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CASE REPORT

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Complete form of pachydermoperiostosis with good initial response to etoricoxib: A case report

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Key Clinical Message

Pachydermoperiostosis is a rare genetic disorder that closely resembles acromegaly. Diagnosis is usually based on distinct clinical and radiological features. Oral etoricoxib therapy showed a good initial response in our patient.

Abstract

Pachydermoperiostosis (PDP) is a rare genetic disorder with unclear etiopathogenesis. We report a case of a 38-year-old male who presented with classic features of PDP. Our patient showed a good initial response to etoricoxib therapy but the safety and efficacy over long-term use are yet to be determined in further studies.

KEYWORDS

clubbing, etoricoxib, osteoarthropathy, pachydermoperiostosis

1 | INTRODUCTION

Pachydermoperiostosis (PDP) also known as Touraine–Solente–Gole syndrome, the primary form of hypertrophic osteoarthropathy (HOA) is a rare genetic disorder that is characterized by clubbing of fingers or toes, pachyderma usually involving the face and scalp and swelling of periarticular tissue, and subperiosteal new bone formation (periostitis).[1] Additional manifestations include hyperhidrosis, acne, arthropathy, and acro-osteolysis of long bones.[2] PDP closely resembles pulmonary HOA, but it has not been demonstrated associating with various heart and lung diseases.[3] PDP is a familial disorder inherited as an autosomal dominant trait with incomplete penetrance and variable expression. Three forms of PDP have been described: complete form (clubbing, pachyderma, and periostitis), incomplete form (clubbing and periostitis without pachyderma), and forme fruste (pachyderma with minimal to absent skeletal changes).[4,5] Diagnosis of PDP is made clinically when at least two of the following features are present: positive family history, clubbing, hypertrophic skin changes, and bone pain/radiographic changes.[6] Manifestations of the disease usually occur in adolescence, progress for 2–3 decades, remain stable thereafter and are associated with significant morbidity as age advances. Adolescent males are predominantly affected, with male-to-female ratio of approximately 7:1.[7,8]

We report a case of an adult male who had visited many health centers but was diagnosed as acromegaly and presented to our center with non-resolving complaints of excessive sweating and progressive enlargement of hands

and feet with gradual coarsening of facial features. From detailed examination and investigations, a diagnosis of PDP was made.

2 | HISTORY

A 38-years-old male, presented to Endocrinology outpatient department (OPD) with the chief complaints of progressive enlargement of both hands and feet, excessive sweating, and prominent folds of skin on the forehead and scalp for 10 years. He also complained of bilateral knee pain which was insidious on onset, mild in severity, and relieved by over-the-counter medications. He had sought medical attention multiple times in the past with the same complaints. He consumes alcohol twice weekly and smokes 3–4 cigarettes per day for 22 years. There is no history of consanguinity or similar illness in the family members. He gave a history of excessive mucoid discharge from bilateral eyelids which appeared swollen. He also complained of profuse sweating (hyperhidrosis) requiring change of clothes multiple times every day. There was no history of fever, chest pain, rashes, fatigue, tremors, changes in voice, headache, visual complaints, or urinary symptoms, and no history of diabetes mellitus, thyroid disorders, or hypertension diagnosed in the patient.

On examination, the patient was well-built, alert, well oriented and hemodynamically stable. He had prominent and multiple skin folds on the forehead and on the scalp (Figure 1 left, and Figure 2). Enlargement of bilateral hands, feet, distal forearm, and swelling of the knee and ankles were also noted. There was grade IV clubbing of all the fingers and toes (Figure 3). Anthropometric measures were as follows: height 165 cm, weight 56 kg, BMI 20.57 kg/m^2, waist circumference of 74 cm, and arm circumference was only 24 cm, and symmetrically reduced muscle bulk was noted in bilateral arms and thighs which appeared small in contrast to the enlarged distal limbs (hand and feet).

Laboratory tests showed normal CBC, RFT, LFT, TFT, URIC ACID, CALCIUM, LIPID PROFILE, URINE R/M/E, ANA, RA FACTOR, AND ANTI CCP. A provisional diagnosis of PDP was made and investigations were done to rule out secondary causes of osteoarthropathy with CXR, ESR, and CRP. Prior to the visit to Endocrine OPD at our Center, he had been evaluated for acromegaly and MRI Sella had been done and reported normal. Insulin-like growth factor 1 (IGF-1) level was not done due to the absence of facial features of acromegaly as well as cost issues.

Radiographs of bilateral knee joints Anteroposterior and Lateral view (Figure 4) show diffuse cortical thickening involving bilateral distal femoral diaphysis and metaphysis with periosteal reaction more appreciated posteriorly, similar but milder changes in fibula and tibia also. Radiographs of the bilateral ankle and foot (Figure 5) show cortical thickening with fluffy periosteal reaction in the bilateral tibia, fibula, milder changes in the talus and navicular in dorsal and medial aspects. Radiographs of the hand (Figure 6) show cortical thickening and periosteal reaction in the dorsal radius and ulna, and cortical thickening in the middle and proximal phalanx of all fingers. The abovementioned radiographic features are consistent with PDP. Chest X-ray PA view shows mild irregularity in the undersurface of scapula noted bilaterally, more in right than the left. MRI of the brain showed no abnormality.

The patient was treated as a case of PDP on an OPD basis and Tab etoricoxib (A selective COX-2 inhibitor) 60 mg once daily was given for initial 3 months. He followed up at the end of the third month and there were marked changes seen in his forehead (Figure 1, right), soft tissue swelling had markedly subsided at both feet as compared to his initial visit, although he complained of nonresolving of hyperhidrosis and knee pain.

FIGURE 1 Coarse facial features, before (left) and after (right) 3 months of etoricoxib therapy.

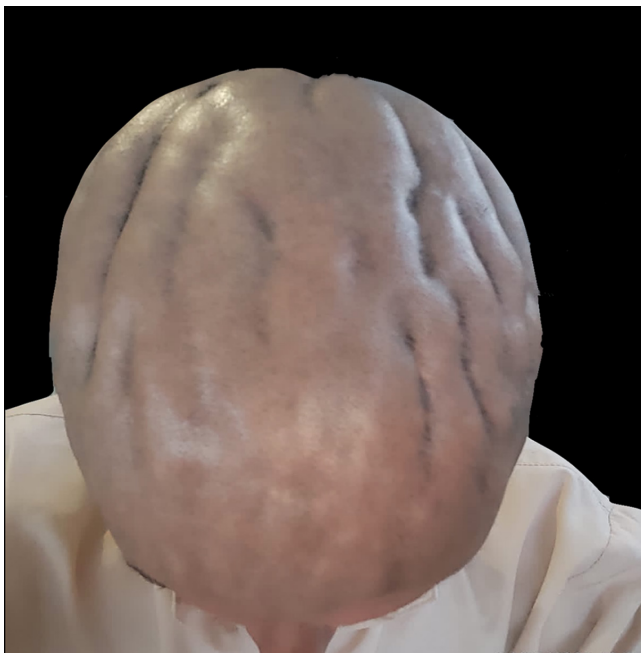

FIGURE 2 Cutis verticis gyrata.

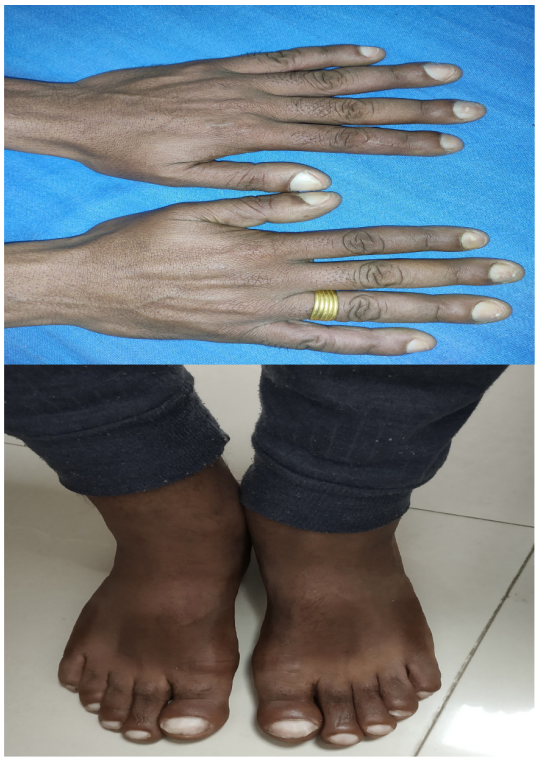

FIGURE 3 Clubbing of fingers and toes.

3 | DISCUSSION

HOA can be broadly divided into primary and secondary HOA. PDP, a rare genetic disorder is a primary HOA that has three forms: complete form, incomplete form, and forme fruste. HOA was described as "hyperostosis" by Friedreich, in 1868. In 1935, Touraine, Solente and Gole introduced PDP as a primary entity of HOA.[9] The precise incidence of this syndrome is unknown. According to one study, it has an estimated prevalence of 0.16%.[10] Men are affected more severely than women. Women often have milder findings, and their disease may remain undetected.[11] The onset of PDP is bimodal. The first peak of incidence is during the childhood of life and the second at adolescence progresses gradually over the next 5–20 years.[12]

The pathogenesis of PDP is not clearly understood. However, various studies have been carried out and have postulated theories regarding its pathogenesis. Mutations in the *SLCO2A1* gene and HPGD gene have been identified. *SLCO2A1* gene encodes prostaglandin transporter protein which is responsible for cellular uptake of prostaglandin E2 (PGE2). HPGD gene encodes 15-hydroxyprostaglandin dehydrogenase, which is a responsible enzyme for prostaglandin catabolism. So, mutation of both above-mentioned genes results in an increased level of PGE2 which is responsible for skin and skeletal changes in PDP.[13] PGE2 is responsible for both stimulation and inhibition of bone resorption by stimulating the cyclic AMP production.[14] There are two main receptors of PGE2 in bone cells that can increase cyclic AMP; namely EP2 and EP4 receptors.[15] The EP4 receptor is important in mediating the stimulation of bone resorption by increasing the activity of osteoclasts.[16] PGE2 acts on the EP2 receptor and stimulates bone formation by stimulation of osteoblast precursor cells.[17,18] Hence, PGE2 can stimulate the activity of both osteoblasts and osteoclasts, which is thought to be responsible for osteolysis and also periosteal bone formation resulting in typical acral and other skeletal features of PDP.[12] Also, PGE2 causes prolonged vasodilation which may explain the digital clubbing.[19,20] Novel *SLCO2A1* mutations have been described in a Lebanese family,[21] a Korean family,[19] and a Chinese family,[22] as having a novel nonsense mutation p.E141* of the *SLCO2A1* gene in a Japanese one[23] and a novel homozygous truncating mutation in *HPGD* gene has been described in Turkey.[24]

The diagnostic criteria for PDP are, Major criteria: Pachyderma, periostosis, finger clubbing, and Minor criteria: Hyperhidrosis, arthralgia, gastric ulcer, cutis verticis gyrata, blepharoptosis, joint effusion, column-like legs, edema, flushing, seborrhea.[8,25] Typical radiological features of PDP are symmetrical Subperiosteal new bone formation in the long bones usually radio-ulna, tibia and fibula (mostly involving the distal end of the bones) but not uncommon in metatarsals, metacarpals, and phalanges. These radiographic features present in our patient were similar to those reported by Sasane et al.[26] and Rastogi et al.[27]

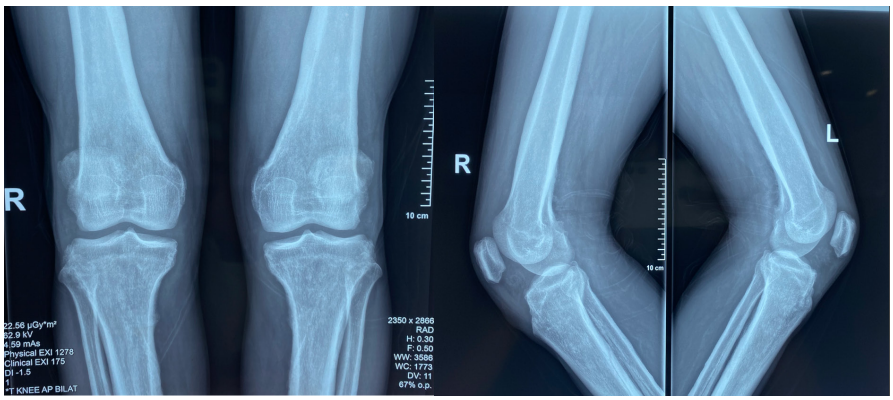

FIGURE 4 X-ray AP and Lateral view of knee joint showing diffuse cortical thickening involving bilateral distal femoral diaphysis and metaphysis with periosteal reaction.

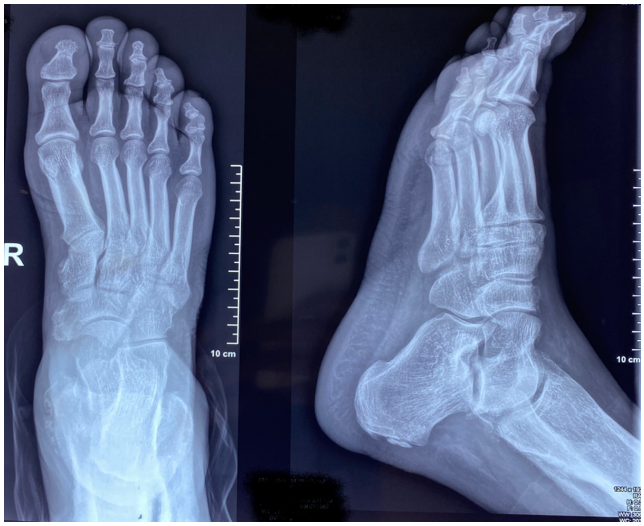

FIGURE 5 X-ray of ankle and foot showing cortical thickening with fluffy periosteal reaction in bilateral tibia, fibula, milder changes in talus and navicular in dorsal and medial aspect.

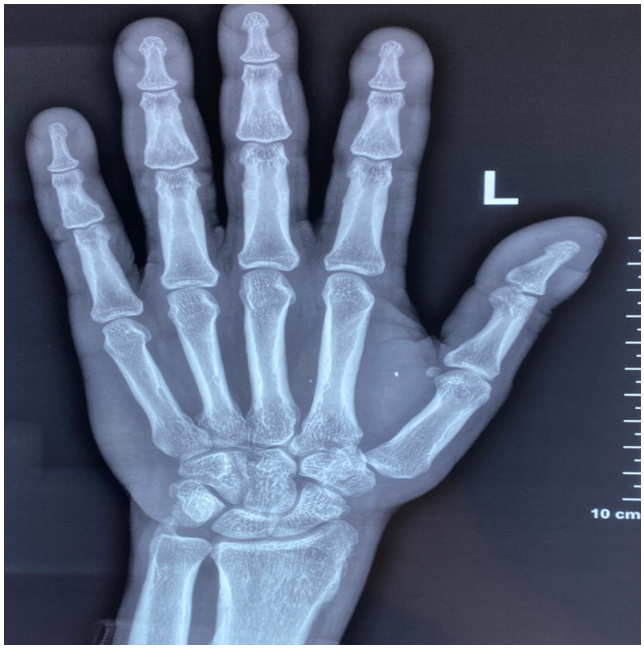

FIGURE 6 X-ray of hand showing cortical thickening and periosteal reaction in dorsal radius and ulna and middle and proximal phalanx of all fingers.

Our patient had all the three major criteria as well as most of the minor criteria like hyperhidrosis, arthralgia, cutis verticis gyrate, acne and seborrhea, rendering the case as a complete form of PDP which was supported by relevant blood investigation and the radiological features completely resembling the picture of PDP. Along with the classical picture of PDP, our patient also had ropy discharge from both eyes (symptoms began along with other PDP features and progressed with time) for which he used various antibiotic eye drops but that could not help him to get rid of the condition. He had prominent skin folds on the scalp resembling sulci and gyri of the brain, referred as cutis verticis gyrate and prominent folds on the skin of the forehead resembling "Leonine Facies" which is usually a late feature of the disease. In addition to these features, Honório MLP et al.[1] have also reported soft nodules with alopecia on their surfaces.

The clinical picture is very important in distinguishing primary and secondary HOA supported by the radiological findings. The secondary form is usually preceded by lung disorders (carcinoma of the lung, cystic fibrosis, or any other form of chronic infectious conditions of lungs), cardiac disorders, and gastrointestinal or hepatobiliary disorders like inflammatory bowel disease, carcinoma, and cirrhosis.[28,29] As none of any premorbid conditions was present in our patient and also the involvement of epiphysis was evident from X-rays (secondary form typically spares epiphysis),[30] secondary HOA can be ruled out.

PDP closely resembles acromegaly on the basis of clinical and radiological pictures but contrary to PDP, there is facial bone enlargement, jaw prognathism, nose enlargement, zygomatic-arch prominence, blow ridge, forehead protrusion, swelling of lips and tongue, in acromegaly. Elevation of IGF-1 level is diagnostic for acromegaly. We could not perform this test due to the inconvenience to the patient but he had an MRI head which supported ruling out acromegaly. Also, the absence of typical acro-facial

enlargement made acromegaly less likely in our patient. Less common entities like thyroid acropachy (a complication of autoimmune thyroid disease), Psoriatic onychopachydermo periostitis, hypervitaminosis A, and Caffey disease are the differentials that should be kept in mind during workup with the symptomatology as we have discussed.[8,29,31] But due to lack of clinical, laboratory, and radiological findings these differentials are less likely in our case.

The definitive treatment for PDP has not been well established. Treatment is mostly targeted at specific symptoms and is usually supportive. Nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, or corticosteroids are proved to be effective in alleviating arthralgia or arthritis-related symptoms.[32] Retinoids like isotretinoin improve cosmetic features of this disease by inducing apoptosis within sebaceous glands and also decreasing procollagen mRNA in fibroblasts.[33,34] Also, Colchicine gives a beneficial therapeutic response and improves skin manifestations by reducing chemotactic activity and tissue edema,[35] and injection of botulinum toxin type A (BTX-A) may provide temporary cosmetic improvement for leonine facies.[36]

It has been experienced that arthroscopic synovectomy help to reduce joint swelling in patients with marked joint effusion or recurrent arthritis symptoms,[37,38] but not much therapeutic benefit is experienced for arthralgia.[39] Bisphosphonates like pamidronate or risedronate have been reported effective for joint symptoms due to antiresorptive and osteoclast inhibitory properties.[37,40] Studies have reported the significant role of etoricoxib, a selective cyclooxygenase-2 inhibitor, in relieving or retarding pachydermia progression and also improvement of clubbing and joint swelling.[39,41] Two cases reported by Zhang et al.[39] has highlighted the efficacy of etoricoxib in improving coarse facial features and joint pain but the dose of etoricoxib was less (30 mg/day, combined with oral aescin) compared to our case. In our patient, etoricoxib 60 mg per oral once daily was started considering its safety and efficacy profile. Follow-up was done in 3 months. Eye symptom was completely resolved with a significant improvement of pachyderma in the face and scalp, however, hyperhidrosis and arthralgia still persist.

4 | CONCLUSION

PDP is a rare genetic condition with unclear pathogenesis. Our patient had a complete form of PDP, with typical radio-clinical features. The patient responded well to etoricoxib therapy with a significant improvement of the pachyderma in the face and scalp. Safety and efficacy over long-term use are yet to be determined in further studies.

AUTHOR CONTRIBUTIONS

Abinash Baniya: Conceptualization; resources; writing – original draft. **Ayam Bhattarai:** Resources; writing – review and editing. **Bibek Devkota:** Conceptualization; writing – original draft. **Saurav Khatiwada:** Conceptualization; writing – review and editing. **Pramod Kumar Kafle:** Writing – original draft. **Achyut Krishna Phuyal:** Writing – original draft. **Manoj Shahi:** Writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

We have no conflict of interest to disclose concerning this work.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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