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【 CASE REPORT 】

Non-occlusive Mesenteric Ischemia with Significant Hyperphosphatemia

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Abstract:
An 86-year-old Japanese woman was referred to our hospital due to the sudden onset of abdominal pain. Abdominal contrast-enhanced computed tomography (CT) revealed no signs of ischemic bowel; however, laboratory investigations revealed metabolic lactic acidosis, elevation of inflammatory markers, and a remarkable elevation in the serum phosphate level. A prompt surgical evaluation revealed non-occlusive mesenteric ischemia (NOMI). Elevated serum phosphate levels may suggest extensive bowel ischemia or infarction, which can lead to a prompt surgical evaluation, even in the absence of specific radiological findings.

Key words: critical care, emergency medicine, gastroenterology, hyperphosphatemia, phosphate, bowel ischemia/infarction

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Introduction

Diagnosing intestinal ischemia or infarction is challenging, especially in cases of non-occlusive mesenteric ischemia (NOMI). A rapid diagnosis and laparotomy can improve the prognosis and mortality. However, abdominal contrast-enhanced computed tomography (CT) findings are not always specific, and there are no serum markers that are specific enough to diagnose bowel ischemia or infarction. It is often difficult to decide whether or not a prompt surgical evaluation is required.

We herein report a case of NOMI with no specific radiological findings but with a remarkably elevated serum phosphate level.

Case Report

An 86-year-old Japanese woman was admitted to the hospital with a 12-hour history of sudden-onset abdominal pain. Her medical history was significant for diabetes mellitus, hypertension, angina pectoris, and chronic kidney disease. Upon admission, her O_2 saturation was 98% with the administration of 4 L of oxygen, and her limbs were cold with mottled skin. Arterial blood gas testing showed metabolic lactic acidosis (pH 7.110, pCO_2 32 mmHg, HCO_3 9.5 mmol/L, lactate 9.1 mmol/L). Further laboratory investigations revealed leukocytosis, marked inflammatory signs, and renal dysfunction (Table). Furthermore, a remarkable elevation in serum phosphate level (14.4 mg/dL), disproportionate to the renal dysfunction, was noted.

One hour after admission, her systolic blood pressure decreased from 139 to 60 mmHg and she went into shock. Abdominal contrast-enhanced CT revealed a dilated colon and small intestine, containing fluids. However, complete or closed-loop obstruction was not observed. Free air, ascites, and other signs of an ischemic bowel, such as the absence of bowel wall enhancement, pneumatosis intestinalis, hepatic portal venous gas, and bowel wall thickening, were not seen (Fig. 1).

Although there was no clear evidence of bowel obstruction, ischemia, or infarction, the patient underwent emergency laparotomy after six hours of intensive resuscitation. The ileum and whole colon, except for the sigmoid, were discontinuously necrotic despite pulsation of the superior mesenteric artery (Fig. 2). There was no bowel obstruction.

The patient was diagnosed with NOMI. Therefore, ileal resection, subtotal colectomy, and jejunostomy were per-

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Table. **Laboratory Investigation.**

		Normal range (woman)
Blood analysis		
Hemoglobin (g/dL)	17.0	11.6-14.8
Hematocrit (%)	50.7	35.1-44.4
Red blood cell count (per mm^3)	5,080,000	3,860,000- 4,920,000
Platelet count (per mm^3)	185,000	158,000-348,000
White blood cell count (per mm^3)	10,680	3,300-8,600
Neutrophils (%)	76.6	49.7-72.7
Lymphocytes (%)	16.0	24.5-38.9
Total protein (g/dL)	7.0	6.6-8.1
Albumin (g/dL)	3.7	4.1-5.1
Total bilirubin (mg/dL)	0.6	0.4-1.5
Aspartate aminotransferase (U/L)	34	13-30
Alanine aminotransferase (U/L)	19	7-23
Lactate dehydrogenase (U/L)	302	124-222
Alkaline phosphatase (U/L)	93	38-113
Gamma-glutamyl transpeptidase (U/L)	18	9-32
Creatine kinase (U/L)	115	41-153
Total cholesterol (mg/dL)	163	142-248
Urea nitrogen (mg/dL)	64	8-20
Creatinine (mg/dL)	1.58	0.46-0.79
Uric acid (mg/dL)	6.4	2.6-5.5
Glucose (mg/dL)	274	73-109
Hemoglobin A1c (NGSP)(%)	7.8	4.6-6.2
Sodium (mmol/L)	136	138-145
Potassium (mmol/L)	6.2	3.6-4.8
Chloride (mmol/L)	100	101-108
Phosphate (mg/dL)	14.4	2.7-4.6
Calcium (mg/dL)	9.1	8.8-10.1
C-reactive protein (mg/dL)	15.56	0-0.14
Arterial blood gas analysis:		
with administration of 4 L of oxygen		
pH	7.110	7.350-7.450
pO_2 (mmHg)	271	75-100
pCO_2 (mmHg)	32	35-45
Bicarbonate (mmol/L)	9.5	20.0-26.0
Lactate (mmol/L)	9.1	0.5-1.6

formed. The residual jejunal length was 120 cm. After surgery, the patient recovered, and her serum phosphate level immediately returned to its normal range.

Discussion

In the present case, abdominal contrast-enhanced CT revealed no signs of ischemic bowel, such as free air, ascites, or complete or closed-loop obstruction. However, we observed not only signs of metabolic lactic acidosis and inflammation but also a remarkable elevation in serum phosphate (14.4 mg/dL) levels, which was not explained by her renal dysfunction. A prompt surgical evaluation revealed a large portion of necrotic bowel, and the diagnosis of NOMI was made.

NOMI is acute mesenteric ischemia due to splanchnic arteries' vasoconstriction without any structural stenosis and is pathologically characterized by different degrees of ischemia in different segments of the intestine (1). The known risk factors for NOMI are old age, cardiac disease (e.g. myocardial infarction, heart failure, and aortic insufficiency), renal/hepatic disease, and a history of cardiac surgery or chronic hemodialysis (1, 2). Although the presence of elevated lactate levels with metabolic acidosis and inflammatory markers is helpful, they do not prove bowel ischemia; therefore, no laboratory data are considered specific for NOMI (1, 2). In abdominal contrast-enhanced CT, patients with NOMI can show the absence of bowel wall enhancement, pneumatosis intestinalis, collection of hepatic portal venous gas, bowel dilation, edematous bowel wall thickening, and intraperitoneal fluid collection (1-3); however, these signs are non-specific, and only 20-60% of patients have these findings (2). Because there are no laboratory or radiological findings specific enough to diagnose NOMI, diagnosing NOMI and evaluating ischemia are challenging.

In our patient, she had an impaired left ventricular func-

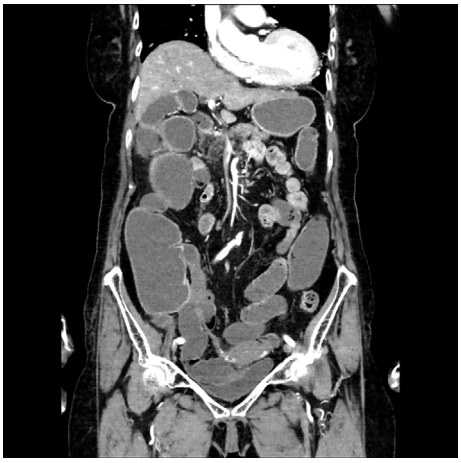

Figure 1. Radiological findings on admission. Contrast-enhanced abdominal computed tomography (CT) revealed a dilated colon and small intestine but no free air, ascites, or complete/closed loop obstruction. The absence of bowel wall enhancement, pneumatosis intestinalis, hepatic portal venous gas, and bowel wall thickening were not seen. There was no clear evidence of mesenteric artery occlusion, bowel ischemia, or infarction.

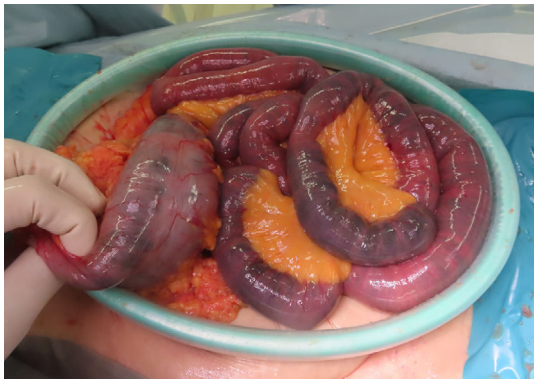

Figure 2. Surgical findings. The ileum and whole colon, except for the sigmoid, were discontinuously necrotic despite the pulsation of the superior mesenteric artery (left side is the head side).

tion, peripheral vascular disease due to diabetes mellitus, and chronic kidney disease as risk factors for NOMI. Laboratory investigations revealed leucocytosis, marked inflammatory signs, and metabolic lactic acidosis; however, abdominal contrast-enhanced CT only revealed a dilated colon and small intestine, indicating no signs of ischemia; thus, it was difficult to decide whether or not the surgical evaluation was needed.

After the surgery, we found that the serum phosphate (14.4 mg/dL) level had been markedly elevated before the surgery, being disproportionately high relative to her renal function (creatinine 1.58 mg/dL). There were no remarkable causes of this hyperphosphatemia, i.e. medications or tumor lysis.

In the 1970s, animal experiments demonstrated that inorganic phosphate levels in serum and peritoneal fluid were significantly increased in intestinal infarction (4). Other animal experiments have also suggested that elevated serum phosphate levels may be a marker of bowel ischemia (5, 6). The mechanism underlying the elevation of serum phosphate levels is believed to involve the release of phosphate from the sloughing intestinal mucosa of the ischemic bowel (7). The elevation of phosphate levels in bowel ischemia has also been reported in humans and can occur with irreversible massive ischemia and necrosis (8, 9). We suspect that the remarkable elevation of serum phosphate levels in our case resulted from the release of inorganic phosphate from the ischemic bowel. The fact that the serum phosphate levels immediately returned to the normal range after surgery may support this idea. However, recent textbooks and guidelines rarely mention the elevation of serum phosphate levels in bowel ischemia or infarction, and sometimes it is not measured, making it a forgotten marker of bowel ischemia or infarction.

Elevated serum phosphate levels are not a sensitive marker of bowel ischemia, but our study suggests that marked hyperphosphatemia may be a specific marker for extensive bowel ischemia or infarction, meriting a prompt surgical evaluation. As the measurement of serum phosphate levels is easy and inexpensive, it can be a useful index. We should be mindful of the serum phosphate level, as it is a forgotten marker of bowel ischemia or infarction. Hopefully, this will improve the mortality and prognoses of patients with bowel ischemia or infarction, especially in the absence of specific radiological findings.

Conclusion

During the evaluation of bowel ischemia or infarction, we should be mindful of the serum phosphate level, as it is a forgotten marker. Even in the absence of specific radiological findings, elevated serum phosphate levels may suggest extensive bowel ischemia or infarction, and a prompt surgical evaluation may improve the mortality and prognosis.

Patient consent was obtained for the publication of this article.

The authors state that they have no Conflict of Interest (COI).

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