



Pathology Biology Section – 2011

G39 **Enterobacter Cloacae Peritonitis Secondary to Hemorrhagic Cystitis in a Long-Term Substance Abuser**

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The goal of this presentation is to illustrate an unusual case of peritonitis caused by hemorrhagic cystitis.

This presentation will impact the forensic science community by illustrating the need for microbiological cultures and routine histology in cases of peritonitis where an obvious source or rupture site is not identified.

Introduction: The major causes of peritonitis are appendicitis, perforations associated with diverticulitis, peptic ulcers, gangrenous

gallbladder, gangrenous obstruction of the small bowel, incarcerated hernia, and volvulus. Peritonitis secondary to cystitis is a rare, life threatening condition due to the unique anatomic characteristics of the urinary tract. Predisposing factors include anatomic anomalies of the urinary tract, vascular impairment, previous surgery, irradiation and high virulence pathogens. Making a diagnosis of peritonitis due to cystitis is difficult and the prognosis is usually poor.

Materials and Methods: This case involves a 47-year-old African- American female with a long standing history of alcohol, heroin, and cocaine abuse, who was found dead in her secure apartment. According to witnesses, she experienced flu-like symptoms for the past few days. The examination of the scene revealed a cluttered and unkempt dwelling with numerous empty and full malt liquor cans. Dark colored stains were noted on the bed and the floor, and a bucket with vomitus was discovered near the deceased.

Results: Postmortem examination revealed a poorly nourished African-American female, weighing 102 pounds and measuring 65.5 inches (BMI – 16.5). Signs of prior drug abuse, i.e., multiple remote circular scars ("skin popping" sites), were noted on the upper and lower extremities. At autopsy, the abdominal cavity contained 500 cc of serous fluid. Fibrinous exudate was observed on the dusky red small and large intestines. The urinary bladder contained 20 cc of dark-brown blood and exhibited a markedly thickened, hemorrhagic mucosal surface. No gross perforation was identified.

Microscopic examination revealed severe full thickness acute and chronic inflammation, focal hemorrhage, and necrosis of the urinary bladder. Both kidneys displayed acute tubulointerstitial nephritis.

Peritoneal fluid and urine cultures grew *Enterobacter Cloacae*. Postmortem toxicology was positive for Ethanol (0.011% in the blood; 0.020% in the vitreous fluid).

No other pathologic abnormalities or trauma were identified during the autopsy.

Conclusion: Secondary peritonitis follows contamination of the peritoneum by organisms released from the infected organs or perforated viscera. Peritonitis due to acute cystitis is a rare occurrence with only a handful of reports published in the medical literature. Most of the cases involved gangrenous inflammation of the urinary bladder with or without perforation.

In this case, integrity of the bladder wall was preserved. The significant amount of blood in the bladder cavity and severe acute transmural inflammation with hemorrhage and focal necrosis supported the diagnoses of hemorrhagic cystitis.

Hemorrhagic cystitis results from damage of the transitional epithelium and blood vessels by infection (bacteria, viruses) and non- infection etiologies (drugs, toxins, radiation). In this case, *Enterobacter Cloacae* colonies were isolated from urine and peritoneal fluid. It is worth noting, that in adults *Enterobacter* affects individuals with underlying physical or structural anomalies, metabolic disorders or immunodeficiency causing complicated urinary tract infections. *Enterobacter* comprises 1.9% to 9.6% of all UTI pathogens.

The past history of the deceased played an important role in the evolution of what started as an innocent urinary tract infection (UTI) to a fatal condition. A number of studies have shown that drugs of abuse, including cocaine, opiates, and alcohol, alter not only neuropsychological and pathophysiological responses of individuals but also immune functions. This decedent's extensive history of polysubstance abuse and malnutrition (BMI of 16.5; normal 18-24) apparently caused severe debilitation of the immune system with rapidly progressive infection and the resultant grim outcome.

Peritonitis, Hemorrhagic Cystitis, Cocaine User