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IMPA NEWS

THE OFFICIAL NEWS LETTER OF THE INDEPENDENT MEDICAL PRACTITIONERS ASSOCIATION

PRESIDENTS MESSAGE

Dear Colleagues,

First let me wish all of you a very happy new year for 2019. This is my first communication to you as the president of IMPA and also the first communication through the IMPA news letter. As I mentioned in my acceptance speech on the day of election I will definitely carry on the work started by my predecessor. So for a start the monthly CPD activity is already planned for until April 2019. Then I have also planned 3 more activities for the year under my presidency.

1. A membership drive - I feel we need to get into our fold every physician working in the private sector full time and it appears that there are many like this whom we have missed.
2. Augmentation of the CPD activity - what we plan to do under this is that all the CPD activity conducted in the OPA auditorium will be recorded and published as podcasts in the IMPA website.
3. We also plan to contribute to the development of the private sector medical services

It is clear from our constitution that our vision is :

1. To safeguard and further the interests and status of our members as a body
2. To promote and encourage co-operation and loyalty among members in the best interests of the medical profession
3. To maintain professional and ethical standards of the medical profession, particularly relating to the science of medicine, surgery and medical clinical research

Please underline the words "status", "best interests of the medical profession" and "professional and ethical standards" in the above vision statement. As a premier Sri Lankan association of private practitioners in Sri Lanka it is our duty and responsibility to actively attempt to live by the ideals pronounced by our founding fathers in our constitution which is probably one of the oldest in the whole of Southeast Asia. To this end I as the president am going to take some bold steps in consultation with my current office bearers. These would be spelled in detail after a preliminary consultation and a meeting with the office bearers. This consultative meeting is scheduled to be held on 18th January at the IMPA office.

Please keep in mind about our membership drive and try and canvass and enroll those who are qualified to be members of our association.

This is YOUR association. Let us get together and KEEP IT GOING.

Dr Ananda Perera

Editor - Dr A L P de S Seneviratne

INDEPENDENT MEDICAL PRACTIONERS ASSOCIATION
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STEROID INDUCED OSTEOPOROSIS

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Overview

Osteoporosis is a common condition characterized by low Bone Mineral Density (BMD) and an increased risk of fragility fractures. It affects up to 30% of women and 12% of men at some point of their lives. Osteoporosis is frequently presented with low trauma fractures, often of the vertebrae, forearm or hip although any bone is liable to fracture in this condition. Peripheral fractures are detected easily but vertebral fractures can be missed very often. Their presentation is more insidious and the usual ways of presentation are, chronic back pain, height loss and kyphosis. Sometimes they present with back pain after lifting heavy weight. Height loss of more than 5 cm should raise the suspicion of vertebral fractures.

Several risk factors are associated with the development of osteoporosis. Two of the most important risk factors are, age and female gender while other common and potentially modifiable risk factors include long term corticosteroid therapy, chronic inflammatory conditions such as Rheumatoid Arthritis, Inflammatory Bowel Disease and chronic kidney disease, malabsorption and untreated premature menopause. Long term antidepressants, antiepileptics, long term Depot Medroxyprogesteron Acetate and Proton Pump Inhibitors are some of the other medication causing secondary osteoporosis.

Glucocorticoid induced Osteoporosis (GIOP)

Glucocorticoids are used in clinical practice for the management of many inflammatory conditions. But their use is not free of significant complications to the user. One of the major morbid complications are osteoporosis with resultant fractures and is associated with pain and significant disability.

Epidemiology of GIOP

Glucocorticoid induced osteoporosis is one of the leading causes of medication induced osteoporosis. The incidence of new fracture after one year of treatment with steroids may be as high as 17%, and can occur in 30% to 50% of chronic steroid users. A rapid decline in Bone Mineral Density (BMD) begins within 3 months of glucocorticoid use and peaks at 6 months, followed by a slower, steady loss with continued use. Fractures can occur within 3 months of initiation of treatment and even with low doses as 2.5 mg per day. This indicates there is no safe dose in steroid therapy in terms of bone safety. Even inhaled steroids can cause bone loss, if used for prolonged periods. There is evidence that in glucocorticoid induced osteoporosis fractures can occur at higher levels of BMD than in postmenopausal osteoporosis. Studied also indicate that the risk of vertebral and non-vertebral fractures occur in glucocorticoid induced osteoporosis is dose dependent. Furthermore, systemic administration and the prolonged duration of treatment significantly increase the relative risk of fractures.

Pathophysiology of GIOP

Glucocorticoids have a negative effect on BMD and bone structure. Glucocorticoids affect bones directly in two ways. They cause reduction in bone formation at the same time increasing bone resorption. Glucocorticoids inhibit recruitment

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and activity of osteoblasts, promote survival of osteoclasts, and enhance apoptosis of osteoblasts and osteocytes leading to reduced bone formation. Glucocorticoids seem to affect skeletal sites that are mostly composed of trabecular bone, but fractures can occur in cortical bone as well. On the other hand glucocorticoids inhibit intestinal calcium absorption and increase urinary calcium loss leading to secondary hyperparathyroidism which also increases bone resorption.

There is evidence indicating that glucocorticoids may decrease sex hormone levels in men and women, leading to enhanced bone resorption. When bone loss occurs, the metabolically more active trabecular bone is affected earlier and more severely by glucocorticoids than cortical bone itself.

Diagnosis of Osteoporosis

The diagnosis of osteoporosis can be confirmed by Dual Energy X- Ray Absorptiometry (DEXA) scan. It is performed in patients who have an increased risk of fracture on the basis of clinical risk factors. According to SIGN guidelines DEXA should be considered if the ten - year risk of major osteoporotic fracture is more than 10%. If the BMD T score values by DEXA at lumbar spine, femoral neck or total hip are at below -2.5 the diagnosis of osteoporosis is confirmed.

Vertebral fractures are generally taken as diagnostic of osteoporosis, even the spine BMD values are not in the osteoporotic range.

Other investigations indicated to detect an under line cause for the condition include, thyroid function tests, liver and renal function tests, serum calcium and phosphate levels, tests for celiac disease and in males serum testosterone levels.

Fracture risk assessment in a patient suspected to have osteoporosis

Fracture risk can be estimated in several ways but the most commonly used method is the FRAX calculator developed by the WHO, which can be used in all over the world. It operates over an age of 40 to 90 years using common clinical risk factors for osteoporosis. The tool is based on individual patient models according to risk factors as well as the BMD at the femoral neck. And it gives a 10 year probability of a fracture in an individual.

Prevention and treatment of steroid induced osteoporosis

General Measures

Preventive measures should be initiated as soon as the corticosteroid treatment begins as it has been shown in studies that bone loss occurs more rapidly in the first six months of treatment. Corticosteroid users must be encouraged to participate in a weight bearing exercise program and should be advised regarding the positive effect of daily exercises to enhance proximal muscle strengthening. They should maintain normal body weight, refrain from smoking or excess alcohol. Always the lowest effective dose of corticosteroid should be prescribed for the shortest possible period, when long term therapy is indicated.

Calcium and vitamin D

For effective bone protection Calcium and vitamin D prophylaxis should be started as soon as corticosteroid therapy is initiated. The recommended daily dose of Calcium in premenopausal women and men is 1000 mg while that is 1500mg in postmenopausal women. A daily dose of 400 to 800 IU of Vitamin D is advised.

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Bisphosphonates

Bisphosphonates are the first line treatment in patients with osteoporosis of any cause. The most widely used agent is alendronic acid, 70 mg once a week. Oral resindronat 35 mg once a week is a better alternative. This drug has to be taken empty stomach with a glass of water. The most common adverse effect with oral treatment is upper gastrointestinal symptoms. To prevent that patients are advised to stay up right at least for 3o minutes following oral administration Hiatus hernia, gastroesophagial reflux and Barrett's oesophagus are not contraindications to oral bisphosphonate therapy.

In steroid induced osteoporosis, as evidence clearly show, fractures occur at higher levels of BMD than in postmenopausal osteoporosis. Therefore treatment should be considered in patients with a BMD T score of < -1.5. Although it is useful to have a DEXA scan before starting treatment, it is not absolutely necessary in every case to initiate bone protective treatment. Especially in elderly patients who are above 65 years of age, as majority of them will have T scores below -1.5.

Hormone replacement therapy

Hormone replacement therapy has a beneficial bone protective effect in premature menopause in women. Similarly Testosterone treatment is considered in fracture prevention in hypogonadal men.

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Serum Calcium

Reference Range

Calcium concentration, both total and free, is characterized by a high physiological variation, depending on age, sex, physiological state (eg, pregnancy), and even season (owing to the seasonal variation of vitamin D, which is directly involved in the regulation of calcium concentration). Therefore, separate reference intervals have been established according to the age and sex of the individual being tested.

Total calcium reference ranges in males are as follows:

- Younger than 12 months: Not established
- Age 1-14 years: 9.6-10.6 mg/dL
- Age 15-16 years: 9.5-10.5 mg/dL
- Age 17-18 years: 9.5-10.4 mg/dL
- Age 19-21 years: 9.3-10.3 mg/dL
- Age 22 years and older: 8.9-10.1 mg/dL

Total calcium reference ranges in females are as follows:

- Younger than 12 months: Not established
- Age 1-11 years: 9.6-10.6 mg/dL
- Age 12-14 years: 9.5-10.4 mg/dL
- Age 15-18 years: 9.1-10.3 mg/dL
- Age 19 years and older: 8.9-10.1 mg/dL

Free (ionized) calcium reference ranges in males are as follows:

- Younger than 12 months: Not established
- 1-19 years: 5.1-5.9 mg/dL
- Age 20 years and older: 4.8-5.7 mg/dL

Free (ionized) calcium reference ranges in females are as follows:

- Younger than 12 months: Not established
- 1-17 years: 5.1-5.9 mg/dL
- Age 18 years and older: 4.8-5.7 mg/dL

If You're Worried About Losing Calcium

While a total calcium blood test is useful to discover how much calcium is in your blood, it doesn't show how much calcium you may be losing. So if you have osteoporosis or are concerned about your bone health, you'll find a 24-hour urine calcium test useful. It's normal to lose some calcium in your urine, but anything over 200 mg for women and 250 mg for men over 24 hours is considered high. This is called hypercalciuria.

Consuming too much calcium or vitamin D may cause elevated urinary calcium loss– but it's relatively rare (you'd have to be consuming a lot!) More commonly, high urinary calcium loss is caused by factors which increase serum calcium like an overactive parathyroid gland

Normal Calcium Levels and Osteoporosis

The results of a total calcium blood test are usually normal for people with osteoporosis. That means it's possible to have normal blood calcium levels and still be losing bone. However, other bone diseases may show elevated results. These bone diseases include:

- Bone metastases: When cancer cells spread to bones from their original site.

- Paget's disease: A bone remodeling disorder. People with Paget's disease form new bone in irregular places and have bone removed from places they still need it. Paget's disease can cause weak bones, irregular skeletal formation, and high calcium blood levels (especially if the sufferer is dehydrated or doesn't engage in weight-bearing activities often enough.)

Remember, a total calcium blood test isn't used to diagnose disease. Your blood calcium levels don't indicate how much calcium is in your bones, or whether you're losing calcium at an

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abnormal rate. But a total calcium blood test can give an indication of underlying problems - some of which can lead to osteoporosis if left unchecked.

To discover if you're losing calcium at a high enough rate to cause bone loss you'll need the 24-hour urine calcium test. You can refer back to the highlighted "If You're Worried About Losing Calcium" box towards the top of the page for more information.

Symptoms of Low Calcium Levels (Hypocalcemia)
If you recall, we mentioned earlier that you need calcium to maintain the normal function of your muscles and nerves. So the most common symptoms of hypocalcemia are neuromuscular issues like:

- Muscle twitching
- Muscle spasms
- Tingling and/or numbness in the hands, feet, and face
- Confusion
- Bone pain

What causes high calcium?

- Primary Hyperparathyroidism
- Secondary Hyperparathyroidism
- Cancer

Hypercalcemia is common in the advanced stages of the following cancers:

- Multiple myeloma
- Breast cancer
- Parathyroid cancer
- Lung cancer
- Kidney cancer
- Lymphoma
- Leukemia
- Bone metastases

Drugs - Thiazide diuretics

Common Causes of Low Calcium Blood Levels	Rare Causes of Low Calcium Blood Levels
Not getting enough vitamin D from exposure to the sun	Parathyroid hormone resistance
Not getting enough vitamin D from your diet	Vitamin D resistance
Hypoparathyroidism (underactive parathyroid glands)	Certain seizure medications. Plus certain medications used to treat gastroesophageal reflux disease, peptic ulcers, Helicobacter pylori, and some allergic actions. If you're worried that medication you're taking could cause low calcium blood levels, discuss your concerns with your healthcare provider.
Chronic kidney disease	A defective vitamin D receptor (VDR) gene– it provides instructions for making the vitamin D receptor protein, which allows your body to respond to vitamin D
Pancreatitis (inflamed pancreas)	
Hypomagnesaemia (not enough magnesium)	

Symptoms of High Calcium Levels (Hypercalcemia)

- Nausea and vomiting
- Loss of appetite
- Constipation
- Fatigue
- Muscle weakness
- Increased thirst
- Increased need to urinate

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