CONDITION

Osteitis fibrosa

Osteitis fibrosa is bone damage caused by prolonged excess parathyroid hormone.

WHAT IT IS
Bone weakening from prolonged parathyroid hormone excess

BODY SYSTEM
Bones and hormone glands

MAIN SYMPTOMS
Bone pain, fractures, muscle weakness, bone deformity

WHAT CAUSES IT
Prolonged parathyroid hormone excess

HOW IT IS DIAGNOSED
Blood tests and characteristic bone imaging

MAIN TREATMENT
Control of excess parathyroid hormone

CAN IT BE CURED?
Sometimes - underlying hormone excess is treatable

IS IT AN EMERGENCY?
Sometimes - confusion, collapse, or suspected fracture

LONG-TERM OUTLOOK
Bone strength often improves after hormone control

Overview

Osteitis fibrosa is a bone condition caused by persistently high levels of parathyroid hormone, or PTH. PTH helps control calcium and phosphate levels in your blood. It is made by four small parathyroid glands, usually located behind the thyroid gland in your neck.

When PTH stays very high, it signals the body to release too much calcium from bone. Normal bone is gradually broken down and may be replaced by weaker fibrous tissue. Some people develop hollow-looking or cyst-like areas known as brown tumours. These are not cancer. Their brown colour comes from bleeding and iron deposits within the affected tissue.

Severe osteitis fibrosa is sometimes called osteitis fibrosa cystica. It is now uncommon where overactive parathyroid glands are found and treated early. It can still occur with severe primary hyperparathyroidism or long-standing kidney-related hyperparathyroidism.

The condition matters because weakened bones are more likely to hurt, change shape, or fracture. Treating the cause can stop further damage and may allow substantial bone healing.

Symptoms

Early bone changes may cause no obvious symptoms. Osteitis fibrosa may be discovered during tests for high calcium, kidney problems, or hyperparathyroidism.

Possible symptoms include:

Symptoms can also come from the underlying hormone problem. High blood calcium may cause thirst, frequent urination, constipation, nausea, poor concentration, or low mood. Kidney-related hyperparathyroidism may occur alongside itching, muscle cramps, and other effects of chronic kidney disease.

These symptoms have many possible causes. Their presence does not mean that you have osteitis fibrosa.

- Ongoing pain or tenderness in the bones
- Muscle weakness, tiredness, or difficulty with physical activity
- Fractures after a minor fall or strain
- Changes in bone shape, including bending of weight-bearing bones
- Loss of height or back pain from weakened spinal bones
- A firm swelling where a brown tumour has formed

Causes and risk factors

Osteitis fibrosa results from prolonged, marked overactivity of PTH. The two main settings are primary and secondary hyperparathyroidism.

In primary hyperparathyroidism, one or more parathyroid glands make too much PTH without an appropriate signal from the body. A non-cancerous gland growth called an adenoma is a common cause. Enlargement of several glands is another cause. Parathyroid cancer is rare.

In secondary hyperparathyroidism, the glands are responding to another long-term problem. Chronic kidney disease is the most important cause linked with severe bone changes. Damaged kidneys cannot manage phosphate and active vitamin D normally. This lowers available calcium and repeatedly stimulates PTH production. The broader group of mineral and bone problems caused by kidney disease is called chronic kidney disease-mineral and bone

disorder, or CKD-MBD.

Less commonly, severe vitamin D deficiency or poor calcium absorption may lead to prolonged secondary hyperparathyroidism. If the parathyroid glands eventually become partly independent after years of stimulation, this is called tertiary hyperparathyroidism.

Factors that increase the chance of osteitis fibrosa include untreated or difficult-to-control hyperparathyroidism, advanced chronic kidney disease, delayed access to testing, and some inherited conditions that affect the parathyroid glands. The condition itself is not usually inherited, although an underlying tendency to develop hyperparathyroidism sometimes is.

Diagnosis

Diagnosis begins with blood tests. These usually measure PTH, calcium, phosphate, kidney function, vitamin D, and alkaline phosphatase. Alkaline phosphatase is an enzyme that may rise when bone is being broken down and rebuilt rapidly.

The pattern of results helps identify the cause. Primary hyperparathyroidism commonly involves a high calcium level with a raised or inappropriately normal PTH level. Kidney-related secondary hyperparathyroidism often has a different calcium and phosphate pattern.

Imaging can show the effects on bone. X-rays may reveal thinning of the hard outer bone, fine loss of bone beneath the covering of certain bones, fractures, or brown tumours. Changes in the fingers, skull, jaw, ribs, pelvis, and collarbones can be especially informative. A bone density scan can measure reduced bone strength, but it cannot confirm osteitis fibrosa by itself.

Ultrasound or other neck imaging may help locate an abnormal parathyroid gland before surgery. These scans do not establish the diagnosis on their own; blood tests establish that PTH is excessive.

A brown tumour can resemble a bone cancer or another bone lesion on a scan. If the diagnosis remains uncertain, a biopsy may be needed. This involves removing a small tissue sample for examination. Bone biopsy is otherwise used only in selected complex cases, particularly when different forms of kidney-related bone damage are difficult to distinguish.

Treatment and management

Treatment focuses on bringing PTH and mineral levels under control. The right approach is based on the cause, the severity of bone damage, kidney function, symptoms, and blood test results.

For primary hyperparathyroidism, surgery to remove the overactive parathyroid gland or glands is the main definitive treatment when appropriate. PTH can fall quickly after successful surgery. Medical monitoring or medicines that alter calcium and PTH levels may be considered when surgery is not suitable or is being delayed.

Kidney-related secondary hyperparathyroidism usually requires coordinated management of CKD-MBD. Treatment may include controlling phosphate intake and absorption, correcting vitamin D problems, using medicines that reduce PTH release, and optimising dialysis where relevant. Surgery may be considered when severe hyperparathyroidism does not respond adequately to medical treatment.

Bone pain may need pain relief and support with movement. A fracture may require a brace, cast, or surgery. Brown tumours often shrink after PTH is controlled, so they do not always need direct removal. Surgery on a lesion may be considered if it causes pressure, major deformity, repeated bleeding, or a continuing risk of fracture.

After treatment for severe hyperparathyroidism, healing bones may rapidly take calcium from the blood. This is known as hungry bone syndrome. Calcium can fall enough to cause tingling, cramps, or muscle spasms. Blood tests and supervised calcium or vitamin D replacement may be needed. You should not start high-dose supplements on your own because calcium and phosphate balance can already be abnormal.

Complications

Untreated osteitis fibrosa can lead to fractures, chronic pain, reduced mobility, and permanent changes in bone shape. Weak spinal bones can collapse and contribute to height loss or a curved posture.

A brown tumour may press on nearby nerves or organs, depending on its location. Jaw lesions can affect eating or dental alignment. Rarely, spinal involvement can put pressure on the spinal cord.

The underlying hyperparathyroidism may also cause complications outside the bones. These can include kidney stones in primary hyperparathyroidism and worsening mineral imbalance or blood vessel calcification in chronic kidney disease.

Prevention

Osteitis fibrosa cannot always be prevented because the underlying parathyroid or kidney condition may not be preventable. Inherited causes of hyperparathyroidism also cannot be

prevented.

Severe bone damage is less likely when hyperparathyroidism is recognised and controlled early. If you have chronic kidney disease or known parathyroid overactivity, keeping up with recommended blood tests and follow-up can identify rising PTH and mineral changes before major bone injury develops.

Do not take calcium, vitamin D, or phosphate-containing products specifically to prevent this condition unless they have been recommended after review of your blood results.

Living with it and what to expect

The course depends mainly on how much bone damage has occurred and whether the PTH excess can be controlled. Blood calcium and PTH may improve rapidly after effective treatment, but bones recover more slowly.

Over months, bone pain and muscle strength may improve. Bone density can continue to increase for several years. Brown tumours may gradually shrink. Follow-up commonly includes blood tests and, when useful, repeat bone density or other imaging.

Not every change fully reverses. An established deformity, collapsed spinal bone, or badly damaged joint may remain even after the hormone problem is corrected. Rehabilitation, physiotherapy, mobility aids, or orthopaedic care can help you stay active and independent.

Long-term monitoring remains important. Hyperparathyroidism can return after treatment, and people with chronic kidney disease may need continuing management because the kidney-related cause persists. Most daily activities can often be resumed as bone strength and symptoms improve, but recovery time is individual and fractures may require longer restrictions.

When to get medical help

Arrange routine medical review if you develop persistent bone pain, unexplained muscle weakness, loss of height, a new bony swelling, or a fracture after a minor injury. Review is also important if you have known hyperparathyroidism or chronic kidney disease and your symptoms are changing.

Seek urgent assessment for sudden severe bone pain, inability to use a limb, or a visible change in limb shape after an injury. These can indicate a fracture.

Get emergency help for:

These symptoms may reflect spinal pressure, severe calcium imbalance, or another serious problem requiring prompt assessment.

- New weakness or numbness in both legs
- Loss of bladder or bowel control with back pain
- Severe confusion, collapse, or difficulty staying awake
- Repeated vomiting with marked thirst, weakness, or dehydration
- Breathing difficulty caused by a rapidly enlarging neck or jaw swelling

Common questions

Is osteitis fibrosa a type of cancer?

No. Osteitis fibrosa is bone damage caused by excess PTH. Brown tumours are non-cancerous areas of bone breakdown, bleeding, and fibrous tissue. Because they can look like cancer on imaging, further scans or a biopsy may sometimes be needed.

Is osteitis fibrosa the same as osteoporosis?

No. Both can weaken bones, but they develop differently. Osteoporosis reduces bone strength without the characteristic severe PTH-driven changes of osteitis fibrosa. A person can have both conditions.

Can osteitis fibrosa be reversed?

It can improve substantially when the excess PTH is controlled. Pain, bone density, and brown tumours may improve over months or years. Long-standing deformity or damage from a previous fracture may not fully reverse.

Do brown tumours need to be removed?

Not always. Many shrink after treatment of hyperparathyroidism. Removal may be considered if a lesion threatens a fracture, presses on nearby structures, causes major functional problems, or does not improve after PTH control.

Can osteitis fibrosa come back after treatment?

It can return if hyperparathyroidism comes back or remains uncontrolled. Ongoing blood tests are particularly important after parathyroid treatment and for people with chronic kidney disease.

Can I exercise with osteitis fibrosa?

Activity may help maintain strength and mobility, but weakened bones can fracture under excessive strain. The safest type and intensity depend on your bone damage and any existing

fractures. A tailored rehabilitation or physiotherapy plan may be useful.

Sources

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