IJKD KIDNEY DISEASES

A Rare Case of Acute Kidney Injury: McKittrick Wheelock Syndrome

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McKittrick-Wheelock syndrome is a rare complication of rectosigmoid villous adenoma leading to secretory diarrhea, prerenal acute kidney injury and severe fluid and electrolyte imbalances. There are about 50 cases reported in literature. We represent a case of 71 year-old patient with persistant chronic diarrhea, prerenal azotemia, severe hypokalemia, and hyponatremia. Initially, the patient's kidney function and serum potassium values were normalized by conservative treatment, and villous adenoma was removed by surgery.

IJKD 2019;13:132-3
www.ijkd.org

Keywords. acute kidney injury, hyponatremia, hypokalemia, secretory diarrhea

Case Report

INTRODUCTION

McKittrick-Wheelock syndrome is caused by a secretory colorectal tumor and is characterized by fluid and electrolyte depletion. Dehydration, mucous diarrhea, symptoms of hyponatremia (lethargy, headache, etc.), and hypokalemia (paresthesia, cramps, ileus, etc.) are the major symptoms of the *McKittrick-Wheelock* syndrome.[1] Approximately 50 cases were reported in the literature,[2] first described in 1954.[3] We report a case of *McKittrick-Wheelock* Syndrome associated with acute kidney injury.

CASE REPORT

A 71 year-old female patient was admitted to the emergency department with complaints of nausea, vomiting, diarrhea, and weakness. The patient had hypertension for 5 years, and also had diarrhea for 4-5 years.

Physical condition was moderate to good, consciousness was slightly cloudy. Blood pressure was 84/59 mmHg, pulse rate was 104/min, body temperature was 36.5 ℃ and respiratory rate was 20/min. She had dehydration signs and oliguria. The tone of the bowels increased and tenderness was present in the physical examination of the abdomen. There was a contamination on digital rectal exam, which was thought to be a white aqueous secretion.

Biochemical parameters of the patient at the beginning and in postoperative period were shown in Table 1. In the patient's arterial blood gas; P_H: 7.38, P_{CO_2}: 24.9 mmHg, $HCO3^-$: 14.5 mmol/L, lactate: 1.92 mmol/L. There was a trace amount leukocyte and erythrocyte in urinalysis. Sodium, potassium and chlorine in spot urine were 9 mEq/L, 21 mEq/L, and 7 mEq/L respectively. The patient was admitted to nephrology intensive care unit with acute kidney injury. Hemodialysis was applied for uremic symptoms. The patient underwent intravenous isotonic saline and approximately 120 mEq intravenous potassium replacement therapy on the first day. With improvement of the patient oral intake continued on the next day (oral and IV, 120-160 mEq/d; potassium replacement). There were no significant features in the stool microscopy

Table 1. Biochemical Parameters at the Beginning and in Postoperative Period

Parameters	Beginning	Postoperative
Urea (mg/dL)	320	9
Creatinine (mg/dL)	6.58	0.61
Sodium (mEq/L)	114	138
Potassium (mEq/L)	2.6	3.87
Chlorine (mEq/L)	62	105

and culture. Urea: 60 mg/dL, creatinine: 0.73 mg/dL, sodium: 135 mEq/l, potassium: 3.52 mEq/L, and chlorine: 96 mEq/L were determined on the fourth day of the fluid and electrolyte therapy .

In the blood test, TSH: 0.01, free T4: 2.27 were detected. Thyroid ultrasonography was performed and increased right lobe size was detected. Multiple nodules were detected in both lobes. Thyroid scintigraphy was performed, and toxic multinodular goiter was reported. The patient underwent thyramazol 10 mg/day. The time of gastrointestinal transit in the period of thyrotoxicosis was shortened, which may manifest itself with increased stool frequency with occasional mild steatosis. Since our patient's diarrhea continued after the thyroid function tests returnd to normal after treatment, we did not think the diarrhea is due to the thyrotoxicosis .

Colonoscopy was applied to our patient, and a non-obstructive polypoid mass was seen at 7-8 cm of the rectum, and biopsies from the mass were taken. The lesion was removed with the preferred low anterior resection method in the upper and middle rectal cancers. Histopathologically, a villous lesion leading to *McKittrick-Wheelock* syndrome was reported as mild dysplasia in epithelial cells.

DISCUSSION

Colonic villous adenoma occurs usually in the rectum and rectosigmoid regions, and it is usually a sessile tumor and can be up to 18 cm in diameter. Size, villous configuration and degree of dysplasia should be specified to estimate the risk of malignancy.[4] About 3% of villous adenomas are hypersecretory; larger sizes are more likely to cause liquid-electrolytes imbalances.[5] But the main cause of hypersecretion is increased prostaglandin E synthesis. In patients with villous adenomas of the rectum studies show that the level of prostaglandin E2 (PGE2) is 3 to 6 times higher than normal.[6] Colonic malignant tumors generally develop from benign adenomas. The adenomas have a 2.5% risk to evolve into colon cancer in 10 years, but the risk is increased for villous and larger adenomas. Current therapy consists of tumor resection (endoscopic or surgical) or brachytherapy, after correction of renal function, fluid and electrolytes imbalances.[7] Endoscopic resection is an option and is not very effective, especially because of the location and size of the tumor in the *McKittrick-Wheelock* syndrome.[8]

CONCLUSION

In conclusion, acute kidney injury with chronic diarrhea, severe electrolyte imbalances, and tumor in the rectosigmoid region should bring the *McKittrick-Wheelock* syndrome to mind.

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Received June 2018
Revised August 2018
Accepted October 2018