Positive Quality Intervention: Management of Hyperphosphatemia with a Low-Phosphorous Diet

Description: This PQI will focus on the management of high serum phosphorous levels (hyperphosphatemia) through dietary interventions.

Background: Maintaining inorganic phosphate (Pi) levels relies on a variety of homeostatic mechanisms. These include dietary intake and intestinal absorption, kidney excretion, bone deposition, and hormonal regulation. Normal serum phosphate levels range from 2.5-4.5 mg/dL. Hyperphosphatemia can result from tumor lysis syndrome, kidney disease, hypoparathyroidism, use of bisphosphonates, and certain oncolytic agents including panobinostat, monoclonal antibodies (brentuximab, obinutuzumab, otteruzumab, ibritumomab, and ofatumomab), TKIs, FGFR inhibitors, proteasome inhibitors, lenalidomide, and CAR-T cell therapy. This is an important condition to avoid as hyperphosphatemia can cause fatigue, shortness of breath, anorexia, nausea, vomiting, sleep disturbances, carpopedal spasm, hyperlexia, and seizures.

PQI Process: If a patient develops hyperphosphatemia that requires intervention according to institutional protocols during routine lab monitoring:

- Assess the patient for other lab abnormalities contributing to high phosphate levels
  - Vitamin D toxicity - excessive levels of 1,25-dihydroxyvitamin D can increase serum phosphate levels
  - Other electrolyte issues
    - Low magnesium levels (<1.7 mg/dL)
    - Low sodium levels (<135 mEq/L)
  - Renal function - impaired renal function can reduce tubular reabsorption and increase phosphate levels
  - Tumor lysis syndrome (TLS) - if one of the following lab abnormalities is also present, the patient should be immediately referred for evaluation for TLS
    - Uric Acid >8 mg/dL
    - Potassium >6 mEq/dL
    - Calcium <7 mg/dL

- Determine dietary intake of phosphorous and other possible contributing factors
  - Use of herals, supplements, and bisphosphonates
    - Common herbal supplements that can cause increased phosphate levels are: ginseng, bitter melon, coriander, flaxseed, horseradish, milk thistle, onion leaf, stinging nettle, and turmeric
  - Intestinal absorption of phosphate varies widely between dietary sources and contributes to phosphate burden in the diet
    - Inorganic phosphate, found mainly in preservatives and processed foods, has an oral bioavailability of ≥90%
    - Organic phosphate from animal sources has an oral bioavailability of 40-60%
    - Organic phosphate from plant-based sources has an oral bioavailability of 20-40%

- Monitor ongoing lab values to ensure phosphate levels return to within normal limits and refer patient to be seen by a provider if any of the following complications arise:
  - Severe constipation lasting 2 or more days despite laxative use
  - Nausea interfering with the ability to eat despite antiemetic use

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○ Vomiting or diarrhea occurring 4 or more times in a 24-hour period

**Patient-Centered Activities:** Provide patient-specific education on low-phosphorus diets

- Discuss the following dietary interventions with the patient and assess ongoing adherence
  - Increase fluid intake to 2-3 quarts of water daily
  - Decrease dietary intake of sodium to <2.3 grams per day
  - Adjust dietary phosphorous intake; make sure to consider the source of phosphorus and its corresponding bioavailability
  - Avoid processed foods and alcohol
  - Read labels and watch for sources of inorganic phosphate (phosphoric acid, pyrophosphates, polyphosphates, dicalcium phosphate, sodium phosphate)
- Provide referral to a registered dietitian
- Consider providing the patient with the table in Figure 1 to aid in reducing dietary phosphorus

**Figure 1:** Table summarizing sources of dietary phosphorous

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**References:**