It’s Not Miller’s Time

A Case Report of Septic Barium Peritonitis

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Introduction:

Septic barium peritonitis is a rare, but serious condition that requires timely intervention. Septic peritonitis can occur as a primary cause from hematogenous spread or a secondary complication from surgical procedures, penetrating wounds, or an underlying disease process.[6] In one study septic peritonitis occurred in 6-35% of patients following a gastrointestinal surgery.[1] The most common cause of gastrointestinal perforation in dogs is from dehiscence of a enterotomy site following removal of a foreign body.[5] Dehiscence of an enterotomy site is reported to occur between 3-9 days post operatively.[6] Clinical signs of a septic abdomen may be non-specific and include lethargy, anorexia, vomiting, tachycardia and a painful distended abdomen.[8] With barium peritonitis a historical finding is administration of barium sulfate after a gastrointestinal perforation. This could be due to gastrointestinal surgery, a linear foreign body, administration of non-steroidal anti-inflammatories and steroids, ulcerations, or a mass within the intestinal tract. The prognosis for a septic barium peritonitis is guarded to poor due to several complications including vascular shock, hypoproteinemia, electrolyte imbalance, and small bowel obstructions that are associated with adhesion formation.[9] The case outlines how septic barium peritonitis can be successfully treated with the correct diagnostics, interventions, and intensive post-operative management.

History and Presentation:

Miller, a 2-year-old male neutered lab, presented to MSU-CVM emergency service on February 17, 2018 with a septic abdomen. Approximately one week prior to presentation Miller was seen at his primary veterinarian for continued episodes of vomiting. Due to Miller's prior history of eating foreign material, there was suspicion for an intestinal obstruction. Miller's owners elected for exploratory surgery on February 12th that revealed a foreign body to be the

cause of the obstruction. Recovery from surgery and anesthesia was uneventful and Miller was discharged on February 14th. Four days post operatively Miller started vomiting again and was brought back to his primary veterinarian. Due to the suspicion of an intussusception a barium sulfate study was performed. Radiographs showed that barium sulfate had leaked into his abdominal cavity. He was referred to the Animal Emergency and Referral Center (AERC) in Flowood, Mississippi, but with no surgeon on site, he was subsequently referred to MSU-CVM for further diagnostics and emergency surgery for a septic abdomen contaminated with barium sulfate suspension.

At the time of presentation Miller was depressed, but alert and responsive. He was tachycardic with a pulse of 140 beats per minute, tachypneic with a respiratory rate of 60 breaths per minute, and normothermic with a temperature of 101.4 F. His mucus membranes were pale pink and tacky with a capillary refill time of less than 2 seconds. He was determined to be approximately 8% dehydrated and hypertensive with a mean arterial blood pressure of 162 mmHg. His abdomen was tense on palpation and a fluid wave was present. An abdominal FAST scan revealed a fluid score of 4/4, while thoracic FAST scan showed no evidence of free fluid. The remainder of his physical examination was unremarkable.

Pathophysiology

Barium sulfate is insoluble and remains within the lumen of the gastrointestinal tract. Therefore, it is an acceptable diagnostic tool when gastrointestinal perforation is not suspected.[7] When barium sulfate is accidently introduced into the abdominal cavity and mixes with intestinal contents there is a time-dependent, synergistically deleterious effect.[9] The barium begins to adhere in small fibrin-covered clumps to parietal and visceral surfaces due to the additives found in the suspension.[3] The inflammatory response produced by the body leads to formations of these

“barium granulomas”. The body responds by stimulating fibroblastic proliferation and vascularization on the periphery of the granuloma.[9] This fibroblastic proliferation allows adjacent loops of bowel or nearby organs become entrapped in fibrous adhesions. The longer the barium remains in the abdominal cavity the more adhesions and granulomas form and increase mortality rates. Due to the time dependent effect of barium within the abdominal cavity it is essential to begin treatments within 2 hours after barium leakage has occurred.[9]

Barium complicates a typical septic peritonitis by causing a severe hypoproteinemia and hypovolemia[9]. This is due to inflammatory response which involves vasodilation, exudation of extracellular fluid that is protein rich and cellular infiltration. This is caused from an increase in vascular permeability.[7] As oncotic pressure decreases more fluid is extravasated and the hypovolemia worsens.

Diagnostic Approach

Diagnosis of a septic abdomen involves a thorough history, clinical signs, several diagnostic modalities. A barium study is contraindicated if perforation is suspected. Ideally contrast should be avoided, although alternative oral contrast media can be used such as Iohexol and Amidotrizoate sodium meglumine as they are both water soluble and readily absorbed by the body.[4] Miller was unfortunately given barium sulfate as an intussusception was suspected by the primary veterinarian. Radiographs confirmed the presence of barium sulfate leakage which tends to accumulate in the cranial abdomen around the small intestines and omentum. One retrospective study suggests ultrasonography is useful diagnostic tool. The presence of free gas, peritoneal effusion, bright mesenteric fat, and loss of serosal wall layering have been associated with patients with intestinal perforations.[5]

Glucose and lactate levels of the blood and abdominal fluid can be used as indicators for a septic abdomen.[1] The abdominal fluid glucose should be lower than the peripheral blood because the bacteria are consuming the glucose for energy. Conversely the abdominal fluid lactate will be higher than the peripheral fluid due to production from the bacteria and tissue hypoxia. A peripheral blood to abdominal fluid glucose difference >20mg/dL is indicative of a septic abdomen.[6] Miller's peripheral blood glucose was 196mg/dL while his abdominal fluid glucose was 113mg/dL giving a difference of 83mg/dL. A peripheral blood to abdominal fluid lactate difference >2.0mmol/L is also indicative of a septic abdomen.[6] In Miller's case his peripheral blood lactate was 4.2mmol/L, while his abdominal fluid lactate was 14.1mmol/L giving a difference of 9.9mmol/L.

Cytologic evaluation of the abdominal fluid can provide sufficient evidence for diagnosis of septic barium peritonitis. Marked suppurative inflammation with intracellular barium sulfate granules and evidence of intracellular bacteria are markers.[9] Aerobic and anaerobic culture and sensitivity were performed on the abdominal fluid. *Staphylococcus*, *Escherichia coli* and *Bacillus cereus* grew from the enrichment broth. It is recommended to start antibiotics containing beta lactams, aminoglycosides or fluoroquinolones until culture and sensitivity come back.[9] Miller was started on intravenous Unasyn and Enrofloxacin. *Staphylococcus, Escherichia coli* and *Bacillus cereus* grew from the enrichment broth and were sensitive to the antibiotics chosen.

Treatment and Management

Surgery is the definitive treatment for a septic abdomen although it is critical to stabilize the patient first. Treatment involves intensive intravenous fluids, broad spectrum antibiotics started prior to culture and sensitivity and adequate pain medications. One study suggests that intravenous fluid therapy is one of the most important treatment options to reduce mortality rates

in a barium peritonitis case.[9] Miller was given a quarter shock bolus dose of plasmalyte at 90ml/kg and reassessed after fifteen minutes. Another quarter shock bolus was given until his blood pressure, heart rate, and respiratory rate were improved.

Miller went to surgery for an exploratory celiotomy following confirmation that he had a septic abdomen. Two of the enterotomy sites were found to be leaking and were repaired. The integrity of the four enterotomy sites were evaluated by intraluminal injection of sterile saline solution. A serosal patch procedure was done over each enterotomy site. A sample of the abdominal fluid was obtained for bacterial culture and antibiotic susceptibility testing. The barium sulfate suspension was adhered to the parietal and visceral surfaces throughout the abdominal cavity. Copious irrigation, direct wiping with gauze and omentectomy were used to assist in removal of the barium sulfate. One study found a decrease in adhesions formed in those animals treated with copious irrigation and removal of the omentum.[2]

Abdominal and closed suction drains, PEG tube, and urinary catheter were placed before closing the abdomen. There is controversy as how patients should be managed post-operatively. Two retrospective studies have compared closed suction drainage, open peritoneal drainage and vacuum-assisted drainage, although neither study showed a difference in survival rate between techniques.[1] Closed-suction abdominal drainage allows for continuous active drainage of abdominal fluid into a closed collection system. This allows for easy quantification of daily fluid levels and has become the most frequent used method of post-operative management.[1]

Case Outcome:

The prognosis for a barium peritonitis is guarded to poor with a higher mortality rate than a septic peritonitis.[6] There is a time-dependent and synergistically lethal effect when abdominal

contents are mixed with barium sulfate. Of those that survive the initial barium peritonitis, up to 30% of patients experience a small bowel obstruction due to progression of abdominal adhesions.[2] Miller stayed at MSU-CVM for 10 days post operatively for intensive monitoring because mortality rate is highest during the first two weeks.[7]

Post-operative care during the first 24-48 hours requires continuous care and adjustments to the therapy plan as needed. Miller was maintained on norepinephrine intra-operatively and post-operatively due to moderate hypotension (mean arterial pressure of 60mmHg) It is important that the mean arterial pressure be maintained above 60-70mmHg to ensure proper perfusion to the body's organs especially the kidneys and brain.[6] Fluid therapy needs to be adjusted based on insensible losses, urine volume, and ongoing losses.[6] To calculate for this Miller had a urinary catheter, two Jackson Pratt (JP) drains, and a percutaneous endoscopic gastrotomy tube (PEG) placed and fluid totals calculated in mls/kg/hr.

The most significant and challenging changes Miller experienced post-operatively were his hypoalbuminemia and edema formation. Hypoalbuminemia is a common post-operative complication affected up to 69% of patients.[6] Miller's hypoalbuminemia was severe at 1.3g/dl one day post operatively. To combat the severe hypoalbuminemia a plasma transfusion was warranted. Early enteral nutrition was started to reduce the risk of hypoproteinemia and support the enterocytes of the gastrointestinal tract. Miller was offered high sources of protein including plain boiled chicken and Purina EN canned diet. A PEG tube was placed but only needed a few days as Miller developed an appetite. The subcutaneous edema formation can be explained by the decrease in oncotic pressure.[3] Compression bandages were applied to Miller's limbs to force fluid to be reabsorbed into the intravascular compartment. Massaging and warm packing also were indicated as therapy to reduce excess fluid and swelling.

At the time of discharge Miller was stable with vital parameters within normal limits. The JP drains, urinary catheter and PEG tube had been removed and no subcutaneous fluid accumulation had occurred from the incision sites. His hypoalbuminemia was improving with albumin levels of 2.1g/dl. Miller was discharged on 10 days of Clavamox (18.5mg/kg PO q12h) and Enrofloxacin (10mg/kg PO q24h) and instructed to recover with strict cage rest and leash walks to decrease the risk of incision dehiscence. A two week recheck was scheduled with his primary veterinarian and was reported to have increased albumin levels up to 2.5g/dl and a healed incision site. Miller's owners report that he has been doing well at home and has not had gastrointestinal signs reoccur.

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