CASE REPORT

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Sudden Death Due to Spontaneous Rupture of the Spleen from Infectious Mononucleosis

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ABSTRACT: An 18-year-old white male showing no external signs of trauma was discovered dead in his bathroom. A ten-day history of illness resulting in symptomatic treatment for viremia one day before death was elicited. Autopsy revealed splenic rupture, massive peritoneal hemorrhage, and atypical lymphocytes. Laboratory tests on postmortem samples confirmed the diagnosis of infectious mononucleosis.

KEY WORDS: pathology and biology, mononucleosis, spleen

Rupture of the spleen is not a common finding [*1,2*] in patients with infectious mononucleosis (IM). When it occurs, it presents between the 10th and 21st day after the onset of the disease [*3*]. Lai [*4*] has estimated that this event occurs in 0.5% of IM cases. Wechsler et al [*5*] reported no deaths in a series of 556 cases; Thomsen [*6*] saw a mortality of about 1% in 549 cases; and York [*7*] found one ruptured spleen in 940 cases of IM. However, IM is not a rare disease in the United States; 25 to 50 cases per 100 000 population occur annually [*1*]. An easy calculation predicts from these statistics a rate of 250 to 500 ruptured spleens per year, many of which would be expected to be fatal. In contrast, Srivastava et al [*3*] related that only 55 cases of IM with spontaneous splenic rupture had been reported by 1970. We report here a case that came to the forensic pathology service as a sudden death from an unknown cause.

Case Report

An 18-year-old white male had been ill with flu-like symptoms for approximately ten days before seeing a physician. He was treated symptomatically and felt much better the following morning. In the mid-morning his parents found him semiconscious in the bathroom following a bowel movement. He was pronounced dead in a local emergency room. An autopsy was ordered.

Postmortem examination 6 h after death revealed a 1.8-m (72-in.) tall, 56-kg (124-lb),

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emaciated, and dehydrated white male. The inguinal and axillary lymph nodes were enlarged. There was 2000 cm^3 of recent hemoperitoneum (Fig. 1). The spleen was enlarged (550 g) and had a tense capsule.

At the inferior spleen pole there was a 2-cm tear in the capsule. The spleen pulp was soft. A blood-filled bleb or bulla was noted on the anterior lateral and hilar surface of the spleen (Fig. 2). A 2-cm-diameter, soft, tense, accessory spleen was in the tail of the pancreas. The liver was congested and had multifocal white infiltrative areas measuring up to 2 mm. The peritonsillar and pulmonary hilar lymph nodes were enlarged. There was no jaundice.

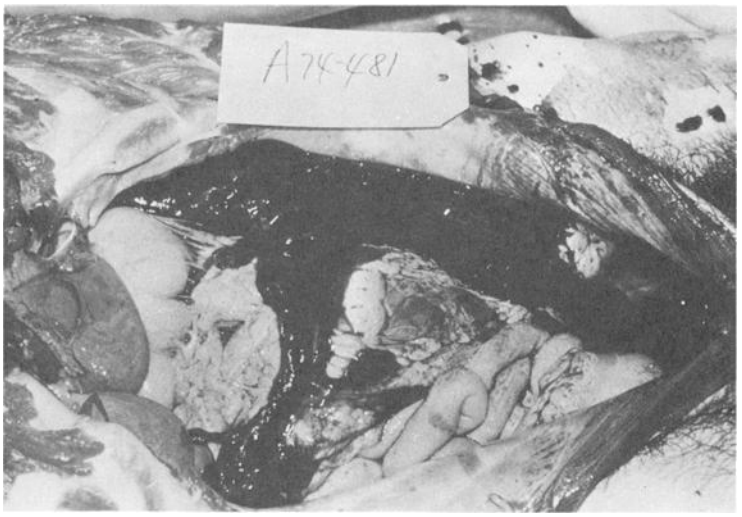

FIG. 1—*Recent hemoperitoneum.*

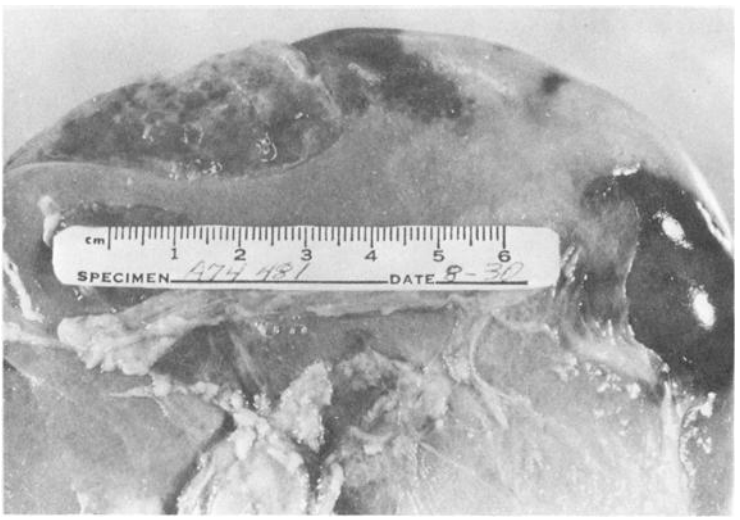

FIG. 2—*Spleen hilar surface capsule bleb and torn capsule.*

Microscopic

The peripheral smear (buffy coat) (Fig. 3) and spleen touch preparations (Fig. 4) revealed mononuclear cells with abundant vacuolated cytoplasm and pale areas in the large folded nucleus.

The bone marrow was hypercellular and contained many large mononuclear cells with vacuolated cytoplasm and pale nuclear areas.

The liver contained periportal mononuclear round cell aggregations, mononuclear round cells in sinusoids and central veins, and hepatocytic cloudy swelling, with necrosis of hepatocytes.

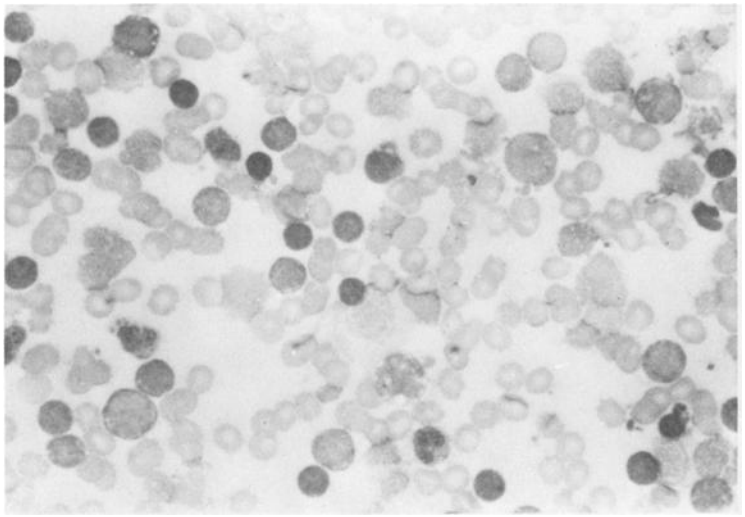

FIG. 3—*Peripheral smear (buffy coat) touch preparation showing mononuclear cells with abundant vacuolated cytoplasm.*

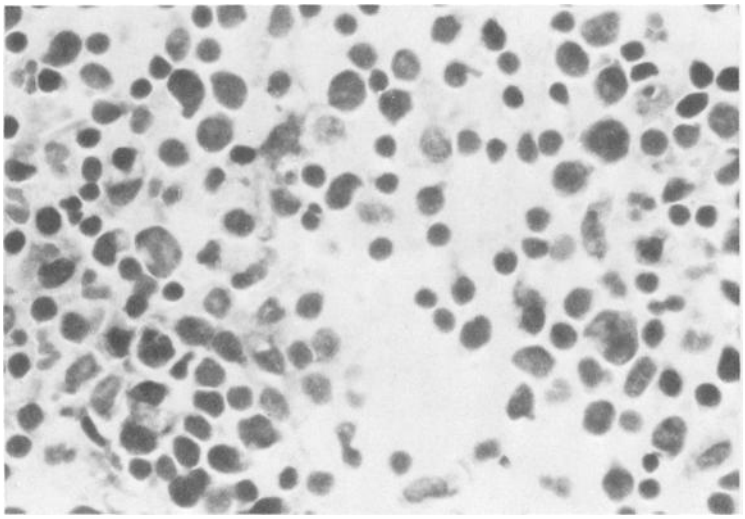

FIG. 4—*Spleen touch preparation showing mononuclear cells with abundant vacuolated cytoplasm.*

Examination of the spleen revealed sinusoidal congestion, atypical lymphocytes distributed diffusely through the pulp and sinusoids, and infiltrating trabeculae. Lymphoid follicles were present but had rudimentary or absent germinal centers. Evidence of hemorrhage was found in the bone marrow, liver, and spleen.

Lymph node observations included vascular congestion with red blood cells, intact architecture, and infiltration by numerous large mononuclear cells.

Laboratory Study

Stool culture showed no enteric pathogens. Tests of the vitreous humor revealed a chloride concentration of 83 meq/litre; carbon dioxide, 35 meq/litre; potassium, 1.9 meq/litre; sodium, 145 meq/litre; urea nitrogen, 10 mg/dl, and glucose, 93 mg/dl.

Blood was collected from the heart by using aseptic technique during postmortem examination and tested in various ways.

Routine examination of the blood produced a culture of *Pseudomonas aeruginosa*; total protein concentration, 8.1 g/dl; albumin, 4.2 g/dl; cholesterol, 150 mg/dl; uric acid, 6.1 mg/dl; creatinine, 2.5 mg/dl; total bilirubin, 0.8 mg/dl; alkaline phosphatase, 105 units/ml; and a negative alcohol result.

The liver arsenic test was negative, as were drug screens on urine, small bowel contents, stomach contents, and pericardial fluid. The examination of the urine for heavy metals showed a lead concentration of 4.0 μg/dl.

Serology showed a positive "monoslide" (Monospot®) test. A presumptive heterophil test resulted in a titer of 1:448. Following absorption with guinea pig kidney cells, the titer was 1:224 and fell to 1:7 after absorption with beef red blood cells.

Discussion

The diagnosis of IM is based on a constellation of findings [*1–3,6,8*]: hepatomegaly; splenomegaly; generalized lymphadenopathy, focal hepatic necrosis [*8*]; focal visceral hemorrhages; atypical mononuclear cells in the sections and touch preparations of spleen, liver, and lymph nodes; and the positive monoslide (Monospot) and differential heterophil tests.

The Paul-Bunnell-Davidsohn [*1,2,6,8–10*] differential absorption test measuring titers of agglutinins before and after absorption with guinea pig kidney and beef red blood cells differentiates agglutinins of IM from hematologic disorders, and the quantitative Monospot test [*1,9,10*] and atypical lymphocytes confirm the diagnosis of IM.

The basic structure of the spleen is weakened in IM [*2–4*]. A possible mechanism for spontaneous rupture [*6*] of the spleen in patients with infectious mononucleosis is degeneration of follicular arteries, vasculitis of larger veins, inflammation of the capsule, and splenomegaly creating a spleen condition susceptible to rupture during a Valsalva maneuver while straining at stool. Trauma [*6*] (vomiting, coughing, exercise [*11*]) so minimal as to not be noted by the patient or at autopsy may be the cause.

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