J Forensic Sci, July 2005, Vol. 50, No. 4
Paper ID JFS2004473
Available online at: www.astm.org

CASE REPORT

Melissa A. Pasquale-Styles,[1] *M.D. and Carl J. Schmidt,*[1] *M.D.*

Blunt Force Injury of the Abdomen Complicating Previously Undiagnosed Peliosis Hepatis in a 2-Year-Old Female

ABSTRACT: Peliosis hepatis is an abnormal accumulation of blood-filled lakes in the liver that is most commonly seen in adults and is generally associated with chronic wasting diseases, use of androgenic steroids or bacterial infection. Few cases have been reported in children. We report a case of a 2-year-old female with no past medical history who presented with homicidal blunt force abdominal injury. The autopsy revealed lacerations in the liver and previously undiagnosed peliosis hepatis.

KEYWORDS: forensic science, peliosis hepatis, liver, blunt force trauma, abdomen, homicide, child

The deceased was a 32-month-old black female who had no significant past medical history. She was driven to an emergency room by her mother who reported that she been coughing and had vomited prior to transport. The mother said the deceased became short of breath and then unresponsive in the car on the way to the hospital. In the emergency room, resuscitative measures were unsuccessful. Hospital personnel observed "bruises on the belly and other parts of the body" which were interpreted to be signs of child abuse.

Upon external examination, the body was that of a well-developed, well-nourished female child who measured 37.5 in. in length (50th to 75th percentile for height) and weighed 15 lb (90–97th percentile for weight) (1). The upper frenulum was torn, and multiple red-purple bruises (up to 5 cm in dimension) were on the back, right thigh and both legs. Internal examination revealed severe liver injury and associated hemoperitoneum of 500 mL. The surface of the right liver had a large, adherent blood clot and underlying patches of superficially lacerated liver parenchyma (Fig. 1). The left posterior liver had two lacerations measuring 1.0 and 3.0 cm, respectively, with shredding of the adjacent liver parenchyma. The inferior margin of the liver was torn, and Glisson's capsule was separated from the parenchyma throughout the right liver lobe by a partially clotted, subcapsular hematoma. The sectioned liver had red-brown parenchyma with diffusely distributed cyst-like spaces filled with blood ranging from 0.5 to 2.0 cm in greatest dimension (Fig. 2). Some of the spaces were filled with laminated blood clots.

Microscopic examination showed acute hemorrhage with intact red blood cells in areas of disrupted, injured liver parenchyma. Uninjured areas of liver had cyst-like spaces filled with blood (Fig. 3). Some of the spaces were lined by hepatocytes, and some spaces were lined by thin fibrosis and endothelium (Figs. 4 and 5). Some pools of blood had intact red blood cells, some had thin fibrin stranding, and some had well-formed, laminated blood clots indicating chronicity. The blood lakes were not within portal triads and had no associated biliary or vascular structures. The hepatocytes, sinusoids and portal triads were normal. The cause of death was determined to be blunt trauma to the abdomen, and the manner of death was homicide.

Discussion

Peliosis hepatis is an abnormal collection of pools of blood that are randomly distributed in the liver parenchyma. The pools may or may not communicate with the sinusoids, and they have no relationship with blood vessels or the biliary tree. Parenchymal and phlebectactic types have been described and are differentiated by the lining of the blood-filled spaces. Parenchymal types are lined by hepatocytes, and phlebectactic types are lined by endothelium and fibrosis (2). The difference in the linings are thought to represent stages of the same process, and both types occur in the same organ. Ultrastructural studies showed that both types have the same abnormalities of the sinusoidal barrier (3).

The pathogenesis of peliosis hepatis is not known, but proposed theories have included congenital malformations, vascular varicosities with or without vasculitis, vascular rupture, primary focal hepatic necrosis, obstruction of liver blood outflow or direct lesions of the sinusoidal barrier causing an increased permeability of the endothelium (2–5). Electron microscopy has shown that the peliotic cavities are formed by marked dilatation of either the space of Disse or the sinusoidal lumen and have wide areas of communication between the sinusoidal lumen and perisinusoidal space.

Peliosis is not limited to the liver. It has frequently been described in the spleen as well as other organs (6,7). Most cases have been reported in association with other conditions that include chronic wasting diseases such as malignancy or tuberculosis, use of androgenic

[1] Wayne County Medical Examiner Office, 1300 East Warren Avenue, Detroit, MI 48207.

Received 6 Nov. 2004; and in revised form 29 Jan. 2005; accepted 29 Jan. 2005; published 25 May 2005.

Copyright © 2005 by ASTM International, 100 Barr Harbor Drive, PO Box C700, West Conshohocken, PA 19428-2959.

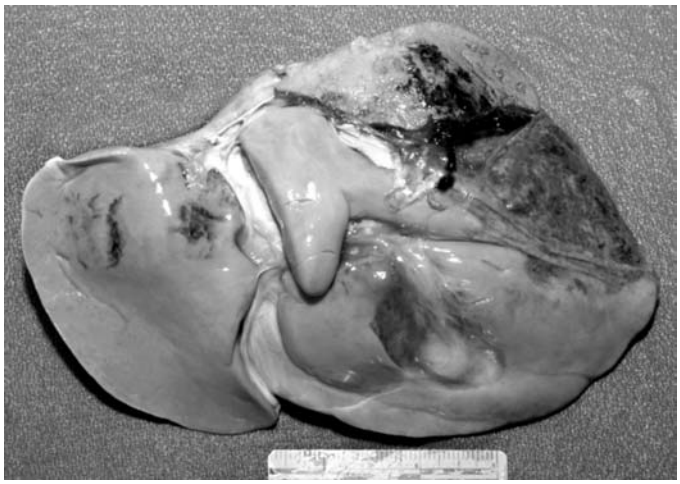

FIG. 1—*Posterior surface of the liver with laceration and hemorrhage on the right side of the liver and subcapsular peliosis on the left side.*

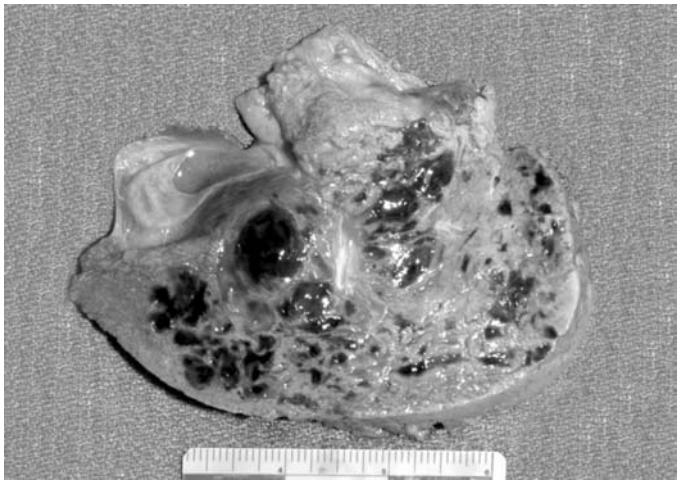

FIG. 2—*Cross-section of the liver showing multiple blood-filled cyst-like spaces.*

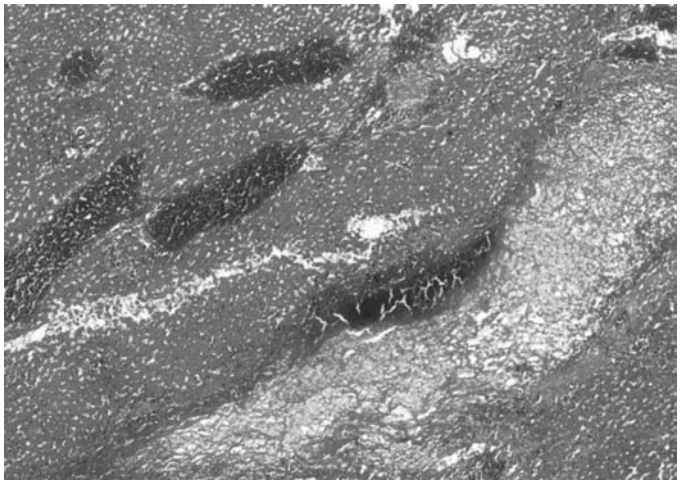

FIG. 3—*Histopathology of peliosis hepatis. (H & Ex 50).*

steroids, renal transplantation or infections (2,3,8,9). In the pediatric population, peliosis hepatis has also been linked to wasting illnesses such as cystic fibrosis, steroids and bacterial infection (2,10,11).

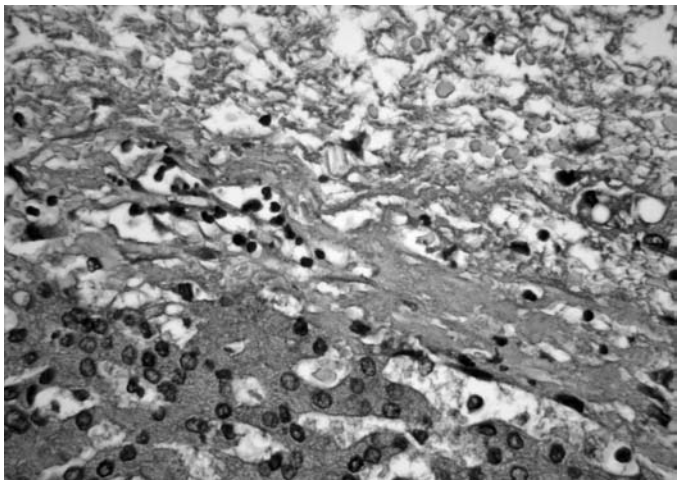

FIG. 4—*Parenchymal-type peliosis hepatis. Blood-filled spaces lined by hepatocytes. (H & Ex 100).*

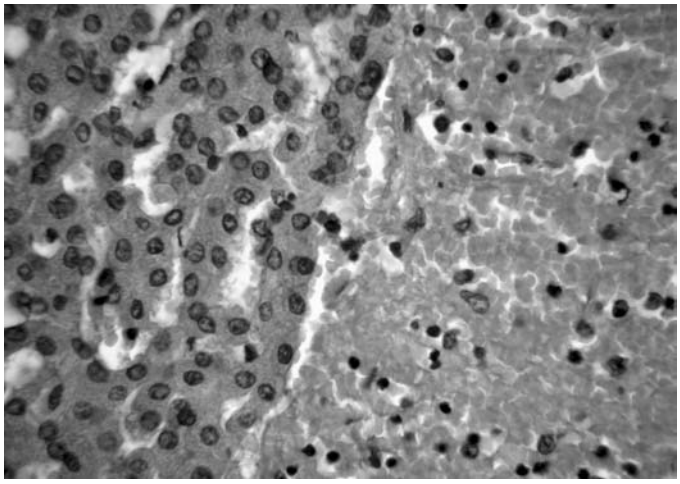

FIG. 5—*Phlebectatic-type peliosis hepatis. Blood-filled spaces lined by fibrosis and endothelial cells. (H & Ex 100).*

Cases that lack corresponding illness or drug use, however, have also been described in the literature. Hayward et al. reported a 50-year-old woman with peliosis hepatis and no illness or drug use who had two episodes of spontaneous hemorrhage requiring partial hepatectomy (12). Cragg et al. described a 13-month-old white male with an otherwise unremarkable medical history who had peliosis hepatis localized to the right side of the liver, requiring a partial hepatectomy due to hemorrhage (13). Zak proposed that the lesions could rarely be due to a congenital malformation in those cases with no other possible etiology (2).

Previously reported cases of spontaneous hemorrhage from peliosis have presented with acute abdominal pain with tenderness and guarding, nausea, vomiting, hypotension, tachycardia and syncope (12,14,15). The presenting symptom in a 13-month-old child was lethargy and subsequent evaluation revealed hepatomegaly and anemia (13). The only symptoms provided in this case were "coughing and vomiting," after which the child lost consciousness due to blood loss and never recovered. The dissection of Glisson's capsule away from the liver parenchyma by a hematoma would have caused significant abdominal pain.

Spontaneous hemorrhage rarely results in death, but it is not unusual to see extensive destruction of liver parenchyma when

fatal hemorrhage occurs (16). However, in this case, the presence of recent bruises on various parts of the body and the multifocal liver lacerations indicate that a physical beating caused the hemorrhage. It is reasonable to conclude that the abnormal liver was predisposed to hemorrhaging from any abdominal trauma, yet it is of interest to note that it is the first reported case in the literature of hemorrhage and/or death from traumatic injury of the liver in a person with peliosis hepatis.

She was a normally developing, well-nourished child with no other anatomic evidence of disease or infection. Had she not been injured, her long-term prognosis would most likely have been good. Peliosis hepatis is most often asymptomatic and an incidental discovery at autopsy. Hepatomegaly may be absent, and liver function may be normal or only marginally affected (9,11,12). It can remain asymptomatic or rarely progress to fulminant hepatic failure (11,14). It may resolve after removal of the associated drug, disease or infectious agent, and it can spontaneously regress (9,14). It rarely causes hemorrhage or death.

During further investigation, the mother admitted to physically beating the deceased two days before her death, but the red-purple color observed consistently in all of the small and large bruises on her body as well as the extent of the internal injuries indicate that the fatal beating occurred within a day of her death. During the two days since the mother admitted to beating the child, the deceased was also in the custody of various other family members, all of who denied hitting the child. The family offered no other explanations for the bruises or abdominal injuries. Due to the gaps in the clinical history, the mother was charged only with child abuse, and she was acquitted in a bench trial.

Acknowledgment

The authors would like to thank Joe Sopkowicz and Kelly Root for their photography.

References

1. Ludwig J. Handbook of autopsy practice. 3rd ed. New Jersey: Humana Press, 2002.
2. Zak FG. Peliosis hepatis. Am J Pathol 1950;26:1–6. [PubMed]
3. Zafrani ES, Cazier A, Baudelot AM, Feldmann G. Ultrastructural lesions of the liver in human peliosis: a report of 12 cases. Am J Pathol 1984;114:349–59. [PubMed]
4. Paradinas FJ, Bull TB, Westaby D, Murray-Lyon IM. Hyperplasia and prolapse of hepatocytes into hepatic veins during longterm methyltestosterone therapy: possible relationships of these changes to the development of peliosis hepatis and liver tumours. Histopathology 1977;1:225–46. [PubMed]
5. Degott C, Rueff B, Kreis H, Duboust A, Potet F, Benhamou JP. Peliosis hepatis in recipients of renal transplants. Gut 1978;19:748–53. [PubMed]
6. Ichijima K, Kobashi Y, Yamabe H, Fujii Y, Inoue Y. Peliosis hepatis: an unusual case involving multiple organs. Acta Pathol Jpn 1980;30:109–20. [PubMed]
7. Warfel KA, Ellis GH. Peliosis of the spleen: a report of a case and review of the literature. Arch Pathol Lab Med 1982;106:99–100. [PubMed]
8. Czapar CA, Weldon-Linne CM, Moore DM, Rhone DP. Peliosis hepatis in the acquired immunodeficiency syndrome. Arch Pathol Lab Med 1986;110:611–3. [PubMed]
9. Perkocha LA, Geaghan SM, Yen TSB, Nishimura SL, Chan SP, Garcia-Kennedy R, et al. Clinical and pathologic features of bacillary peliosis hepatis in association with human immunodeficiency virus infection. N Eng J Med 1990;323:1581–6.
10. Shapiro P, Ikeda RM, Ruebner BH, Connors MH, Halsted CC, Abildgaard CF. Multiple hepatic tumors and peliosis hepatis in Fanconi's anemia treated with androgens. Am J Dis Child 1977;131:1104–6. [PubMed]
11. Jacquemin E, Pariente D, Fabre M, Huault G, Valayer J, Bernard O. Peliosis hepatis with initial presentation as acute hepatic failure and intraperitoneal hemorrhage in children. J Hepatol 1999;30:1146–50. [PubMed]
12. Hayward SR, Lucas CE, Ledgerwood AM. Recurrent spontaneous intrahepatic hemorrhage from peliosis hepatis. Arch Surg 1991;126:782–3. [PubMed]
13. Cragg A, Castaneda-Zuniga W, Lund G, Salomonowitz E, Amplatz K. Infantile peliosis hepatis. Pediatr Radiol 1984;14:340–2. [PubMed]
14. Wang SY, Ruggles S, Vade A, Newman BM, Borge MA. Hepatic rupture caused by peliosis hepatis. J Pediatr Surg 2001;36:1456–9. [PubMed]
15. Kohr RM, Haendiges M, Taube RR. Peliosis of the spleen: a rare cause of spontaneous splenic rupture with surgical implications. Am Surg 1993;3:197–9.
16. Engel P, Jacobsen GK. An unusual case of retroperitoneal seminoma and fatal peliosis of the liver. Histopathology 1992;22:379–82.

Additional information and reprint requests:
Carl J. Schmidt, M.D.
Wayne County Medical Examiner Office
1300 East Warren Avenue
Detroit, Michigan 48207
E-mail: cjschmidt@co.wayne.mi.us