PARATHYROID HORMONE (PTH) TESTING
Policy Number: CMP - 047
Effective Date: January 21, 2017

INSTRUCTIONS FOR USE
This Medical Policy provides assistance in interpreting UnitedHealthcare benefit plans. When deciding
coverage, the enrollee specific document must be referenced. The terms of an enrollee's document (e.g.,
Certificate of Coverage (COC) or Summary Plan Description (SPD)) may differ greatly. In the event of a conflict,
the enrollee's specific benefit document supersedes this Medical Policy. All reviewers must first identify
enrollee eligibility, any federal or state regulatory requirements and the plan benefit coverage prior to use of
this Medical Policy. Other Policies and Coverage Determination Guidelines may apply. UnitedHealthcare
reserves the right, in its sole discretion, to modify its Policies and Guidelines as necessary. This Medical Policy
is provided for informational purposes. It does not constitute medical advice.

UnitedHealthcare may also use tools developed by third parties, such as the MCG™ Care Guidelines, to assist us
in administering health benefits. The MCG™ Care Guidelines are intended to be used in connection with the
independent professional medical judgment of a qualified health care provider and do not constitute the
practice of medicine or medical advice.

BACKGROUND
Parathyroid glands are little glands in the neck that secrete parathyroid hormone (PTH) which along with Vitamin
D are the main regulators of calcium and phosphorus homeostatis.1 The most important actions of PTH are (1)
rapid mobilization of calcium and phosphate from bone and the long-term acceleration of bone resorption, (2)
increasing renal tubular reabsorption of calcium, (3) increasing intestinal absorption of calcium (mediated by an
action on the metabolism of vitamin D), and (4) decreasing renal tubular reabsorption of phosphate.

The PTH is normally measured concomitantly with serum calcium levels. High calcium levels are often caused by
parathyroid disease.2 Abnormally elevated PTH values may indicate primary, secondary, or tertiary
hyperparathyroidism. Abnormally low PTH levels may result from hypoparathyroidism and from certain
malignant diseases such as squamous cell carcinoma of the lung, renal carcinoma, pancreatic carcinoma, or
ovarian carcinoma.

Primary hyperparathyroidism (PHPT) occurs in 1% of the population and is the most common cause of hypercalcemia in an outpatient population. Even patients with “mild” disease and moderate hypercalcemia can have lipoprotein abnormalities, hypertension, glucose intolerance, and increased morbidity and mortality from cardiovascular disease. Additionally, there is the risk of low bone mineral density and increased fractures.

Since parathyroid gland disease (hyperparathyroidism) was first described in 1925, the symptoms have become known as “moans, groans, stones, and bones...with psychic overtones.” Nearly all patients with parathyroid problems have symptoms including kidney stones, frequent headaches, fatigue, depression, high blood pressure and the inability to concentrate.

PTH testing is indicated as one of the first approaches in the evaluation of hypercalcemia. It is considered medically necessary for:

- Evaluation of patients with a combination of clinical signs and symptoms strongly suggesting hyperparathyroidism such as weakness, fatigue, bone pain, confusion, depression, nausea, vomiting, polyuria, etc., in which parathyroid disease is suspected;
- Evaluating patients with a combination of clinical signs and symptoms of hypoparathyroidism (e.g., Chvostek’s sign, Trousseau’s sign, dysphagia, tetany, increased deep tendon reflexes);
- Evaluating patients with previously diagnosed hyperparathyroidism or hypoparathyroidism;
- Evaluating patients with ectopic parathyroid hormone-producing neoplasms;
- Evaluating and monitoring therapy of secondary hyperparathyroidism in chronic renal disease and/or status post renal transplantation;
- Immediate follow-up of patients who have undergone thyroidectomy and/or parathyroidectomy;
- Evaluating patients with an abnormal total or ionized calcium level;
- Distinguishing nonparathyroid from parathyroid causes of hypercalcemia;
- Evaluation of patients with a magnesium deficiency;
- Evaluation of patients with insufficient or excessive Vitamin D; and
- Evaluating a patient with osteoporosis to rule out parathyroid hormone involvement.

**Diseases and conditions**

*Rickets and Osteomalacia*

Rickets and osteomalacia are diseases of growing bone that are usually caused by a deficiency or impaired metabolism of Vitamin D (predominant cause), magnesium, phosphorus, or calcium. When Vitamin D is deficient, hypocalcemia develops which then stimulates the secretion of PTH.

*Osteoporosis*

Osteoporosis is a term that describes the loss of calcium from bones. When one (or more) of the parathyroid glands are overactive (hyperparathyroidism), there is excess PTH secreted. The excess PTH has a direct effect on the bones and causes the calcium to leave the bones and go into the blood stream. The result of too much PTH is that bones lose their density and hardness and become osteoporotic and prone to fractures.
Calcium

High calcium levels are almost never normal. High blood calcium levels increase the risk of stroke, heart attack, and heart failure due to the aggressive buildup of calcium in the arteries as well increases the risk of osteoporosis. Normally, parathyroid glands monitor blood calcium levels and when these levels drop, the parathyroid glands produce PTH. As PTH controls calcium in the blood, it fluctuates inversely with calcium and high calcium levels are associated with low PTH. If blood calcium is high and PTH is high, then there is the possibility of a parathyroid gland tumor.

Magnesium

Endocrine function and magnesium homeostasis are linked. Hypoparathyroidism, hyperthyroidism and aldosteronism increase renal loss of magnesium. In acute hypomagnesemia, parathyroid hormone (PTH) is released, but in severe, chronic hypomagnesemia, release of PTH is decreased and contributes to the hypocalcemia of magnesium deficiency.

Celiac Disease and Other Gastric Disorders

Patients with celiac disease or other gastric disorders may have problems absorbing calcium from their diet. Therefore, the parathyroid glands can possibly enlarge and produce excessive PTH, which removes calcium from the bones, to restore blood calcium levels. Decreased bone mineral density has been reported in patients with celiac disease in association with secondary hyperparathyroidism. Thus, these patients may have very significant osteoporosis, high PTH levels, low normal calcium and high alkaline-phosphatase (shows increased bone destruction).

Kidney Disease

Many patients with severe kidney failure on dialysis will develop secondary hyperparathyroidism to some degree or another. In many circumstances, all four parathyroid glands become enlarged and overproduce PTH in response to the high phosphorus in the blood associated with renal failure.

POLICY

For the following CPT code(s) in Table 1, the patient should have a diagnosis (ICD-10-CM) code(s) listed in the attached files below.

Table 1. HCPCS Codes (Alphanumeric, CPT® AMA)

<table>
<thead>
<tr>
<th>HCPCS Code</th>
<th>Description</th>
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<tr>
<td>83970</td>
<td>Parathyroid hormone</td>
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Limitations

PTH measurements should always be interpreted in conjunction with total calcium or ionized calcium level. Ionized calcium may be helpful in addition to the total calcium when the total calcium level is equivocal. PTH levels done in the absence of a change in the patient’s clinical status or serum calcium may be denied.

Immunoradiometric (IRMA) or immunochemiluminescent (ICMA) assays for intact parathyroid hormone are the current tests of choice. Since displacement-type radioimmunoassays (RIAs) are not sufficiently sensitive to measure PTH levels in hypoparathyroidism, methods other than IRMA and ICMA will be considered not medically reasonable or necessary. Recent injection of radioisotope may interfere with immunoradiometric assays for intact parathyroid hormone (IRMA).

It is expected that PTH levels will be limited to 4 times per year; however, testing once per month may be warranted in patients with renal failure. Medically necessary testing may be done more frequently in the early post operative period of a parathyroidectomy to monitor the success of surgery.

Parathormone testing will be covered only one (1) per day, unless samples were obtained intra-operatively to monitor level of function during parathyroidectomy or thyroidectomy. Any claims for parathormone testing billed in excess of this guideline (regardless of assay) will be considered not medically necessary.

PTH testing in patients who have undergone thyroidectomy and/or parathyroidectomy is only covered for immediate post-operative follow-up. Long term follow-up would be performed by monitoring calcium. A record of a serum calcium level obtained simultaneously with the parathormone assay should be maintained in the beneficiary’s medical record.
REFERENCES


POLICY HISTORY/REVISION HISTORY

<table>
<thead>
<tr>
<th>Date</th>
<th>Action/Description</th>
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<tbody>
<tr>
<td>01/21/2017</td>
<td>Updated ICD10 codes as per CMS recommendations. Removed ICD9 code file.</td>
</tr>
<tr>
<td>10/01/2015</td>
<td>Removed ICD9 table. Embedded ICD9/ ICD10 PDF files.</td>
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