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**REVIEW**

- 2408** Assessment of diaphragmatic function by ultrasonography: Current approach and perspectives
Boussuges A, Rives S, Finance J, Brégeon F
- 2425** Mechanisms of resveratrol in the prevention and treatment of gastrointestinal cancer
Wang LY, Zhao S, Lv GJ, Ma XJ, Zhang JB

MINIREVIEWS

- 2438** Clinical benefits of rational-emotive stress management therapy for job burnout and dysfunctional distress of special education teachers
Onuigbo LN, Onyishi CN, Eseadi C
- 2448** Functions and mechanisms of chemokine receptor 7 in tumors of the digestive system
Xin Q, Sun Q, Zhang CS, Zhang Q, Li CJ
- 2464** Computer navigation-assisted minimally invasive percutaneous screw placement for pelvic fractures
Yu T, Cheng XL, Qu Y, Dong RP, Kang MY, Zhao JW

ORIGINAL ARTICLE**Case Control Study**

- 2473** Elevated serum growth differentiation factor 15 in multiple system atrophy patients: A case control study
Yue T, Lu H, Yao XM, Du X, Wang LL, Guo DD, Liu YM

Retrospective Study

- 2484** Research on diagnosis-related group grouping of inpatient medical expenditure in colorectal cancer patients based on a decision tree model
Wu SW, Pan Q, Chen T
- 2494** Clinical outcomes of sacral neuromodulation in non-neurogenic, non-obstructive dysuria: A 5-year retrospective, multicentre study in China
Meng LF, Zhang W, Wang JY, Zhang YG, Zhang P, Liao LM, Lv JW, Ling Q, Wei ZQ, Zhong T, Xu ZH, Wen W, Li JY, Luo DY
- 2502** Magnetic resonance imaging features of minimal-fat angiomyolipoma and causes of preoperative misdiagnosis
Li XL, Shi LX, Du QC, Wang W, Shao LW, Wang YW
- 2510** Evaluation of internal and shell stiffness in the differential diagnosis of breast non-mass lesions by shear wave elastography
Xu P, Wu M, Yang M, Xiao J, Ruan ZM, Wu LY

Observational Study

- 2520** Recovery from prolonged disorders of consciousness: A dual-center prospective cohort study in China
Chen WG, Li R, Zhang Y, Hao JH, Du JB, Guo AS, Song WQ
- 2530** Gene testing for osteonecrosis of the femoral head in systemic lupus erythematosus using targeted next-generation sequencing: A pilot study
Sun HS, Yang QR, Bai YY, Hu NW, Liu DX, Qin CY
- 2542** Real-time three-dimensional echocardiography predicts cardiotoxicity induced by postoperative chemotherapy in breast cancer patients
Zhou F, Niu L, Zhao M, Ni WX, Liu J

Prospective Study

- 2554** Epidemiological and clinical characteristics of COVID-19 patients in Hengyang, Hunan Province, China
Zhong ZF, Huang J, Yang X, Peng JL, Zhang XY, Hu Y, Fu N, Lin HL, Jiang B, Tian YY, Yao HY, Deng LP, Tang XQ, Zhou JC, Tang J, Xie X, Liu Q, Liu J, Dou CY, Dai RJ, Yan B, Yang XF

CASE REPORT

- 2566** Demyelinating polyneuropathy and lymphoplasmacytic lymphoma coexisting in 36-year-old man: A case report
Rozlucka L, Semik-Grabarczyk E, Pietrukaniec M, Żak-Golqb A, Grabarczyk M, Grosicki S, Holecki M
- 2574** Lenvatinib for large hepatocellular carcinomas with portal trunk invasion: Two case reports
Komiyama S, Numata K, Moriya S, Fukuda H, Chuma M, Maeda S
- 2585** Biopsy-proven acute phosphate nephropathy: A case report
Medina-Liabres KRP, Kim BM, Kim S
- 2590** Endodontic management of the maxillary first molar with special root canals: A case report and review of the literature
Zhang ZH, Yao HL, Zhang Y, Wang X
- 2597** Novel approach for the diagnosis of occult cytomegalovirus cholangitis after pediatric liver transplantation: A case report
Liu Y, Sun LY, Zhu ZJ, Qu W
- 2603** Anti-N-methyl-D-aspartate-receptor antibody encephalitis combined with syphilis: A case report
Li XY, Shi ZH, Guan YL, Ji Y
- 2610** Collision tumor of squamous cell carcinoma and neuroendocrine carcinoma in the head and neck: A case report
Wu SH, Zhang BZ, Han L

- 2617** Successful treatment of plasma exchange-refractory thrombotic thrombocytopenic purpura with rituximab: A case report
Chen J, Jin JX, Xu XF, Zhang XX, Ye XN, Huang J
- 2623** Ovarian endometrioid carcinoma resembling sex cord-stromal tumor: A case report
Wei XX, He YM, Jiang W, Li L
- 2629** Headache and sick sinus syndrome: A case report
Bi YC, Gong L
- 2634** Modified pararectus approach for treatment of atypical acetabular anterior wall fracture: A case report
Wang JJ, Ni JD, Song DY, Ding ML, Huang J, He GX, Li WZ
- 2641** Pleomorphic rhabdomyosarcoma of the spermatic cord and a secondary hydrocele testis: A case report
Chen X, Zou C, Yang C, Gao L, Bi LK, Xie DD, Yu DX
- 2647** Comprehensive treatment of rare multiple endocrine neoplasia type 1: A case report
Ma CH, Guo HB, Pan XY, Zhang WX
- 2655** Low-grade fever during COVID-19 convalescence: A report of 3 cases
Zhuang SF, Hu J, Qiao N, Lan ZH, Lai JY, Wu JG, Wu XY
- 2662** Diffuse alveolar hemorrhage with histopathologic manifestations of pulmonary capillaritis: Three case reports
Xie J, Zhao YY, Liu J, Nong GM
- 2667** Presentation of gallbladder torsion at an abnormal position: A case report
Chai JS, Wang X, Li XZ, Yao P, Yan ZZ, Zhang HJ, Ning JY, Cao YB

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Biopsy-proven acute phosphate nephropathy: A case report

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Abstract

BACKGROUND

Acute phosphate nephropathy (APN) is a disease that can occur when exposed to high doses of phosphate. The most common cause of APN is the use of oral sodium phosphate for bowel cleansing preparations. However, there are other less commonly known sources of phosphate that are equally important. To date, our literature search did not identify any report of excessive dietary phosphate as a cause of APN.

CASE SUMMARY

We report an unusual case of a 39-year-old diabetic male who presented with epigastric pain and oliguria. Work-up showed elevated serum creatinine, potassium, and calcium-phosphate product, and metabolic acidosis. The patient was admitted in the intensive care unit and received emergent renal replacement therapy. Kidney biopsy revealed tubular cell injury with transparent crystal casts positive for Von Kossa staining, which established the diagnosis of APN.

CONCLUSION

This case confirmed that APN may occur with other sources of phosphorus, highlighting the importance of good history taking and kidney biopsy in patients with predisposing factors for APN. Raising awareness on the possibility of APN and its timely recognition and management is imperative so that appropriate measures can be instituted to prevent or delay its progression to end stage renal disease.

Key words: Hyperphosphatemia; Nephrocalcinosis; Acute phosphate nephropathy; Renal insufficiency; Chronic kidney disease; Biopsy; Case report

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Core tip: The classic case of acute phosphate nephropathy (APN) is caused by oral sodium phosphate for bowel cleansing preparations. In this case report, we present a rare incident of biopsy-proven APN caused by excessive dietary phosphate intake in a 39-

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year-old diabetic male. APN was diagnosed by history of increased dietary phosphorus intake, clinical presentation of acute kidney injury, laboratory findings of hyperphosphatemia and elevated calcium phosphate product, and kidney biopsy findings, which showed tubular crystals positive for Von Kossa stain. This case highlights the importance of good history taking and kidney biopsy for the diagnosis of APN.

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INTRODUCTION

Acute phosphate nephropathy (APN), previously called acute nephrocalcinosis, is a disease that can occur when exposed to high doses of phosphate. The classic cause of APN is the use of oral sodium phosphate for bowel cleansing preparations^[1-3]. Jahan *et al*^[4] reported a case of APN secondary to excessive intake of sodium phosphate tablets for hypophosphatemia. Aside from these, there are other less known sources of phosphate that are equally important. To date, our literature search did not identify any report of excessive dietary phosphate as a cause of APN. We report a case of a 39-year-old diabetic male who developed APN secondary to excessive dietary phosphate intake.

CASE PRESENTATION

Chief complaints

Epigastric pain for 5 d and acute onset oliguria.

History of present illness

A 39-year-old man with diabetes mellitus was admitted at Seoul National University Bundang Hospital for evaluation and management of acute kidney injury (AKI). He consulted at the emergency room due to epigastric pain that started five days prior and decreased urine volume noted on the day of the visit. There was no accompanying vomiting, diarrhea, or fever.

History of past illness

The patient has been on insulin for diabetes mellitus since 5 years ago and has a history of being hospitalized for chronic pancreatitis and alcoholic hepatitis one year ago. He had no previous surgeries. He denied use of angiotensin converting enzyme inhibitor or angiotensin receptor blocker, and herbal or dietary supplements.

Personal history

The patient had recently travelled to Japan. He worked as a chef, and upon detailed history taking, the patient claimed his diet only consisted of tomato meatball pasta and carbonara for nine consecutive days prior to his hospitalization. Each serving of meatball pasta contained 100 g shredded mozzarella cheese (656 mg phosphorus/100 g) and 1 slice of cheddar cheese (936 mg phosphorus/100 g), while each serving of carbonara included 100 g mozzarella cheese and 125 g camembert cheese (347 mg phosphorus/100 g). Pasta also has 253 mg phosphorus for every 100 g. The amount of phosphorus he consumed is estimated to be more than twice the recommended intake for adult men.

Physical examination

Upon admission, vital signs were normal. Physical examination showed signs of volume overload.

Laboratory examinations

Laboratory test results showed azotemia (serum creatinine 12.85 mg/dL and urea nitrogen 85 mg/dL) and elevated potassium (6.7 mmol/L), uric acid (9.0 mg/dL) and phosphorus (3.62 mmol/L) levels. Patient was hypocalcemic at 1.65 mmol/L (corrected calcium 1.79 mmol/L). Serum magnesium (0.66 mmol/L) was normal.

Parathyroid hormone was within acceptable limits for chronic kidney disease (CKD) patients. He also presented with uncompensated metabolic acidosis (pH 7.147, bicarbonate 11.8 mmol/L). Patient's latest serum creatinine before hospitalization was 1.68 mg/dL three months prior (estimated glomerular filtration rate 50.8 mL/min per 1.73 m²). Due to the rapid deterioration of renal function, further work-up was requested to establish other possible causes of AKI and/or CKD progression. Antineutrophil cytoplasmic antibodies (ANCA), fluorescent antinuclear antibody (FANA), and anti-streptolysin O (ASO) were negative. Patient had normal C3 and C4 levels (95 mg% and 26.30 mg%, respectively). Random urine protein/creatinine ratio was 1322.81 mg/g.

Imaging examination

On ultrasound, the kidneys were normal-sized (right 12.1 cm and left 11.6 cm), with increased cortical echogenicity. No suspicious focal lesions or signs of urinary obstruction were noted.

Further diagnostic work-up and Pathological examination

Kidney biopsy under ultrasound guidance was performed (Figure 1A) on the fourth hospital day. The kidney biopsy revealed normal to mildly enlarged glomeruli, but with partially shrunk glomerular tufts; marked focal atrophy, tubular cell injury with some transparent crystal casts. There was also marked focal interstitial fibrosis and focal infiltration of mononuclear cells and a few eosinophils. Fibrointimal thickening of blood vessels were also noted. The immunofluorescence was negative for immunoglobulins and complement factors. The tubular crystal materials were positive for von Kossa staining, which established the diagnosis of APN (Figure 1B).

FINAL DIAGNOSIS

The final diagnosis of the presented case is acute phosphate nephropathy.

TREATMENT AND FOLLOW-UP

An emergent acute renal replacement therapy (RRT) was performed on the first hospital day. During hospital stay, the patient was continuously assessed for improvement of symptoms and renal function recovery. Monitoring of renal function and electrolytes was likewise done (Table 1). Unfortunately, the patient did not recover renal function and was still hemodialysis-dependent during the 3-mo follow-up period. He was maintained on and compliant with thrice weekly 4-h hemodialysis sessions. He did not report of any adverse event. On follow-up, patient was asymptomatic, and had normal electrolyte levels. His latest serum creatinine was 7.84 mg/dL.

DISCUSSION

We reported a unique case of APN diagnosed by history of increased dietary phosphorus intake, AKI, hyperphosphatemia, and kidney biopsy findings. Although the electrolyte imbalances present in the patient can be observed in renal failure of any etiology, the positive Von Kossa staining of the tubular crystals on histopathologic examination strengthens our diagnosis of APN.

APN is a tubulointerstitial nephropathy caused by the deposition of calcium phosphate crystals in the renal tubular epithelial cells, causing reactive oxygen damage, resulting in acute tubular injury, and subsequently tubular atrophy and interstitial fibrosis, with concomitant loss of kidney function. APN occurs in conditions that result in calcium or phosphate elevations in the blood or urine such as sarcoidosis, malignancy, medullary sponge kidney, and cystinosis.

Since phosphate concentration is regulated by oral phosphorus load and renal excretion, hyperphosphatemia can be attributed to excess phosphorus intake over a short period of time. The normal dietary intake of phosphorus among adults is 1.5 g but individuals with high dairy product intakes have diets with higher phosphorus density values. Other risk factors for the development of APN include female gender, advanced age, hypovolemia, ileus, hypertension, diabetes mellitus, liver disease, and renal impairment^[5]. Tissue necrosis, a regular feature of liver failure, can lead to increased phosphate levels^[6], especially in cases of failed spontaneous recovery from liver disease^[7]. Liver dysfunction, such as in cases with portal hypertension, can also

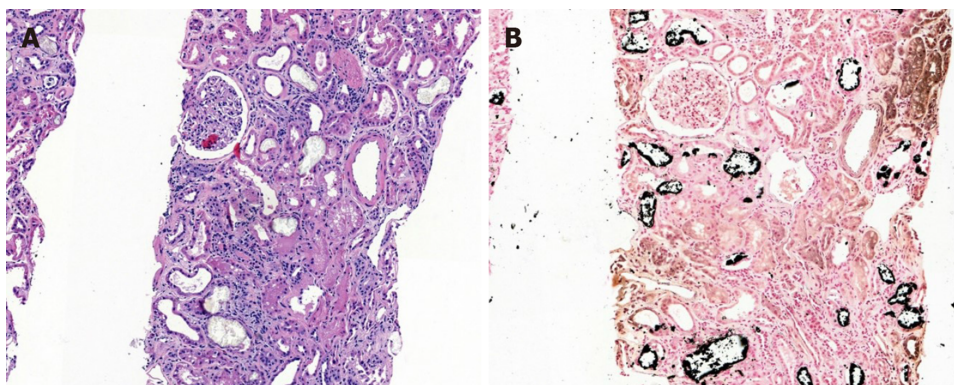


Figure 1 Renal biopsy specimen under light microscopy. A: Hematoxylin and eosin stain; B: Von Kossa stain. Tubular calcium phosphate crystals staining positive. Magnification $\times 100$.

possibly increase the permeability of colonic mucosa to sodium phosphate^[8]. The use of medications contributing to renal hypoperfusion such as renin-angiotensin system inhibitors, diuretics and non-steroidal anti-inflammatory drugs also predisposes to APN. Risk factors in this case were chronic hepatitis, diabetes mellitus and renal impairment.

Diagnosis

The classic diagnostic criteria for APN include AKI, recent exposure to sodium phosphate, no evidence of hypercalcemia, renal biopsy findings of diffuse tubular injury with calcium phosphate deposits, and no other significant pattern of renal injury. However, the diagnosis of APN should also be considered in patients with other sources of phosphate, especially in patients with any of the risk factors. A kidney biopsy should be done in such patients for definitive diagnosis. The most important findings on biopsy are calcium phosphate deposits primarily in the tubular lumen and cytoplasm of tubular epithelial cells which stain intensely with Von Kossa stain^[9], and tubular injury, with tubular atrophy and interstitial fibrosis.

Calcium-phosphate product (CPP) is generally regarded as the indicator of the risk of calcium phosphate precipitation in the kidney. Normal range is $21\text{--}45\text{ mg}^2/\text{dL}^2$. CPP usually increases in cases of APN. Our patient's CPP at the time of presentation was $80\text{ mg}^2/\text{dL}^2$, strengthening the diagnosis of APN.

Management

Patients who develop APN require rapid correction of electrolyte abnormalities and aggressive fluid resuscitation to promote renal phosphate excretion and prevent further calcium phosphate precipitation. However, aggressive fluid therapy should not be instituted in patients who are oliguric and with signs of fluid overload. In mild cases, conservative treatment such as phosphate-binding resins and calcium gluconate, is recommended. Hemodialysis is indicated in cases of severe hyperphosphatemia, symptomatic hypocalcemia, and severe renal failure with associated complications. Kidney injury in APN is usually reversible^[10] however despite appropriate management, it may progress to chronic irreversible kidney disease. In a cohort of 21 patients by Markowitz *et al*^[11], all patients developed chronic renal insufficiency and 19% eventually had end stage renal disease (ESRD) that required RRT.

CONCLUSION

Since most cases of APN are clinically silent or often present with non-specific symptoms, its true incidence cannot be determined. It is very likely that it is underrecognized as a cause of AKI and CKD. This case confirmed that APN may occur with excessive dietary phosphorus intake. Therefore, it is important to emphasize proper dietary restrictions to patients with predisposing factors such as renal insufficiency. APN should be considered in patients with acute onset renal failure of unknown cause or patients with pre-existing CKD who develop rapid worsening of kidney function for no apparent reason, especially in patients who have risk factors for APN. A kidney biopsy is essential to provide a definitive diagnosis.

Physicians should be cognizant of APN as most of the studies have revealed its long-term consequences such as irreversible, progressive renal injury. Timely

Table 1 Relevant laboratory parameters at different time points

	Reference range	Day -90, baseline	Day 1, admission, and start of dialysis	Day 30	Day 90, follow-up
Urea nitrogen (mg/dL)	10-26	26	85	30	37
Creatinine (mg/dL)	0.7-1.4	1.68	12.85	7.32	7.84
Na (mmol/L)	135-145	-	133	139	133
K (mmol/L)	3.5-5.5	-	6.7	5.8	5.3
P (mg/dL)	2.5-4.5	4.5	11.2	6.1	4.3
Ca (mg/dL)	8.8-10.5	9.3	6.6	6.6	9.4
Mg (mg/dL)	1.5-2.0	-	1.6	-	-
Intact PTH (pg/mL)	152-684 ¹	-	205	-	-
Albumin (g/dL)	3.3-5.2	4.0	3.3	2.3	3.2
pH	7.38-7.469	-	7.147	7.365	-
Bicarbonate (mmol/L)	21-29	-	11.8	25.1	-
Carbon dioxide (μmol/mol)	24-31	-	14	28	-

¹CKD Stage 3a-5 not on dialysis: Optimal PTH level is not known. CKD Stage 5 on dialysis: Approximately 2-9 times the upper limit of normal for the assay^[12]. Na: Sodium; K: Potassium; P: Phosphorus; Ca: Calcium; Mg: Magnesium; PTH: Parathyroid hormone; CKD: Chronic kidney disease.

recognition and management of APN is imperative so that appropriate measures can be instituted to prevent or delay its progression to ESRD.

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