence of hemosiderin and fibrosis in all four cases, but the recognition of fibroblast proliferation and a reactive vascular pattern was best seen on routine hematoxylin and eosin stains. The gross and microscopic autopsy findings, along with available investigative information, established the diagnosis of chronic physical abuse.

KEYWORDS: forensic science, chronic child abuse, repetitive trauma, blunt force trauma, homicide

Child abuse, or nonaccidental injury inflicted by caregivers, is a leading cause of morbidity and mortality in children (1–4). In cases of acute fatal child abuse, a complete forensic autopsy can reveal a constellation of classically described markers, including external blunt force trauma, subdural hematoma, abdominal injuries, and retinal hemorrhages (5–9). These autopsy findings, along with thorough investigation by law enforcement officials, can often clearly define the manner and cause of death (10). In such cases, the additional discovery of older abusive injuries supports a diagnosis of death secondary to deliberately inflicted trauma, because it demonstrates a pattern of chronic physical abuse. When investigative information is minimal or inconclusive, the pathologic identification of injuries occurring at two or more distinct times may represent the primary rationale for diagnosing repetitive, nonaccidental trauma.

We discuss four cases of fatal child abuse in which acute blunt force abdominal trauma was the cause of death; in each instance the manner of death was homicide. In all four cases, histologic findings demonstrated evidence of remote blunt force injuries in addition to acute trauma, reasonably establishing a diagnosis of chronic physical abuse.

Case 1

A 2-year-old, 25 lbs, girl was reportedly found unresponsive at a relatives' residence where she had been playing in their living room. She was transported to a local hospital where she was pronounced dead. At autopsy, external examination revealed multiple blunt force injuries including acute contusions to the back of the

head, frontal scalp, and chest. Internal findings included avulsion of the right pulmonary hilum, a 300 mL right hemothorax, a large liver laceration, and a 100 mL hemoperitoneum. Acute hemorrhage was present around the right adrenal gland and pancreas. Sections of the pancreas and acutely lacerated liver demonstrated multiple areas of fibrosis, with scattered mononuclear cells containing hemosiderin (Figs. 1 and 2).

Case 2

An otherwise healthy 2-year-old, 35 lbs, boy with no significant past medical history was dropped off at his Grandmother's house one afternoon. As per the report, he became increasingly lethargic, and sometime that evening was discovered unresponsive. He was transported to a local hospital and stabilized. Two days later he began to develop signs of hypoxic encephalopathy and was ultimately pronounced dead. An autopsy revealed areas of acute hemorrhage in the abdominal wall, and 300 mL of blood was present in the abdomen. The sources of the bleeding were soft tissues surrounding the head of the pancreas and the base of the small bowel mesentery. In addition, a laceration to the duodenum was detected. Sections from the pancreatic head showed the acute hemorrhage, but also revealed reactive fibroblast proliferation, as well as macrophages containing hemosiderin (Fig. 3).

Case 3

A 1-year-old, 19 lbs, girl was transported to a local Emergency Department by her mother. She was apneic and required immediate intubation. The child was briefly stabilized, but her condition rapidly deteriorated and she could not be resuscitated. An autopsy revealed a large midline tear involving the inferior surface of the liver with an associated 360 mL hemoperitoneum. Hemorrhage was present around the transverse colon, duodenum, and pancreas.

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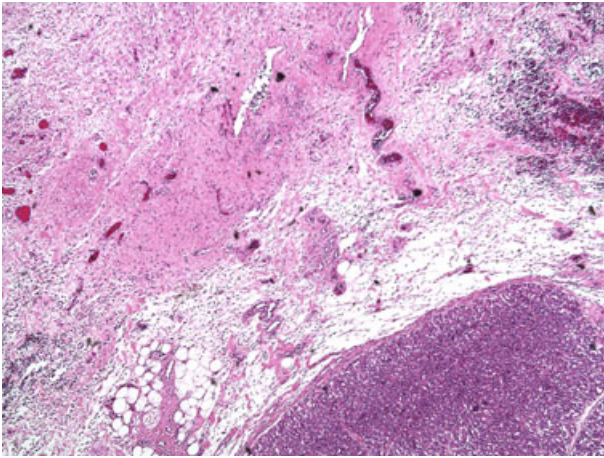


FIG. 1—This image shows the increased fibrosis (upper left) vascular proliferation, edema (center), and hemorrhage (upper right) in the connective tissue adjacent to the pancreas in sections of the retroperitoneum. (Case 1) H&E, 40 \times .

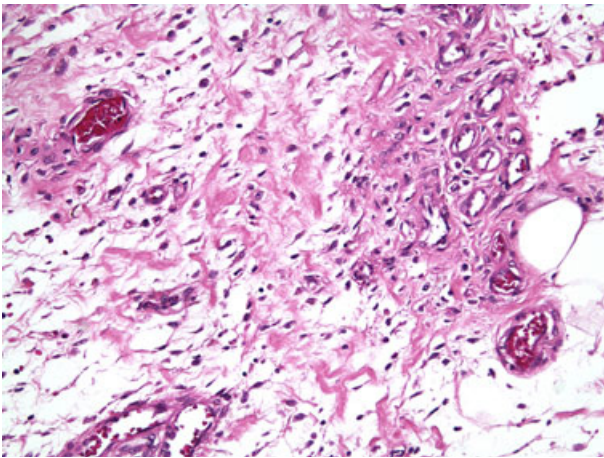


FIG. 2—The (reactive) vascular proliferation is best seen in the same sections from this case. It can be detected by looking for capillary proliferations adjacent to areas of edema. (Case 1, section of pancreatic head and retroperitoneum) H&E 200 \times .

Microscopic examination demonstrated acute hemorrhage in the liver parenchyma and in connective tissues surrounding the organs noted above (Fig. 4). Scar tissue formation was present in the hepatic parenchyma adjacent to the recent liver injury.

Case 4

An unresponsive 2-year-old, 15 lbs, girl was taken to a Community Hospital, where she was pronounced dead. The attending physician noted several ecchymoses on the head, chest, and feet. At autopsy a laceration of the duodenum was present next to the head of the pancreas, along with an associated 320 mL hemoperitoneum. In addition, several bruises of the abdominal wall, face, and torso were detected. In sections of the pancreas reactive fibroblast proliferation with scar formation and hemosiderin-laden macrophages were present (Figs. 5 and 6).

To assure that the healing changes observed in the tissue sections were likely due to physical injury, they were compared with sections taken from the same anatomic areas in similarly aged children

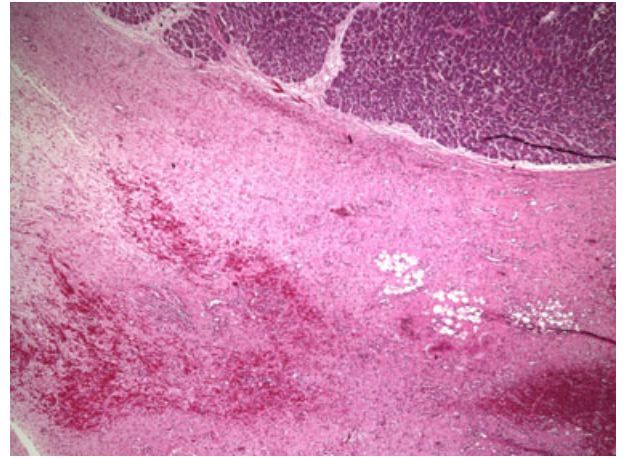


FIG. 3—Sections from the retroperitoneum show dense fibrosis. The lack of edema and dense collagen seen on these sections is indicative of organization and is >5 days old. We estimate this injury to be remote and on the order of “weeks to months” old. Furthermore, acute hemorrhages are seen in the scar tissue. Highlighting the fact that the tissues injured in the past are often re-injured in the acute fatal blunt force abdominal trauma. (Case 2, section of pancreatic head and retroperitoneum) H&E 100 \times .

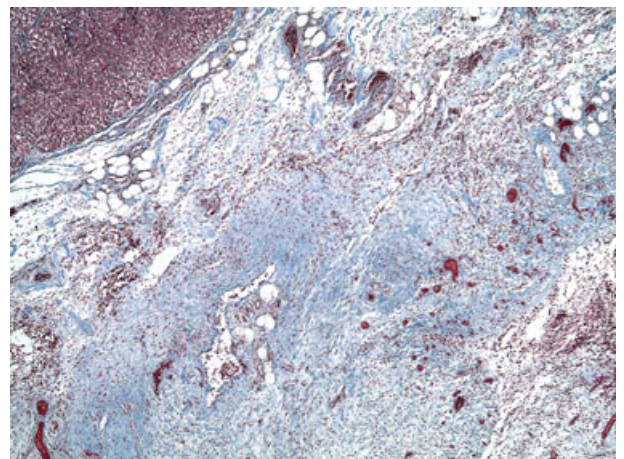


FIG. 4—On cases where the amount or deposition of fibrosis is irregular, trichrome stains will highlight the collagen formation. (Case 3, section of pancreatic head and retroperitoneum) Masson's Trichrome 100 \times .

who had died from natural causes, and who had no history of injury. Review of tissues from these comparison cases did not show evidence of tissue repair or injury. The healing changes observed in tissues from the reported cases are consistent with resolving physical injury, do not likely represent the sequelae of an undiagnosed, natural disease process, and were not present in the comparison cases. For these reasons, it is reasonable to conclude that the histologic findings in the reported cases resulted from prior physical injury.

Discussion

These cases illustrate several issues relevant to the investigation of deliberately inflicted fatal trauma in the younger pediatric population. The first involves how a diagnosis of fatal, nonaccidental trauma is established in an infant or toddler. While autopsy findings are usually the key element in a pediatric death investigation, they

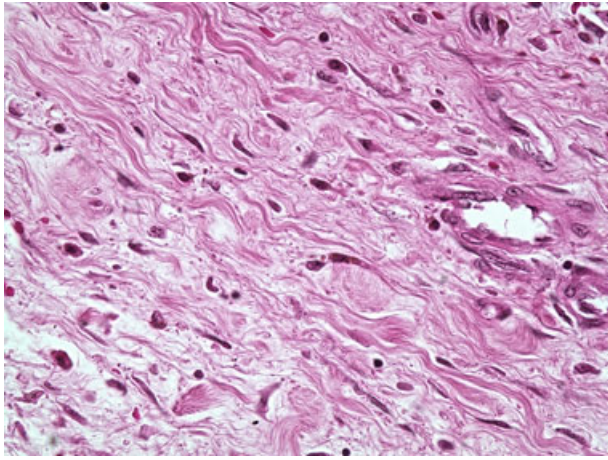


FIG. 5—Reactive myofibroblasts are cells with cigar-shaped nuclei and wavy cell processes extending from either end of the cell. These cells are responsible for the dense fibrosis which gives the sections a pink appearance at low power. (Case 4, section of pancreas and retroperitoneum) H&E 400 \times .

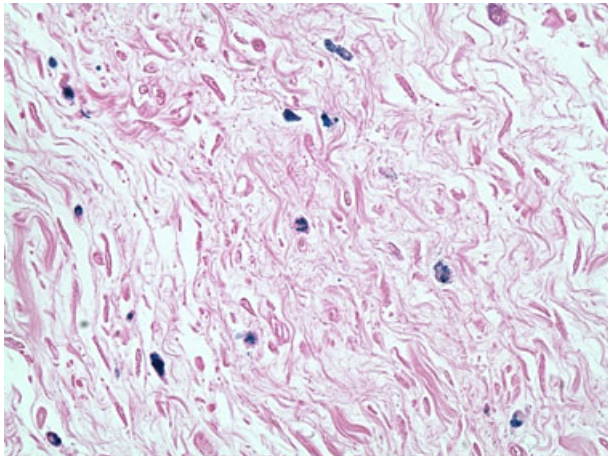


FIG. 6—Another marker of remote injury is the presence of hemosiderin-laden macrophages which respond to injury by cleaning up red blood cells and cellular debris. They are not always easily seen on H&E, but Prussian Blue stains will show their presence or absence in areas of fibrosis. (Case 4, section of pancreas and retroperitoneum) Iron, Prussian Blue Reaction 400 \times .

should always be considered in the context of pertinent investigative information. In this regard, maximizing the amount of available information is desirable. Any mechanism which would help the investigative process, including use of a multidisciplinary review team, should be considered. In some instances, investigative information generated by such a review may be critical in either establishing or ruling out abusive injury. Even when autopsy findings alone mandate a manner of homicide, it is the investigative findings that will likely address other relevant issues, such as the time and place of injury.

In some cases, investigative findings may be inconclusive, either because it is limited in volume, or is ambiguous. In such instances, pathologic findings which indicate multiple applications of significant force effectively tip the scales in favor of nonaccidental trauma. In any of the reported cases, given that they all demonstrated both acute and healing abdominal injuries, it would be unlikely that they: (i) had been accidentally injured at an earlier time,

and then accidentally re-injured in the same area just prior to death, (ii) had been accidentally injured at an earlier time, and then intentionally injured in the same area just prior to death, or (iii) had been intentionally injured at an earlier time, and re-injured accidentally in the same area just prior to death. Unless the investigative process favors one of the scenarios noted above, it is far more likely that both the healing and acute injuries were deliberately inflicted. Such an opinion would be strengthened when the acute abdominal injuries are accompanied by other markers of acute injury, as in Cases 1 and 4.

Another issue involves the role of histopathology in forensic cases. Although there has been recent discussion in the forensic pathology community regarding the utility of routine histopathology, these cases serve to demonstrate that microscopic tissue examination remains a vital and necessary part of the overall investigative process. A recent study by Molina et al. (11) showed that in 189 cases where histologic sections were taken of select organs, in only one case did the microscopic examination provide evidence to affect the cause of death that was detected strictly with gross examination. In all four of these cases, nothing detected with microscopic examination changed the cause or manner of death. Of the four reported cases, two (no. 2 and no. 3) showed gross findings suggestive of possible older injury. In the other two, older injury was not suspected grossly, and would have been missed if tissue microscopy had not been performed.

It is reasonable to question what role, if any, resuscitation may have played in the production of the acute injuries illustrated by these cases. First, it should be remembered (at least as far as these four reported cases are concerned) that any hypothetical resuscitation trauma would have to coincidentally involve a previously injured area. Second, cardiopulmonary resuscitation is not commonly associated with intra-abdominal injury. Third, when resuscitation is unsuccessful, it should not be associated with significant amounts of acute soft tissue hemorrhage. Finally and most importantly, the fact that resuscitation has to be performed begs the issue as to why it was needed in the first place. In other words, a full autopsy should reveal the underlying cause of death. If such a cause was unrelated to alleged resuscitation injuries, it should be readily diagnosed. For these reasons, it is unlikely that resuscitation caused or contributed to the acute injuries observed in these four cases.

In pediatric cases, blunt force abdominal trauma most commonly produces injuries involving the liver, spleen, kidneys, and pancreas (12). Past reports discuss injury to these organs and others in cases of pediatric blunt force abdominal trauma (5,6,13,14). These reports only address acute fatal blunt force abdominal trauma and none discuss evidence of prior healing injury to the same anatomic area. Based on our limited experience, we believe a significant proportion of infants and children who present at autopsy with deliberately inflicted acute blunt force abdominal trauma may have sustained similar episodes of trauma in the past. The exact prevalence of healing abdominal injuries in cases of fatal acute abdominal trauma is not known and will require ongoing research. For what it is worth, as initially identifying it in one case (no. 3), we have found it in over half of the subsequent cases of suspected acute, nonaccidental abdominal trauma. The reader is cautioned, however, that given the relatively small numbers of cases, our results may not be statistically significant.

The hallmark findings in each case were identified in microscopic sections of the acutely injured tissues and the retroperitoneum, particularly around the head of the pancreas. Evidence from other studies support our opinion that nonfatal blunt force abdominal trauma causes microscopic tears and hemorrhage to the organs secured within the retroperitoneum (4–6,12,13). The

cascade of tissue repair which ensues after nonfatal blunt force trauma is an orderly process that “patches” the injured parenchyma (14,15). The earliest repair begins approximately 24 h after injury as myofibroblasts and vascular endothelial cells begin proliferating (16). The granulation tissue which forms appears “loose” microscopically secondary to tissue edema. This tissue becomes more organized around post-injury day 3 when collagen deposition begins. The collagen, in concert with the reactive myofibroblasts, aids in contraction of the “patched” tissue and strengthens the once loose, edematous granulation tissue on post-injury day 5 to approximately day 30 (14–17).

A (reactive) myofibroblast (Fig. 5) has a basic spindle cell morphology with cytoplasmic processes which will soon aid in contraction of the tissue (16,18). The reactive myofibroblasts are accompanied by proliferating endothelial cells as seen in Fig. 2 from Case 1. These findings are most easily detected on routine hematoxylin and eosin stains.

The collagen deposition in the extracellular matrix is easily demonstrated on trichrome stains (Fig. 4, Case 3) and a Prussian Blue stain for Iron demonstrates numerous hemosiderin-laden macrophages cleaning up cellular debris at the sites of remote injuries (Fig. 6, Case 4).

Based on these findings, in pediatric autopsies involving inflicted acute blunt force abdominal trauma, we recommend taking specific histologic sections. These include the head of the pancreas, soft tissues of the surrounding retroperitoneum, and any acutely injured tissues. These should be examined to assess the presence or absence of hemosiderin-laden macrophages, granulation tissue, reactive myofibroblasts, edema, collagen fibrosis, or vascular proliferation. In light of the known timing of changes in the healing cascade, identification of specific changes may allow the forensic pathologist to ascertain whether or not the same areas which were acutely injured had potentially sustained prior trauma. Such findings, if present, may correlate with other indicators of chronic abuse, including cutaneous injuries, skeletal trauma, intracranial injuries, or malnutrition.

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