# D-Lactic Acidosis in a Patient after Subtotal Colectomy

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D-Lactic acidosis is a rare complication presenting in patients with short bowel syndrome. DLA is characterized by severe lactic acidosis and mental disturbances. Review of the literature reveals that DLA has so far been diagnosed only after short bowel resection. We present a unique case in which DLA was diagnosed in a patient after large bowel resection. A biochemical mechanism for pathogenesis is suggested, and the primary definition of this syndrome is questioned.

## Patient Description

An 80 year old man was admitted to our department with synchronous adenocarcinomata of the colon. Under colonoscopy, he was found to have adenocarcinoma of the sigmoid colon and the hepatic flexure, vilotubular adenoma in the splenic flexure and numerous polyps along the colon (15 cm of distal colon were clean). His medical history revealed ischemic heart disease and hypertension. Ultrasound demonstrated a right hydronephrotic kidney and no liver metastasis. Intravenous pyelography showed a non-functioning right kidney. Physical examination and laboratory tests were normal.

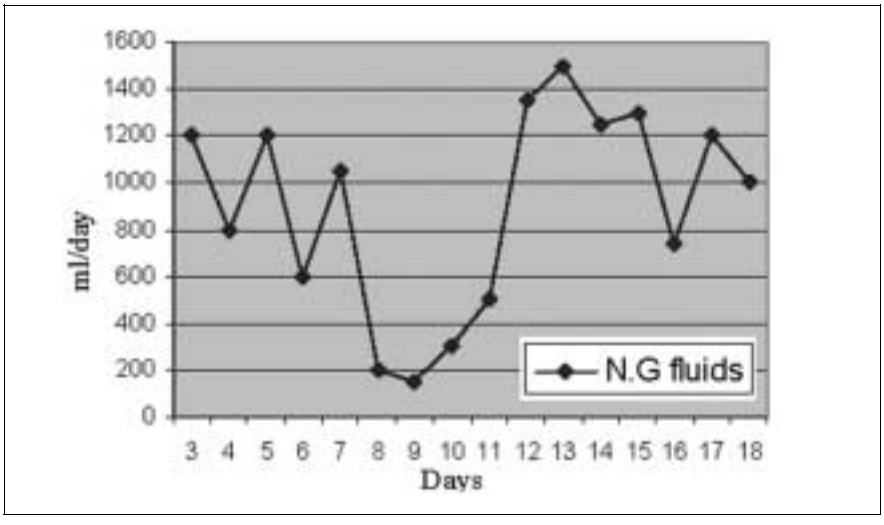
A subtotal colectomy with an ileal J-pouch and right nephrectomy was performed. The operation was uneventful. From the third postoperative day, there was high secretion of biliary fluid from the nasogastric tube [Figure]. On the 6th postoperative day the patient started to have flatus, and on the 7th day he had normal stool. Since the high gastric secretion continued on the 10th postoperative day, total parenteral nutrition was administered. On the 18th postoperative day, the patient developed tachypnea and was confused and disoriented. His temperature was 37°C, blood pressure 145/85 and pulse 112. Physical examination did not show any signs of peritonitis or pulmonary edema (confirmed by chest X-ray). There were no signs of ischemia on electrocardiography. Laboratory tests showed iron deficiency anemia of 8.6 g/dl hemoglobin and 8,300 white blood cells with no electrolyte disturbances. Blood gases showed metabolic acidosis: pH 7.2, bicarbonate 7.2 mEq/L, $pCO_2$ 16 mmHg with an anion gap of 21 mMol/L. Blood lactic acid was 99 mg/dl (normal 5–15 mg/dl). This lactate value of 99 mg/dl represents a concentration of 11 mMol/L (normal 0.6–1.7). Given the anion gap of 21 mMol/L, the increase in anion gap was 10–11 mMol/L (taking a normal anion gap of 12 2 mMol/L). Thus the increase in anion gap is completely explained by the lactate.

On the assumption that sepsis is the most likely cause for the patient's deterioration, blood and urinary cultures were taken, the central line was removed and empiric broad-spectrum antibiotic treatment was initiated, combined with antifungal therapy. Emergency abdominal computerized tomography scan with contrast enema showed no leak from the anastamotic site. All cultures were sterile.

Because of the patient's rapid deterioration and the delirium, the diagnosis of DLA was raised and a frozen urine specimen was sent to the chemical pathology laboratory. The D-lactic acid measured 6,900 µg/L creatinine (normal 0–20). The patient's acidosis was diagnosed as DLA and he was treated with neomycin enemas and oral metronidazole. He recovered within 36 hours and was discharged after a week. He has had no recurrent episodes.

## Comment

DLA is a rare complication, seen in patients with short bowel and preserved colon. DLA was first described by Man et al. in 1979 [1], which occurred in a 30 year old patient 2 years after small bowel resection




Postoperative gastric fluid volume excretion.

DLA = D-lactic acidosis

due to superior mesenteric thrombosis. The patient was suffering from recurrent episodes of delirium, vomiting and metabolic acidosis with wide anion gap [1]. Since then, 29 cases have been reported, all in patients with short bowel syndrome. In 1998 Uribari et al. [2] described DLA as a combination of severe wide anion gap, metabolic acidosis and neurologic disturbances, including nystagmus, ophthalmoplegia, ataxia, confusion and disoriented behavior. An electroencepalograph during an episode of DLA shows slow waves with high voltage, and spinal fluid analysis shows traces of D-lactic acid [3].

D-lactic acid is an optical isomer of L-lactic acid. The latter is produced from anaerobic fermentation of glucose in human cells, and the former by bacteria found in the large bowel. Vella and Farrugia [4] suggested the pathogenesis of DLA in short bowel syndrome. Incomplete digestion of polysaccharides and disaccharides pass rapidly to the large bowel where colonic bacteria ferment polysaccharides and produce organic acids. The mass production of organic acids lowers the intraluminal pH. In a low pH environment, there is an overgrowth of *Lactobacillus* bacteria and inhibition of other colonic flora. L- and D-lactic acid is produced and absorbed into the blood circulation. L-lactate is metabolized by lactate dehydrogenase and D-lactate by D-2 hydroxy acid dehydrogenase found in liver mitochondria, and the adrenal cortex is inhibited by L-lactate. (According to early reports, it was thought there is no equivalent enzyme for D-lactate.) The inhibition of D-lactate degradation causes accumulation of D-lactate and metabolic acidosis [4]. The neurologic symptoms are due to a severe decrease in intracerebral pH that interferes with ATP and neurotransmitter production, especially in the cerebellum [5].

The suggested treatment for DLA episodes is a combination of oral non-absorbable antibiotics, vitamin $B_1$, insulin and a low carbohydrate diet. The role of neomycin enemas is debatable. To prevent recurrent episodes, a diet containing less than 10% carbohydrates is recommended [2,4].

In conclusion, DLA is a vigorous and potentially fatal disease, but easily reversible once diagnosed. DLA is not solely a complication of the short bowel syndrome and the pathology has wider ramifications. Because of its rapid progression, the patient either dies from severe metabolic acidosis or recovers rapidly due to prompt initiation of empiric antibiotic therapy. Many physicians are not aware of this rare diagnosis and few laboratories have the means to analyze this isomer. The profile of patients who could potentially develop DLA, which would aid in the prevention of such episodes in any future victims, could not be described. However, this case report might raise questions on the definition of conventional D-lactic acidosis and perhaps physicians will now become more aware of the need to perform more quantitative analyses in these conditions. In our case of DLA in a patient with normal short bowel shortly after a subtotal colectomy, the primary explanation for the DLA event was the bacterial overgrowth in the ileal J-pouch, but many questions are left open.

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