Overview

Useful For
Assessing the body pool size of oxalate in patients with enzyme deficiencies, such as primary hyperoxaluria (PH) or patients with enteric hyperoxaluria (EH)

Aiding in the diagnosis of PH in a patient with chronic kidney disease (CKD) of indeterminate cause when urinary oxalate is not available

Monitoring patients with renal failure and primary or enteric hyperoxaluria in order to be sure they are receiving enough dialysis

Aiding in maintaining plasma oxalate levels below supersaturation (25-30 mcmol/L)

Testing Algorithm
See Hyperoxaluria Diagnostic Algorithm in Special Instructions.

Special Instructions

Method Name
Enzymatic

NY State Available
Yes

Specimen

Specimen Type
Plasma Na Heparin

Specimen Required
Any client who has never collected a specimen for this test should call 800-533-1710 or 507-266-5700 and ask for the Clinical Specialty Laboratory for more detailed instructions.

Patient Preparation:
1. Fasting (12 hours)
2. Patient should avoid taking vitamin C supplements for 24 hours prior to collection.

Specimen Type: Acidified plasma

Collection Container/Tube: Green top (sodium heparin)

Submission Container/Tube: Plastic vial

Specimen Volume: 5 mL
Collection Instructions:

1. Place specimen on wet ice immediately.
2. Centrifuge for 10 minutes at 3,500 rpm at 4 degrees C within 1 hour of collection.
3. Aliquot plasma into a plastic vial.
4. Adjust the pH of the plasma specimen to a pH of 2.3-2.7 with approximately 10 mcL concentrated (12M) hydrochloric acid (or 20 mcL of 6M HCl) per 1 mL plasma.

Additional Information: Nonacidified specimens can be accepted if the heparinized plasma is properly frozen. However, a disclaimer will be added in nonacidified plasma: Sample was received nonacidified and frozen. In nonacidified samples oxalate values may increase spontaneously.

Forms

If not ordering electronically, complete, print, and send a Renal Diagnostics Test Request (T830) with the specimen.

Specimen Minimum Volume

2 mL

Reject Due To

All specimens will be evaluated at Mayo Clinic Laboratories for test suitability.

Specimen Stability Information

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<tr>
<th>Specimen Type</th>
<th>Temperature</th>
<th>Time</th>
<th>Special Container</th>
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<tbody>
<tr>
<td>Plasma Na Heparin</td>
<td>Frozen</td>
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Clinical and Interpretive

Clinical Information

Oxalate is an insoluble dicarboxylic acid, which is an end product of liver metabolism of glyoxalate and glycerate. Humans lack an enzyme to degrade oxalate, and thus it must be eliminated by the kidney.

Oxalate is a strong anion and tends to precipitate with calcium, especially in the urinary tract.

Consequently, about 75% of all kidney stones contain calcium oxalate in some proportion. In renal failure oxalate is retained in the body, and it can precipitate in tissues causing tissue toxicity, a condition called oxalosis.

In the absence of disease, up to 90% of the body pool of oxalate is produced by hepatic metabolism and the other 10% is provided by oxalate contained in various foods. However, in the presence of gastrointestinal diseases that cause fat malabsorption, the percentage of oxalate absorbed from food can be much greater. The oxalate content of fruits and vegetables is quite variable, some being quite high and others virtually zero.

Oxalate is freely filtered by the glomerulus. A smaller amount is also secreted in the proximal tubule. If the glomerular filtration rate (GFR) is decreased, oxalate begins to be retained in the body. However, in persons without primary
hypoxaluria (PH) or enteric hyperoxaluria (EH), plasma levels do not exceed the normal range until the GFR decreases below 10-20 mL/min/1.73 m².

Plasma oxalate concentration is a reflection of the body pool size. When the pool increases, oxalate may precipitate in tissues and cause toxicity. Plasma oxalate pool size can be increased in various situations:

Increased production and accumulation results from an abnormality in at least 3 different enzymes:

Alanine glyoxalate transferase is necessary for the conversion of glycolate to alanine. A deficiency or intracellular mistargeting of this hepatic enzyme results in increased oxalate production (primary hyperoxaluria type 1).

Glycolate reductase/hydroxypyruvate reductase deficiency in the liver and elsewhere in the body results in increased glyceric acid formation, which leads to increased oxalate production (primary hyperoxaluria type 2).

A third type of PH was recently shown to be due to variants of HOGA1 that encodes the enzyme 4-hydroxy-2-oxalolglutarate aldolase that is found in hepatic mitochondria (primary hyperoxaluria type 3).

Increased oxalate load can be caused by increased absorption from the intestines after consuming large amounts of oxalate-rich foods such as rhubarb, spinach, or nuts.

Certain abnormalities of the gastrointestinal tract can cause fat malabsorption including short bowel syndromes, inflammatory bowel disease, gastric bypass for obesity, and pancreatic insufficiency. All of these gastrointestinal abnormalities result in increased oxalate absorption from the intestinal tract. This condition referred to as EH is due to saponification of calcium by fatty acids in the colon, which in turn frees up oxalate anions for absorption.

Decreased urinary oxalate excretion in chronic kidney disease (CKD) also causes oxalate retention in the body.

PH is typically diagnosed by measuring oxalate levels in urine. However, as kidney function decreases, the renal excretion of oxalate also decreases. In such situations, plasma oxalate levels are often be informative. Although plasma oxalate increases in CKD patients without PH, values are much higher in those CKD patients who do have PH or EH. Plasma oxalate is often used to monitor these patients during critical periods in and around kidney transplantation, dialysis, or liver transplantation.

Oxalate concentration in dialysate fluid is a reflection of the oxalate removed during dialysis.

Reference Values

< or =2.0 mc mol/L

Reference values have not been established for patients under 18 years old or greater than 87 years of age.

Interpretation

In patients with normal renal function, the presence of increased plasma oxalate concentration is good evidence for overproduction of oxalate (primary hyperoxaluria: PH).

In the presence of renal insufficiency, plasma oxalate levels can be markedly elevated in patients with PH or enteric hyperoxaluria (EH). Increased levels of plasma oxalate can be found in dialysis patients without EH or PH, but the degree of elevation is less.(1)
In patients with possible primary hyperoxaluria and renal insufficiency, the diagnosis often can be presumptively made by knowing the plasma level of oxalate. However, ancillary tests, such as the demonstration of oxalate crystals in tissues (other than the kidney) or increased glycolate in dialysate (for patients on dialysis) are frequently necessary to make an accurate diagnosis.

**Cautions**

Because increased production and decreased excretion rates of oxalate can increase the plasma oxalate concentration, the interpretation of any given plasma value must consider the patient's clinical setting.

Proper specimen processing and acidification are essential to obtain a quality result (see Specimen Required).

For external clients only, non-acidified specimens can be accepted if the heparinized plasma is promptly frozen. However, in nonacidified plasma specimens, plasma oxalate values near the reference range can increase up to 50% due to spontaneous oxalate generation.

Extremely high levels of ascorbic acid (vitamin C) in the blood interfere with testing. Due to this, patients should refrain from vitamin C supplements prior to collection.

**Supportive Data**

A difference in plasma oxalate results between the previous and current methods was observed; Overall the new results were 2.5% higher by Bland-Altman analysis (2.5%, y=0.7284x + 2.0093, R² = 0.9537). However, the new method detected significantly lower results for samples with a plasma oxalate concentration greater than 20 mcmol/L (Bland-Altman Bias =-25.6%).

**Clinical Reference**


**Performance**

**Method Description**

This is an enzymatic method based on the reduction of oxalate by oxalate oxidase. The reaction releases hydrogen peroxide, which in the presence of peroxidase reacts with a dye to give a colored end point that is measured using a BioTek EPOCH plate spectrophotometer at 590 nm. (Package insert: Trinity Biotech, Oxalate Kit, V. 07/2016)

**PDF Report**

No

**Day(s) Performed**

Monday through Friday

**Report Available**

3 to 7 days

**Specimen Retention Time**

2 weeks

**Performing Laboratory Location**
Fees and Codes

Fees
- Authorized users can sign in to Test Prices for detailed fee information.
- Clients without access to Test Prices can contact Customer Service 24 hours a day, seven days a week.
- Prospective clients should contact their Regional Manager. For assistance, contact Customer Service.

Test Classification
This test has been modified from the manufacturer’s instructions. Its performance characteristics were determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the US Food and Drug Administration.

CPT Code Information
83945

LOINC® Information

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