

## Toxic Megacolon with Perforation and Hemolytic Uremic Syndrome with Acute Renal failure in a child due to E Coli

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**Abstract:** Toxic megacolon is a lethal and potentially life threatening condition characterized by dilatation of the colon. One of the dreaded and rarely encountered consequence of Toxic megacolon is perforation. Toxic megacolon and perforation secondary to infective colitis is rare in children. Aggressive medical management and prompt surgical intervention is needed to reduce mortality and morbidity in such cases. We report a case of a 3-year-old child with E coli diarrhea complicated by Toxic megacolon with rupture, Hemolytic Uremic syndrome and Acute Renal failure.

**Keywords:** Children, E coli, Hemolytic Uremic Syndrome, Perforation, Toxic megacolon

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### I. Introduction

Toxic megacolon is clinically defined as an acute inflammation with dilatation of the colon<sup>2</sup>. It is defined as a transverse colon with a diameter of more than 5-6 cm, with loss of haustrations<sup>3</sup>. Even though it was first described as a complication of Inflammatory Bowel Disease, it can develop as a complication of a number of colitides like inflammatory, ischemic, infectious, radiation, and pseudomembranous colitis<sup>2</sup>. In this case, toxic colitis happened as a complication of Infective colitis due to E coli. The case was also complicated by Hemolytic Uremic syndrome, Acute Renal failure and sepsis. The alarming complication of toxic megacolon is perforation, even when there is no colonic dilatation<sup>2</sup>. Not many pediatric cases of toxic megacolon with perforation are reported.

### II. Case Report

A three-year-old boy was brought to the emergency department with diarrhea for four days as chief complaint. The patient reported to have fever, emesis and bloody diarrhea during the last two days of the episode. He also presented with history of decreased oral intake, decreased urine output and decreased energy. He was delivered at 41 weeks by Caesarean section (indicated due to failure of progression). Past history is significant for mild asthma needing Albuterol PRN. There is no history of gastrointestinal problems or family history of gastrointestinal diseases. He is up-to-date on Immunizations. He does not have any known allergies. On Physical examination, he was dehydrated and was admitted and started on intravenous fluids, GE diet and probiotics. On admission, his Blood urea nitrogen level was 10 mg/dl, Creatinine 0.31mg/dl, BUN/Creatinine ratio 32, CO2 18 mmol/L, Red Blood cell count 5.81 m/UL Hemoglobin 13.8 g/dl, Neutrophils 83% and Hematocrit 40.4%. Stool culture and GI panel showed E coli 0157:H7, Shiga like toxin producing E coli, Enterogagregative E coli and Sapovirus. Ultrasound taken one day prior to admission was normal. Fluid was stopped on the second day of admission due to concern for Hemolytic Uremic syndrome and fluid overload. He developed Acute Renal failure five days post admission and was started with Hemodialysis. Simultaneously he developed respiratory failure with bilateral pleural effusion, pulmonary edema, DIC and sepsis and was intubated. Renal Ultrasound with Doppler after three days showed "Enlarged echogenic kidney with abnormally elevated resistive indices consistent with medical renal disease." "The patient continued to have bloody diarrhea, improving in frequency and amount of blood.

Patient's abdomen became severely distended and discolored with erythema and swelling, suggestive of abdominal wall cellulitis on the tenth day post admission and paracentesis showed frank blood. Exploratory laparotomy was done and revealed Pancolitis and Left necrotic colon suggestive of Toxic megacolon. Patient underwent subtotal colectomy and silo placement. He was held NPO and nutrition and antibiotics were administered parenterally. He underwent creation of ileostomy, removal of silo and closure of abdominal wall two days later. Patient had significant oral aversion and so Nasogastric tube was inserted and enteral feeds were initiated. Patient had increasing Phosphorous and Potassium following this. He developed metabolic acidosis

and feeds were changed accordingly. Patient started tolerating oral feeds two weeks later, however he developed emesis. X ray abdomen at that time showed dilated bowel loops suggestive of Small Bowel Obstruction, however XR enema with water soluble contrast showed no evidence of obstruction at the ostomy site. Patient was continued on enteral feeds and his symptoms improved with NPO and starting Metronidazole for small bowel bacterial overgrowth which was discontinued prior to discharge. He was restarted on oral feeds and Nasogastric feeding discontinued on discharge. Kidney seemed to have recovered prior to discharge. On discharge, his Phosphorous level was 6.1 mg/dl. Patient recovered well and colostomy was removed three months later. He was discharged on Amlodipine BID, Clonidine patch weekly, Cyproheptadine QHS, Omeprazole BID, Probiotics BID, Sodium citrate TID, Albuterol solution for Nebulization as needed, Ibuprofen as needed, Ondansetron as needed.

### III. Discussion

In 1950, Marshak and Lester first described Toxic Megacolon.” The most widely recognized diagnostic criteria proposed by Jalan et al is as follows:

- Radiographic evidence of colonic dilatation - The classic finding is more than 6 cm in the transverse colon
- Any 3 of the following - Fever ( $>101.5^{\circ}\text{F}$ ), tachycardia ( $>120$  beats/min), leukocytosis ( $>10.5 \times 10^3/\mu\text{L}$ ), or anemia
- Any 1 of the following - Dehydration, altered mental status, electrolyte abnormality, or hypotension.<sup>2</sup>

Benchimol et al. surveyed 10 patients aged 7.3–15.5 years with IBD, and established colonic dilatation larger than 5.6 cm was suggestive of toxic megacolon.<sup>4</sup>Toxic megacolon is a well-known complication of inflammatory bowel disease such as Ulcerative colitis and Crohn’s disease. Severe inflammation extending into the smooth muscle layer paralyzes the colonic smooth muscle causing dilatation leading to Toxic Megacolon<sup>2</sup>. The development of toxic megacolon as a result of infectious colitis is uncommon and is usually due to *Clostridium difficile*, *Campylobacter jejuni*, *Salmonella Shigella* species, *Yersinia* species, Cytomegalovirus, *Cryptosporidium*, Rota virus, Invasive *Aspergillus* and *Entamoeba histolytica*.<sup>2</sup>Perforation or necrosis of the colon in pediatric patients with HUS is rare. *E coli* colitis complicated by toxic megacolon with perforation is rare and to our knowledge, there are only four reported cases of colon necrosis that required colectomy, and there are only four case reports of colon perforation and none published recently.<sup>5</sup>The uniqueness of this case lies in the fact that the toxic megacolon with perforation is sparsely reported in such a young child. Furthermore, his condition was complicated by Hemolytic Uremic syndrome, acute renal failure requiring Hemodialysis, Respiratory failure, pulmonary edema, Sepsis, Disseminated intravascular Coagulation and Metabolic acidosis. The patient’s weakened immune status post renal failure precipitated the occurrence of Toxic megacolon leading to early perforation. Even though Toxic Megacolon with perforation is a rare condition, heightened clinical awareness and early management including timely surgical intervention can reduce mortality and morbidity in Pediatric population. In our case, the patient recovered completely and returns for follow up treatment regularly.

### IV. Conclusion

Toxic megacolon with perforation is a possibility in pediatric population with *E coli* diarrhea, albeit rare. If not identified and treated aggressively it will drastically increase the mortality and morbidity. Mortality rates range from 19% up to 45% and are strikingly higher among patients complicated with perforation.<sup>6</sup> For this reason, we recommend high vigilance in these cases and early intervention including expeditious and aggressive surgical intervention, if necessary<sup>7</sup>.

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