
به نام خدا
Acbka

Acute phosphate nephropathy

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Tehran - IRAN

Acute phosphate nephropathy
Definition

- **APhN** is a clinical pathological entity characterized by:
  - Acute and subsequent chronic renal failure following exposure to:
    - Oral sodium phosphate (OSP) bowel purgatives
    - Sodium phosphate-containing enemas

Kidney International (2009) 76, 1027–1034

Acute phosphate nephropathy
Pathogenesis

- Phosphate exposure have a critical role
- In proximal tubule Na-P2a and Na-P 2c expression is down regulated by increases in serum P or PTH
- Na-P-2b present in the small intestine , levels increase in response to hypophosphatemia and vitamin D
- In contrast to renal Na-P- IIa, intestinal NaP-IIb expression requires days to respond to physiological changes.

Kidney International (2009) 76, 1027–1034

Acute phosphate nephropathy

Pathogenesis

Oral -
Enema

Bowel lumen

OSP increase water loss due to osmotic diarhea and
increase phosphate absorption by Na-P 2b

Intravascular

Hypovolemia + hyperphosphatemia

PT water reabsorption +
Phosphate excretion due to down regulated Na-P2a and Na-P2c due to hyperphosphatemia

DLOH :

Water reabsorption +
Impermible to Ca and P

DT: consentrated watr + high P + high Ca → *extensive calcification by* expression of hyaluronan and osteopontin → obstruction, direct tubular epithelial injury, and inflamation

Acute phosphate nephropathy
Pathogenesis

- **Hyperphosphatemia occurs in the following settings:**
  - **Excessive ingestion of phosphate over a short time period**
  - **Massive release of intracellular phosphate i.e. TLS , rhabdomyolysis**
  - **Phosphate ingestion in the setting of impaired gastrointestinal motility (increased absorptive time)**
  - **Renal dysfunction leading to reduced excretion**

Oral Sodium Phosphate

- Is a hyperosmotic laxative that acts by drawing water into the Gi tract.
- Long time used as a laxative But began to used as a purgative for colonoscopy in 1990

Oral Sodium Phosphate

- Frequently given in favor of standard polyethylene glycol (PEG)-based lavage solutions because of:
  - The smaller required volume
  - Results in better patient compliance
  - Improved colonic cleansing

Oral Sodium Phosphate

- **Recommended regimen of OSP solution consisted of :**
  - **Two 45-ml doses taken 12 h apart, the evening before and the morning of colonoscopy.**
  - **Each 45-ml dose contained 5.8 g of elemental phosphorus.**
  - **Far exceeding the usual dietary intake of 1 g/day**

Oral Sodium Phosphate

- **On 11 December 2008, following continuing reports of APhN :**

  - **FDA issued that over-the-counter OSP products should no longer be used**
  - **but OSP tablets, offered by prescription only.**

Oral Sodium Phosphate

- **FDA warning in 2014:**
  - **Using more than one dose in 24 hours of OSP to treat constipation can cause :**
    - **"Rare but serious harm to the kidneys and heart, and even death"**

Oral Sodium Phosphate

- **Oral sodium phosphate remains available by prescription in a tablet form under the brand names *Visicol and Osmoprep***
  - **A regimen of 20 tablets of Visicol, administered as three tablets with at least eight ounces of clear fluid every 15 min , has a cumulative sodium phosphate content that is near identical to a 45-ml of OSPS.**
  - **Osmoprep has largely replaced Visicol, and current recommendations are for the second administration to consist of 12 rather than 20 tablet**

سدیم فسفات دی بازیک + سدیم فسفات مونو بازیک انما مقعدی ۷ g/۱۹ g/۱۱۸mL (انما سدیم فسفات دی بازیک هپتاهیدرات / سدیم فسفات مونوبازیک)
(SODIUM PHOSPHATE DIBASIC HEPTAHYDRATE / SODIUM PHOSPHATE MONOBASIC MONOHYDRATE ENEMA RECTAL 7 g/19 g/118mL
)SODIUM PHOSPHATE DIBASIC+SODIUM PHOSPHATE MONOBASIC ENEMA RECTAL 7 g/19 g/118mL

صاحب برند : دارو سازی بهوزان

صاحب پروانه : دارو سازی بهوزان

قیمت هر بسته : ۲۲۴.۲۰۰ ریال

بسته بندی : 1 BOTTLE, UNIT-DOSE in 1 BOX

سدیم فسفات دی بازیک هپتا هیدرات/سدیم فسفات مونوبازیک مونوهیدرات انما انما مقعدی ۷ g/۱۹ g/۱۱۸mL (انما سدیم فسفات دی بازیک هپتاهیدرات / سدیم فسفات مونوبازیک)
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صاحب برند : عماد درمان پارس

صاحب پروانه : عماد درمان پارس

قیمت هر بسته : ۲۲۴.۲۰۰ ریال

بسته بندی : 118 MILLILITER in 1 BOTTLE

کد فراورده : ۳۷۷۸۶۳۷۰۶۱۷۱۰۹۰۸

سدیم فسفات دی بازیک هپتا هیدرات/سدیم فسفات مونوبازیک مونوهیدرات انما انما مقعدی ۷ g/۱۹ g/۱۱۸mL (انما سدیم فسفات دی بازیک هپتاهیدرات / سدیم فسفات مونوبازیک)
(SODIUM PHOSPHATE DIBASIC HEPTAHYDRATE / SODIUM PHOSPHATE MONOBASIC MONOHYDRATE ENEMA RECTAL 7 g/19 g/118mL
)SODIUM PHOSPHATE DIBASIC HEPTAHYDRATE / SODIUM PHOSPHATE MONOBASIC MONOHYDRATE ENEMA RECTAL 7 g/19 g/118mL

صاحب برند : عماد درمان پارس

صاحب پروانه : عماد درمان پارس

قیمت هر بسته : ۲۲۴.۲۰۰ ریال

بسته بندی : 118 MILLILITER in 1 BOTTLE

کد فراورده : ۳۷۷۸۶۳۷۰۶۱۷۱۰۹۰۸

کلین ردی تو یوز انما مقعدی ۷ g/۱۹ g/۱۱۸mL (انما سدیم فسفات دی بازیک هپتاهیدرات / سدیم فسفات مونوبازیک) ⚠ دارای پروانه فوریتی
(SODIUM PHOSPHATE DIBASIC HEPTAHYDRATE / SODIUM PHOSPHATE MONOBASIC MONOHYDRATE ENEMA RECTAL 7 g/19 g/118mL
)CLEEN READY-TO-USE ENEMA RECTAL 7 g/19 g/118mL

صاحب برند : Recordati S.p.a.

صاحب پروانه : رضا دارو پارس

قیمت هر بسته : - ریال

بسته بندی : 133 MILLILITER in 1 BOTTLE, PUMP

کد فراورده : ۹۵۳۱۶۶۱۶۵۳۴۳۲۳۶۶

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Excessive elevation of serum phosphate during tumor lysis syndrome: Lessons from a particularly challenging case

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Acute Phosphate Nephropathy

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Keywords. acute kidney injury, rhabdomyolysis, electrolyte imbalance, hyperphosphatemia

We present acute phosphate nephropathy in a 28-year-old man, which was developed after a car accident due to rhabdomyolysis. Treatment of acute kidney injury was done with administration of sodium bicarbonate.

IJKD 2014;8:246-9
www.ijkd.org

CLINICAL PRESENTATION AND *PROGNOSIS*

- **Acute and reversible kidney injury**
  - occurs within hours of the administration of OSP
  - associated with excessive dosing of OSP or other risk factors for hyperphosphatemia
  - AKI is due to prerenal factors or ATN rather than acute phosphate nephropathy
  - Occurs in setting of severe hyperphosphatemia and hypocalcemia, leading to tetany, cardiac arrest, and, in some cases, death
  - Who survive the immediate event typically return to normal or near-normal renal function.

CLINICAL PRESENTATION AND *PROGNOSIS*

- **Acute phosphate nephropathy**
  - **Occur in asymptomatic patients, days to months following OSP administration**
  - **The serum P and Ca are normal when kidney injury is discovered**
  - **Confirmation by renal biopsy**

Acute phosphate nephropathy
Diagnostic Criteria

- AKI
- Recent exposure to OSP bowel purgative
- Renal biopsy findings of acute and chronic tubular injury with abundant calcium phosphate deposits
- No evidence of hypercalcemia or conditions associated with hypercalcemia
- No other significant pattern of renal injury

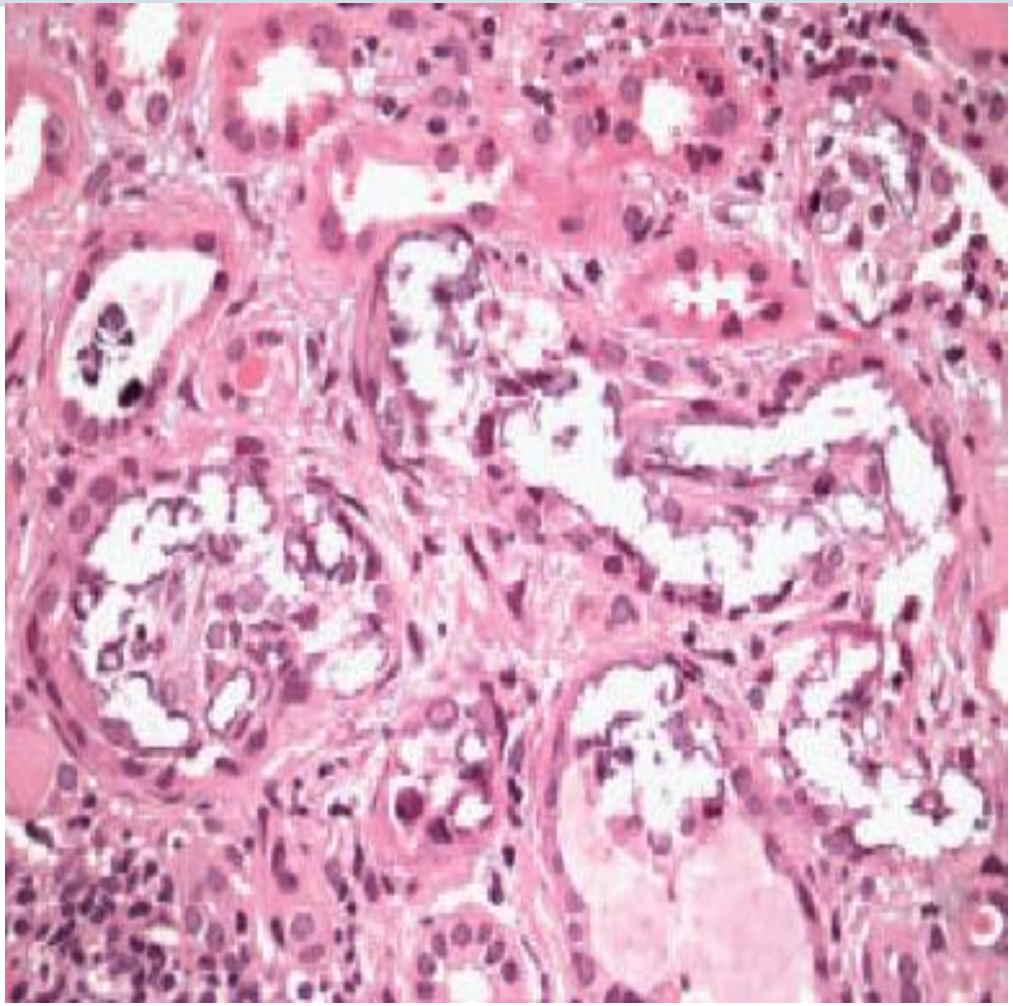
Acute phosphate nephropathy
Pathology

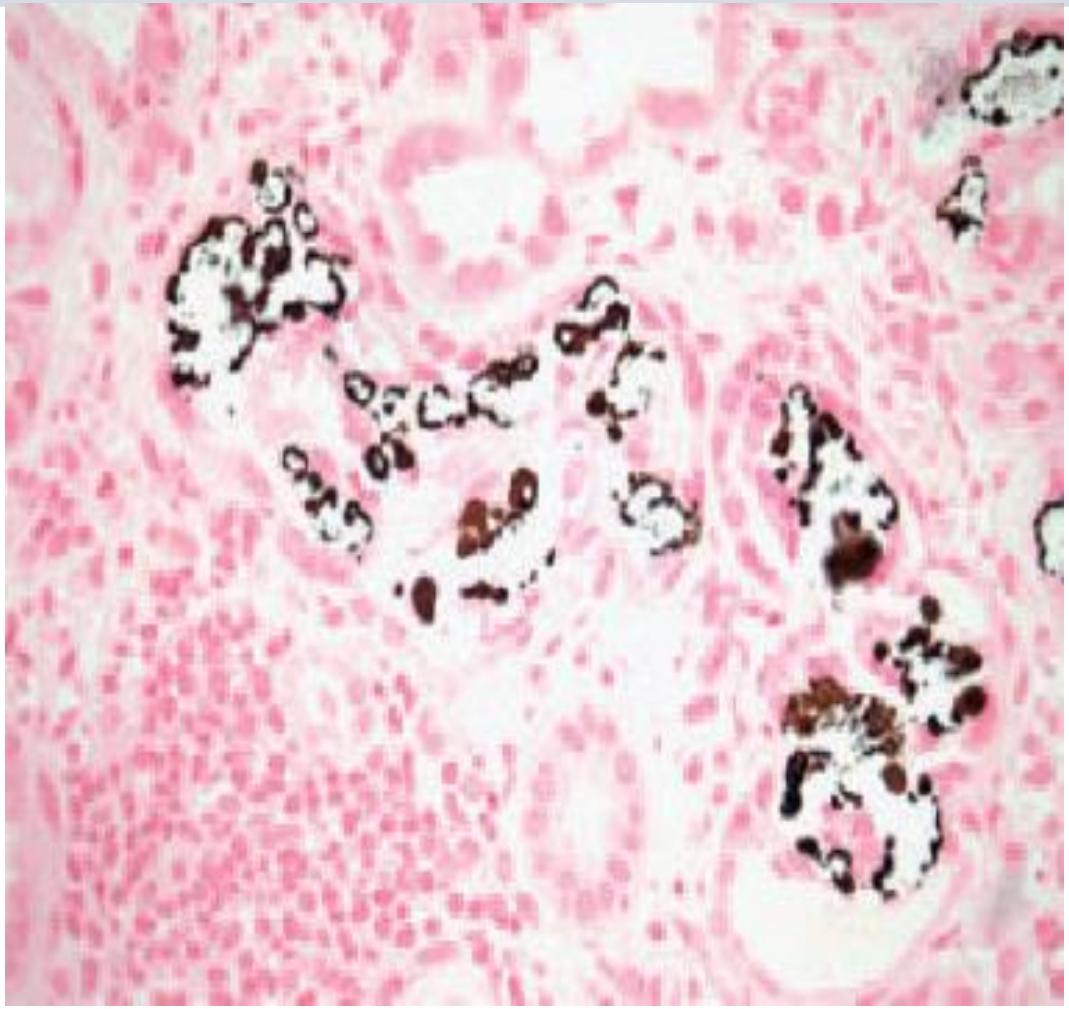
- Renal biopsy findings in APhN involve the tubules
  - Within 3 weeks of OSP exposure, acute tubular degenerative changes predominate and resemble findings seen in ATN.
  - More than 3 weeks following OSPS exposure exhibit evidence of chronicity in the form of tubular atrophy and interstitial fibrosis
  - This pattern of renal injury described as an acute and chronic tubulointerstitial nephropathy

Acute phosphate nephropathy
Pathology

- Regardless of the degree of acuity or chronicity, **the hallmark of APhN is abundant tubular and less prominent interstitial calcium phosphate deposits**
- The extent of tubular calcification is dependent on adequacy of tissue sampling but in biopsies with 10 or more glomeruli,>30 calcifications are typically encountered and >100 calcifications can be seen

Acute phosphate nephropathy
Pathology





Acute phosphate nephropathy
Risk factors

- Age>60
  - more severe hyperphosphatemia in the elderly:
    - may relate to both age-related decline in GFR
    - increases in intestinal transit time
- CKD stage 3 (GFR<60)
  - Low GFR limits renal phosphate excretion and exposes the fewer functioning nephrons to a higher concentration of phosphate

Acute phosphate nephropathy
Risk factors

- **Hypertension :**
  - **The effect of HTN relate to its effect on renal function and the associated vascular scarring, which can impair physiological adjustments to hypovolemia.**
- **Female gender:**
  - **Risk may be estrogen dependent (majority of women with APhN are post-menopausal)**
  - **Weight is a critical determinant of the hyperphosphatemia following the use of OSP**

Acute phosphate nephropathy
Risk factors

- ACEis, ARBs, and diuretics are known to:
  - Exacerbate the pre-renal state
  - Decrease angiotensin-II-dependent bicarbonate reabsorption in the PT, inducing bicarbonaturia and promoting calcium phosphate precipitation in the DT
- Diabetes mellitus
- NSAIDS
- Phosphate dose
- Interval between OSP dosing
- Adequacy of hydration

- ***One might predict that the risk of APhN would increase in parallel with the number of risk factors***

Prevention of APhN

- Avoiding OSP in high-risk patients
- Aggressive hydration before, during, and after OSP administration
- Minimizing the dose of OSP
- Maintaining a minimum of a 12 h interval between OSP administrations

Treatment of APhN

- There is no specifc treatment for established APhN

- Acute HD is likely benefit in patient who is diagnosed within 12 to 24 hours after OSP and still has marked hyperphosphatemia

REFERENCES

- Iranian Journal of Kidney Diseases | Volume 8 | Number 3 | May 2014
- Kidney International (2009) 76, 1027–1034
- J Am Soc Nephrol 16: 3389–3396, 2005
- Arch Intern Med 2012;172:263.
- Am J Gastroenterol 1990; 85:422
- Nephrol Dial Transplant 2005; 20:850.
- N Engl J Med 2008; 359:951
- Am J Gastroenterol 1993; 88:929.
- Arch Intern Med 2008; 168:593.
- J Am Soc Nephrol 2007; 18:3199.
- J Am Soc Nephrol 2007; 18:3192.
- Am J Gastroenterol 2007; 102:2655.
- Gastrointest Endosc 2006; 64:544.
- Am J Gastroenterol 2008; 103:2707
- Am J Kidney Dis 2016; 67:609.
- Kidney Int 2016; 90:13.

Thank you very much