Conflicts of interest

The authors have no conflict of interest to declare.

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postmenopausal women with osteoporosis Treatment Of Peripheral Osteo-

Idiopathic Hypophosphatemic Osteomalacia

Osteomalacia hipofosfatémica idiopática

Dear Editor,

Osteomalacia is a rare metabolic bone disorder which can be
caused by various diseases associated with a deficiency of calcium,
phosphorus or an inhibition of the mineralization process.1 We
report the case of a 62-year patient presenting hypophosphatemic
osteomalacia diagnosed through laboratory data, with no clear eti-
ology, classified as idiopathic.

The patient exhibited bilateral hip fractures of a non tra-
matic origin and had received a prosthesis which had undergone
mobilization. He presented muscle and bone pain lasting 3 years
that limited movement. She reported no dentition or audiologi-
 cal changes, no prior medication or family history of kidney or bone
disease.

Laboratory analysis showed: normal CBC and venous blood
gases, renal and hepatic profiles were normal except for ele-
vated alkaline phosphatase (443 IU/L for n=136), normocalcemia
and hypophosphatemia 0.60 mmol/L (n=0.7–1.60) and elevated
iPTH (79.8 ng/L for n=67) with normal values of 1.25 OHd and
25(OH)2D. The 24-h urine had normal calcium and phosphate,
with no other useful information. Hydroxyprolinuria of 41.4 mg/L
(<17/24 h/m2).

Suspecting hypophosphatemic osteomalacia we obtained the
following test results in search of its etiology: normal serum
protein on electrophoresis with absence of Bences Jones protein
in the urine, normal tumor markers (carcinoembryonic antigen
CEA, 15.3, 19.9, 125, alphaglycoprotein); we studied the patient
for possible digestive malabsorption syndrome (endoscopic, X-ray
study, histological and celiac antibodies) but results were negative;
parathyroid study (neck ultrasound and sestamibi scintigraphy)
was normal as were chest X-rays and abdominal ultrasound; a
bone scan with TC® showed multiple uptake areas in the ribs, heel,
and periprosthetically in both hips and peripheral joints (Fig. 1).

Suspecting a possible oncogenic cause we performed an 11 in.
octreotide scintigraphy for the detection of somatostatin receptors
which resulted negative. Bone mineral density (BMD) of the lumbar
spine (L2–L4) was 0.596 g/m2 with a T score of −4.74. We did not
study the levels of fibroblast growth factor (FGF-23). We

We started treatment with calcitriol (0.75 mcg/24h) and sodium
phosphate (Phosphate-Sandoz®, 2 g/24h) achieving normalization of
blood phosphorus levels, iPTH and FA, with subsequent hyper-
phosphaturia in urine 24 h (2538 mg/L) which had not previously
been observed. After this parameter normalized we added stron-
tium ranelate treatment for the treatment of osteoporosis. The
patient’s symptoms gradually improved. Three years after the
diagnosis, after initiating treatment, the patient can walk, mineral
metabolism analytical controls are normal as well as the TC® bone
scintigraphy. The latest lumbar spine densitometry (L2–L4) shows
0.744 g/m2, with a T score of −2.93 and a −0.82 Z score.

The parameters studied suggest that this case corresponds to
osteomalacia characterized by insufficient mineralization of the
osteoid formation in new bone metabolism points. Different dis-
eseases can lead to osteomalacia through mechanisms that cause
hypocalcemia, hypophosphatemia or defects in the mineraliza-
tion process.1 In this case, the slight decrease in phosphonemia
suggests that it is hypophosphatemic osteomalacia. Interestingly,
phosphonemia was initially accompanied with normality of phos-
hphaturia probably due to a severe depletion of phosphate reserves,
responsible in turn of the evolved osteomalacia process with atra-
matic fractures and crippling myopathy.

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Fig. 2. (a) Bone scan showing displacement of the prosthesis at the femoral shaft level (arrow). (b) Same patient after 6 months of treatment with strontium ranelate.

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Fig. 1. Scintiscan Uptake in Multiple Ribs, Fractured Left Hip, Left Calcaneus and Peripheral Joints.

The case of the patient under study is remarkable because it was not associated with any of the causes described in hypophosphatemic osteomalacia\(^1\)–\(^4\): (i) no family history or other evidence of heredity (e.g. abnormal dentition), (ii) no deficit of phosphate in the diet, (iii) or an association with any drug, (iv) no abnormalities were found that made us suspect a parathyroid endocrine origin; (v) no digestive malabsorption disorders or (vi) kidney problems; (vii) finally, the normality of 11-in. octreotide scintigraphy, tumor markers, Bences Jones proteins in the urine, and abdominal ultrasonography Chest X-rays suggest an unlikely oncogenic source.

We could not, to date, find the mechanism that caused the disturbance, which require us to classify it as idiopathic and keep looking for new possible causes of this disease. Known cases of idiopathic hypophosphatemic osteomalacia are rare.\(^5\),\(^6\) However, treatment based on phosphate supplementation has been effective in stabilizing blood phosphorus levels, but leads to a mild phosphaturia, to be expected based on the contribution.

**Conflict of Interest**

The authors have no disclosures to make.

**References**


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**Agomelatine Adjunctive Therapy for Fibromyalgia**

**Uso de agomelatina como tratamiento coadyuvante en la fibromialgia**

**Dear Editor:**

Fibromyalgia is a chronic disease that can affect the patient from a biological, psychological and social point of view.\(^1\) It includes symptoms such as extreme fatigue, persistent pain, stiffness of muscles, tendons and soft tissue, trouble sleeping, memory disturbances, depression and anxiety, among others.\(^2\)

The antidepressant agomelatine is an agonist of MT1 and MT2 melatonergic receptors\(^3\) and antagonist of postsynaptic serotonin receptors (5-HT2C), with the ability to “resynchronize” the biological clock.\(^4\),\(^5\)

Based on empirical observation of several rheumatologists of San Cristóbal (Venezuela) on the apparent efficacy of agomelatine in fibromyalgia resistant to conventional treatment, we present a case of a 43 year old female patient, diagnosed with fibromyalgia 3 years prior, and treated with L-arginine base, pregabalin, amitriptyline, tryptophan, meloxicam and B vitamin complex. Since no therapy had proven effective pharmacotherapy we indicated agomelatine 25 mg at bedtime as adjuvant treatment and an amitriptyline substitute. The case was followed for 16 weeks and assessed with the following instruments: Fibromyalgia Impact Questionnaire (FIQ), LANSS pain scale, Pittsburgh sleep quality index and the widespread pain index.

Initially, the patient was impaired with physical appearance, sleeplessness, muscle aches and fatigue. In week 2 appreciate a clear improvement in the aforementioned parameters. At six weeks the improvement in sleep disturbance was clinically significant and sleep latency was reduced from 15 to 5 min and increased night-