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# Stump the Consultants: Case Presentation

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# Disclosure

I do not have anything to disclose.

# Case Presentation

9-year-old girl with end-stage kidney disease secondary to perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) vasculitis maintained on intermittent hemodialysis for over 1-year.

On routine monthly labs, she was found to have a phosphorous level of 0.7 mg/dL. Repeat labs obtained showed phosphorous of 1 mg/dL.

She was asymptomatic and physical exam was normal.

# Medical History

| Past Medical History | Past Surgical History | Family History |
| --- | --- | --- |
| • ESKD 2/2 p-ANCA Vasculitis<br>• Posterior Reversible Encephalopathy Syndrome | • HD catheter placement | • No family history of kidney disease, hypertension, autoimmune diseases, dialysis or kidney transplant |

# Laboratory Findings

| | Value (mg/dL) | | Value |
|---|---|---|---|
| **Sodium** | 140 | **Calcium** | 10.2 mg/dL |
| **Potassium** | 4.2 | **Albumin** | 3.4 |
| **Chloride** | 103 | **Intact PTH** | 104 pg/mL |
| **CO2** | 26 | **Alkaline phosphatase** | 150 u/L |
| **BUN** | 35 | **Vitamin D 25-OH** | 22.5 ng/mL |
| **Creatinine** | 6.49 | **Vitamin D 1,25 DiHydroxy** | < 8 |
| **Glucose** | 99 | **Vitamin D3 1,25 DiHydroxy** | <8 |
| **Phosphorous** | 1 | **Vitamin D 2 1,25 DiHydroxy** | <8 |

# Medications

| Medications | |
|---|---|
| Albuterol 90 mcg/inh as needed | Lansoprazole 30 mg daily |
| Amlodipine 10 mg daily | Miralax 17 g daily |
| Aspirin 81 mg daily | Nephronex 3 mL daily |
| Cholecalciferol 2000 U daily | Prednisone 10 mg daily |
| Flovent 110 mcg/inh twice daily | Senna 8.6 mg daily |
| Folic Acid 2 mg daily | Venofer 32 mg IV weekly |
| Labetalol 50 mg twice daily | Zyrtec 5 mg daily |

## Question 1: What is the differential diagnosis for hypophosphatemia in this patient?

## Question 1.5: What would you do next for this patient?

# Initial Management

- Sodium phosphate 11 mMol IV bolus
- Repeat phosphorous level: 2.7 mg/dL
- Calcium carbonate was taken appropriately away from meals
    - Discontinued with normal calcium levels
- Started K-Phos Neutral tablets for supplemental phosphorous
- Encouraged increasing nutritional intake of cheese and other phosphorous containing foods

# Differential diagnosis for hypophosphatemia

- Urinary phosphate wasting
- Chronic use of calcium carbonate or other phosphorous binders
- Decreased nutritional intake
- Gastrointestinal malabsorption

# Differential diagnosis for hypophosphatemia

| | |
|---|---|
| Urinary Phosphate Wasting | • Patient is anuric |
| Chronic use of phosphorous binders | • Calcium carbonate was taken as prescribed, away from meals |
| Decreased nutritional intake | • Normalized protein catabolic rate was 1.05 g/kg/day |
| Gastrointestinal malabsorption | • No abdominal pain, diarrhea or vomiting |

# Question 2: What are some medications or supplements that can cause hypophosphatemia?

# Medications that may cause hypophosphatemia

| | |
|---|---|
| Decreased intestinal phosphate absorption | • Phosphorous binders |
| Shifts of extracellular phosphate into cells | • Salicylate poisoning<br>• Catecholamine action: epinephrine, dopamine, xanthine |
| Increased urinary phosphate excretion | • Carbonic anhydrase inhibitors<br>• Diuretics<br>• Theophylline<br>• Bisphosphonates<br>• Anticonvulsants |

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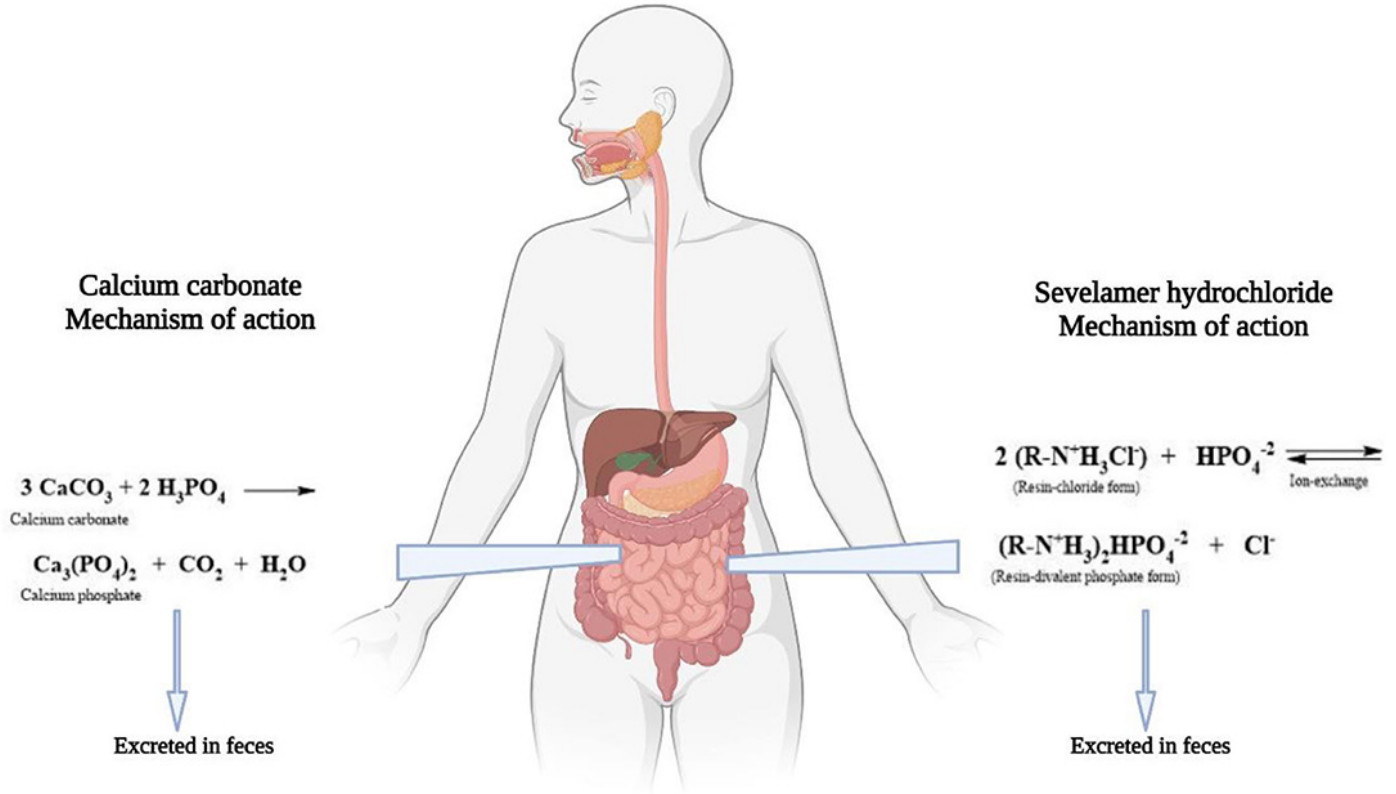
# Phosphorous Binders

- Mechanism of action: reduce serum phosphorous levels by binding to ingested phosphorous in the gastrointestinal tract.
    - Forms insoluble complexes that are not absorbed

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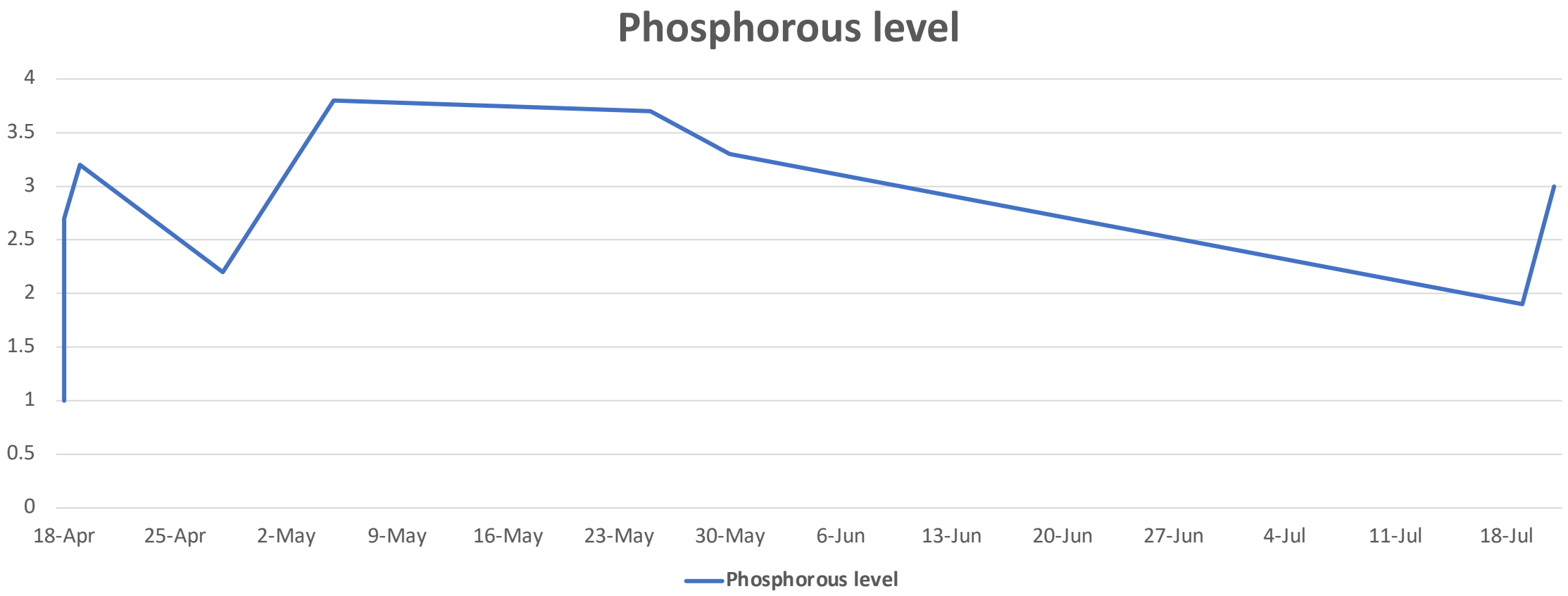
# Common Phosphorous Binders

| | |
|---|---|
| Calcium-containing binders | • Calcium carbonate<br>• Calcium acetate |
| Calcium-free binders | • Aluminum-based binders<br>• Magnesium-based binders<br>• Sevelamer<br>• Lanthanum<br>• Sucroferric oxyhydroxide |

# Back to our patient – after our initial management

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# Our patient continued

- During a session in dialysis – a nurse had mentioned chalk, in which the patient reports she sometimes consumes.
- On further investigating – we discovered the type of chalk she may have been consuming.
- Calabash chalk – generic term to describe geophagical elements
    - Consumed by many pregnant women and breastfeeding mothers as a remedy to morning sickness and increased appetite
    - Sold in raw form or processed form by combining with clay, sanc wood ash or salt
- Geophagia – deliberate or accidental ingestion of soil by humans
    - Practiced in countries of West Africa and South Asia

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# Components of Calabash chalk

- Very few studies on analysis of calabash chalk
- Available data indicates that calabash chalk contains heavy metals
    - Aluminum silicate hydroxide
        - Primarily aluminum due to presence of kaolin clay group
    - Lead
    - Arsenic
    - Chromium

# Aluminum as a phosphorous binder

- Aluminum containing phosphorous binders were historically used as treatment for hyperphosphatemia
- In the 1980s, due to aluminum related neurological and bone disease, led to avoidance of aluminum-containing phosphate binders
- Despite limited data, KDIGO and KDOQI guidelines both suggest avoidance of aluminum-containing binders

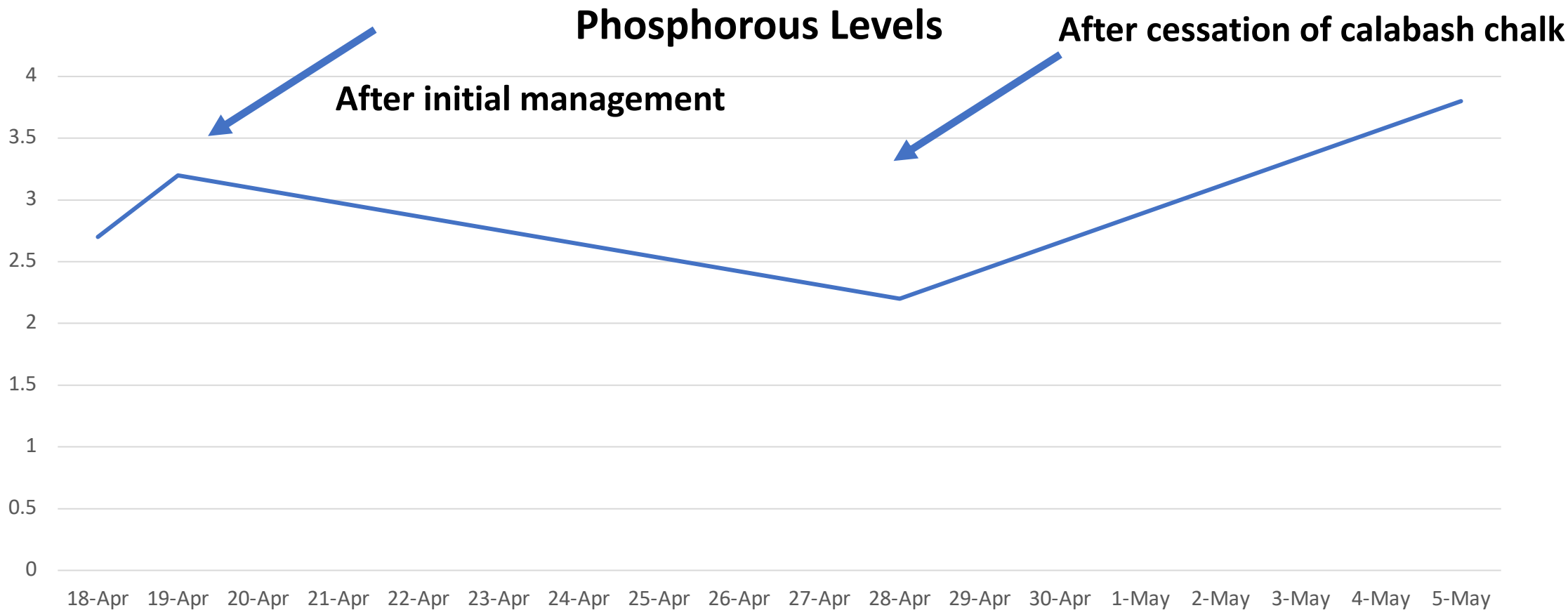
# Additional Laboratory Findings

| | Value | Normal Reference Values |
|---|---|---|
| ***Aluminum*** | 8 mcg/L | < 7 mcg/L |
| ***Arsenic*** | 10 mcg/L | < 5 mcg/L |
| ***Lead*** | 2.7 mcg/L | < 3.5 mcg/L |

# Case Continued

- Aluminum, arsenic and lead were in non-toxic levels
- Primary team asked patient and parents to discontinue any further consumption of calabash chalk
- Phosphorous levels were slowly repleted with oral supplementation and increased dietary phosphorous consumption
- After 1-month, oral supplementation of phosphorous was discontinued and the patient maintained normal levels of phosphorous on monthly labs

# Case Continued



# Key Points

- Aluminum and other cations such as lead, magnesium and iron are potent phosphorous binder
- Detailed supplement, nutritional and medication history can reveal pertinent etiologies for electrolyte derangements
- History taking is pertinent in elucidating certain cultural or ethnic practices that may be unfamiliar to providers

# References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