Section: General Medicine

Case Report

A RARE CASE OF PRIMARY SJÖGREN'S SYNDROME PRESENTING AS OSTEOMALACIA

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ABSTRACT

The clinical presentation of primary sjogren's syndrome is classically tethered to the hallmark symptoms of xerophthalmia and xerostomia, driven by the autoimmune lymphocytic infiltration of the exocrine glands. It also has multiple extra glandular manifestations. But the progression of this pathology to severe, symptomatic metabolic bone disease as the primary presenting manifestation remains an exceptional clinical anomaly. This comprehensive report presents an exhaustive case analysis of a 32-year-old female from India, who presented with a trivial-trauma fracture of the left arm superimposed on profound, undiagnosed osteomalacia.

Keywords: Primary, Sjögren's Syndrome, Osteomalacia, Rare Case.

INTRODUCTION

Primary sjogren syndrome is a chronic, slowly progressive, systemic autoimmune disease characterized fundamentally by the lymphocytic infiltration of exocrine glands, predominantly the salivary and lacrimal glands, leading to the pathognomonic sicca syndrome of dry eyes and dry mouth. It was first described by Swedish ophthalmologist Henrik Sjögren in the early 20th century when the disease was long relegated to a benign, localised epithelitis. However, modern immunological profiling and histopathological mapping have repositioned primary sjogren syndrome as a complex, multi-system connective tissue disorder. While the exocrine architecture is the primary target, the pervasiveness of the autoimmune response frequently extends to extra glandular, non-exocrine visceral organs. This extra glandular involvement is estimated to occur in up to one-third of patients and is a primary determinant of the disease's overall morbidity and mortality.[5,6] Among the extra glandular sites the renal tubules are susceptible due to the expression of shared antigenic epitopes between the ductal epithelium of the exocrine glands and the renal tubular epithelial cells. Renal involvement in pSS commonly manifests as renal tubular acidosis and tubulointerstitial nephritis. The most common tubular defect encountered in primary sjogren syndrome is distal RTA (Type 1), wherein the alpha-intercalated cells of the cortical and medullary collecting ducts lose their capacity to secrete hydrogen ions into the tubular lumen. While the association between pSS and renal tubular acidosis is well-documented in the rheumatological literature,[7] the subsequent progression to severe, clinically overt metabolic bone disease—specifically osteomalacia—remains an exceptionally rare phenomenon.[2,3]

CASE REPORT

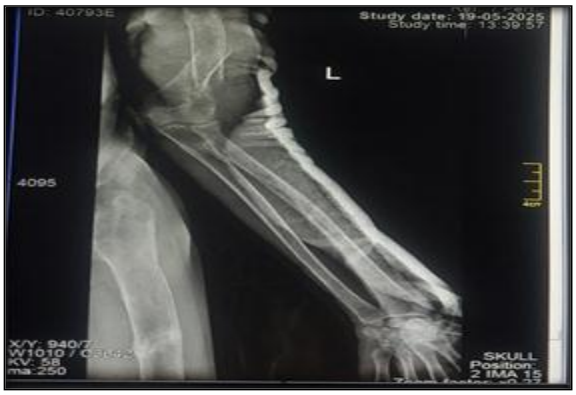

Image 1: X ray of her left arm – showing pathlological fractures.

A 32-year-old unmarried female, was brought to the orthopedic emergency department following a sudden, remarkably trivial fall sustained while performing routine activities at her residence one day prior. This low-impact trauma resulted in a pathological fracture of her left arm.

Past history - Her past medical history was otherwise notable only for a vaguely detailed prior course of empirical anti-tuberculosis therapy, the specifics of which were unavailable. She categorically denied any history of Type 2 diabetes mellitus, systemic hypertension, bronchial asthma, or epilepsy. Furthermore, she possessed no significant family history of autoimmune or renal disease, and her menstrual history was entirely unremarkable.

In search for the aetiology of the presentation a thorough and detailed clinical interrogation revealed that this acute fracture was merely the catastrophic culmination of a deeply overlooked seven-year clinical odyssey. The patient's longitudinal medical history was dominated by an unrelenting pattern of recurrent, fluctuating episodes of profound generalized weakness, extreme lethargy, and easy fatigability. Interestingly other x ray images taken at admission showed features suggestive of severe osteopenia.

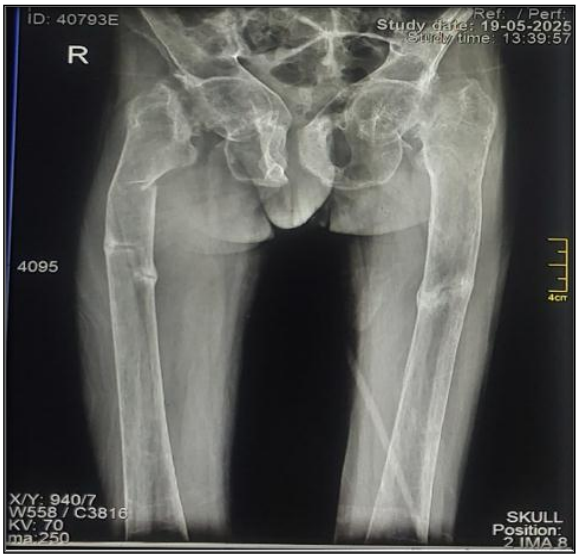

Image 2: X Ray Both Hips

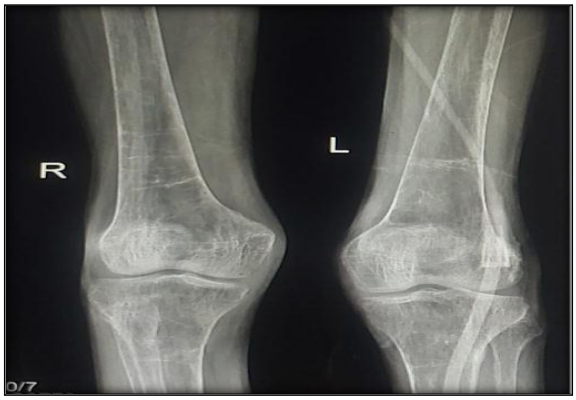

Image 3: X Ray Both Knees

On Examination - at the time of admission, the patient was conscious, oriented to time, place, and person, but in her advanced state of chronic illness. She exhibited a remarkably thin build, profound generalized muscle wasting. A detailed cutaneous and mucosal examination revealed an array of signs pointing toward a systemic connective tissue disorder: she had severe, diffusely dry skin (xeroderma), distinct hyperpigmentation localized over the malar prominences of her cheeks, and diffuse, non-scarring alopecia.

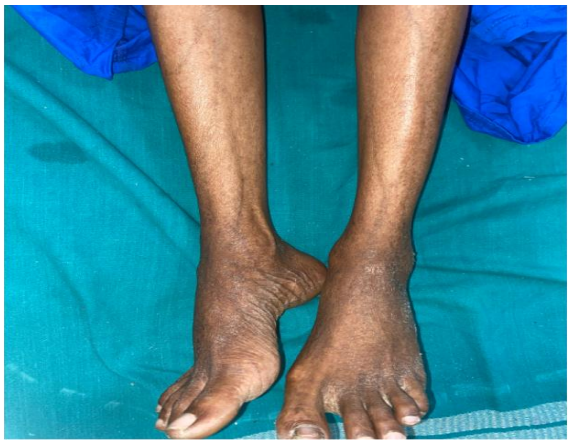

Image 4: Dry Skin

An inspection of her oral cavity revealed extremely poor oral hygiene punctuated by extensive, advanced dental caries.

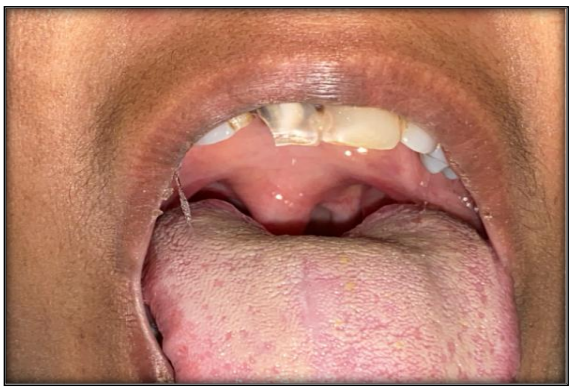

Image 5: Dental Caries of the Patient

Localized musculoskeletal findings were profound. She exhibited gross swelling, deformity, and exquisite tenderness over the left lower arm, anatomically corresponding to the acute fracture site. Additionally, she demonstrated marked, deep bony tenderness over the right upper thigh, a finding highly suspicious for the presence of pseudo fractures or Looser's zones within the pelvic girdle or proximal femur. Systemically, the patient exhibited significant conjunctival and mucosal pallor. There was no clinical evidence of icterus, cyanosis, digital clubbing, lymphadenopathy, or pedal edema. A thorough evaluation for opportunistic infections revealed no external stigmata or clinical markers of active tuberculosis.

Image6: Wasting at The Level of Hands

The chronicity, severity, and unpredictable nature of her undiagnosed neuromuscular and skeletal symptoms had inflicted a socioeconomic and psychological toll. Her inability to sustain basic physical exertion had forced her to transition through multiple employment roles and she was eventually compelled to restrict herself to a highly sedentary lifestyle. Thorough history did not reveal a specific clue towards the cause of her morbidity. All the basic investigations were surprisingly normal and all pathways directed towards the identification of etiology of the presentation had come to a dead end at one point of time. When asked initially patient described her past medication intake to be just nutritional supplements and syrups. Due to the doubt in the etiology she was told to bring all the medications that she used to consume (initially it was deferred as she was primarily admitted for fracture), which finally provided a diagnostic clue. Between acute hospital admissions, the patient had been relying on a prescription from a previous consultation at an outside hospital. Based on this old prescription,she independently consumed over-the-counter tablets and syrups, most notably potassium chloride syrup, whenever she felt the prodromal onset of profound weakness and impending paralysis. This self-directed, empirical therapy strongly suggested the possibility a chronic history of unrecognized, recurrent hypokalemic paralysis. For seven years, the patient endured a cycle of undiagnosed hypokalemic paralysis, (even though at present admission serum potassium of the patient was normal and the presenting complaint was fracture and not weakness) managed with symptomatic, self-administered potassium chloride supplementation.Through rigorous clinical evaluation, a complex metabolic derangement was uncovered, characterized by severe hypocalcemia, paradoxical intact parathyroid hormone (iPTH) levels, and renal phosphate and calcium wasting. These findings, coupled with a highly specific immunological profile featuring strong anti-SSA/Ro and anti-SSB/La positivityand a positive Schirmer test, established the diagnosis of primary Sjögren's syndrome complicated by combined distal and proximal renal tubular acidosis leading to acquired hypophosphatemic osteomalacia.

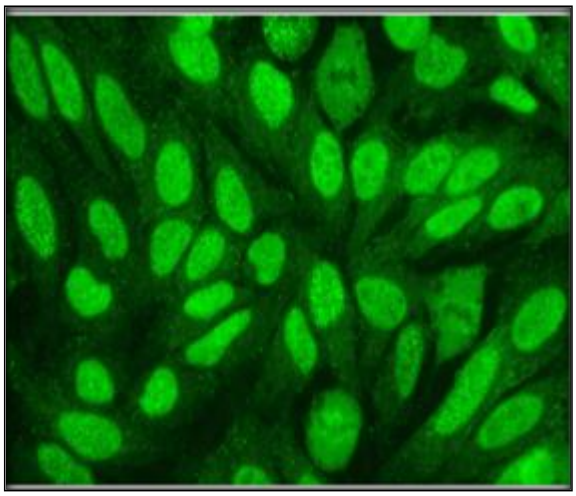

Image 7: Ana Analysis of This Showing Nuclear Speckled Pattern with 3+ Positivity in IIF Method

ENA ANALYSIS OF THE SAME SHOWED
3+ Positivity for all three
--- SSA / SSB / RO 52
Negative for all three
-- ds DNA, anti-smith and anti-histone antibodies.

DISCUSSION

This manifestation occurred without the patient spontaneously volunteering sicca symptoms, highlighting a dangerous diagnostic pitfall prevalent in the Indian clinical demographic.[1]

This paper provides an exhaustive examination of the betraying cues in primary sjogren syndrome induced metabolic bone disease, dissecting the pathophysiology of autoimmune renal tubular acidosis, the cellular mechanisms of skeletal buffering in chronic metabolic acidosis, and the-prognostic implications of osteomalacia when detached from its classic nutritional precipitants.[13,17]

Osteomalacia arises in this context not from a primary defect in bone formation, but as a direct, consequence of chronic metabolic acidosis and profound hypophosphatemia. The human skeleton serves as the body's ultimate reservoir of alkaline buffer. In the presence of a sustained systemic acid load that the kidneys can no longer excrete, the skeletal system is recruited to buffer the extracellular fluid, forcibly releasing calcium carbonate and calcium phosphate from the mineralized bone matrix.[8]

Concurrently, the acidic microenvironment within the bone tissue actively inhibits the synthetic function of osteoblasts while powerfully stimulating osteoclastic bone resorption. Furthermore, chronic acidosis impairs the renal tubular synthesis of 1-alpha-hydroxylase. The simultaneous proximal tubular leak of phosphate deprives the bone of the crucial elemental substrate required for the crystallization of calcium hydroxyapatite. The culmination of these metabolic assaults is the systemic accumulation of soft, unmineralized, and mechanically incompetent osteoid tissue, rendering the skeleton exceedingly fragile and susceptible to low-impact pathological fractures.[9,10]

Despite the theoretical inevitability of bone demineralization in prolonged, untreated RTA, osteomalacia functioning as the inaugural or primary presenting manifestation of pSS is vanishingly rare in global clinical practice. The diagnostic challenge in this specific demographic is massively compounded by two distinct epidemiological factors. First, there is a pervasive, almost universal baseline prevalence of nutritional Vitamin D deficiency[17] across the Indian population, which often provides a convenient, superficially plausible, but ultimately erroneous explanation for generalized skeletal pain. Second, clinical observations confirm that Indian patients with primary sjogren syndrome rarely volunteer sicca symptoms spontaneously.[1] The cultural and socioeconomic thresholds for seeking specialized medical care dictate that insidious, chronic symptoms like a dry mouth or gritty eyes are often dismissed as trivial age-related or environmental annoyances

Discussion of Differential Diagnosis

The laboratory evaluation presented a classic, devastating paradigm of severe metabolic bone disease completely uncoupled from typical regulatory endocrine feedback loops related to Calcium and Phosphatehomeostasis. The primary biochemical evaluation immediately revealed the sheer magnitude of the patient's skeletal and metabolic crisis.

Table 1: Calcium and Phosphate Homeostasis Evaluation

Parameter	Patient Value	Reference Range	Clinical Interpretation
Corrected Serum Calcium	7.2 mg/dL (Repeated 7.3 mg/dL)	8.5 – 10.5 mg/dL	Profound hypocalcemia, indicative of severe total body calcium depletion and failing skeletal reserves.
Serum Phosphorus	2.78 mg/dL (Initial 3.8 mg/dL)	2.5 – 4.6 mg/dL	Low-normal to low, representing systemic phosphate depletion.
Serum Vitamin D3 (25-OH)	18 ng/mL	> 50 ng/mL (optimal)	Deficient. Highly prevalent in the demographic but insufficient alone to cause this extreme degree of skeletal wasting and hypercalciuria.
Intact Parathyroid Hormone (iPTH)	31.4 pg/mL	18.8 – 88.0 pg/mL	Inappropriately Normal. The parathyroid glands are failing to mount a compensatory secondary hyperparathyroid response to severe hypocalcemia.
Serum Magnesium	1.74 mg/dL	1.6 – 2.6 mg/dL	Normal. Effectively excludes severe hypomagnesemia as the primary cause of impaired PTH secretion.
Serum Vitamin B12	224 pg/mL	211.0 – 946 pg/mL	Low-normal, ruling out profound pernicious anemia as a cause of neurological symptoms.

Two striking, confounding paradoxes existed within this initial endocrine profile. The first was the level of Vitamin D. The patient recorded a Serum Vitamin D3 level of 18 ng/mL. While formally classified as deficient, it is crucial in the diagnostic process to recognize that severe, fracture-inducing osteomalacia typically requires profound, near-total depletion of Vitamin D to manifest as an isolated, independent entity. In this specific case, the moderate deficiency acted purely as an aggravating, compounding factor rather than the primary driver of disease.The second, more profound metabolic paradox was the patient's Intact Parathyroid Hormone (iPTH) level. In the face of a critically

low, life-threatening corrected serum calcium of 7.2 mg/dL, a normal physiological feedback mechanism strictly dictates a massive, rapid surge in PTH secretion to mobilize calcium from bone reserves and increase renal tubular calcium reabsorption. Yet, the patient's iPTH was measured at a mere 31.4 pg/mL—an absolutely, inappropriately normal value. This failure of secondary hyperparathyroidism in the setting of prolonged, severe RTA is a complex phenomenon, hypothesized to be due to

1. Chronic intracellular magnesium depletion (though her current extracellular serum level was 1.74 mg/dL),
2. Direct suppressive effects of chronic severe acidosis on the parathyroid chief cells' calcium-sensing receptors, or
3. A complex interplay of systemic autoimmune inflammation blunting the endocrine axis.

Renal Excretion Indices and Tubular Function

To definitively separate a primary bone disease from a primary renal wasting syndrome, detailed analyses of 24-hour and spot urine collections were executed.

Table 2: Renal Excretion Indices and Tubular Function

Parameter	Patient Value	Normal / Target Value	Clinical Interpretation
Total Urine Volume (24h)	1500 mL	Varies	Normal output, ruling out anuric renal failure.
24-Hour Urinary Calcium	237 mg/day	< 4 mg/kg/day	For a severely wasted 35 kg patient, this represents a massive, unchecked renal calcium leak (>6.7 mg/kg/day).
Spot Calcium:Creatinine Ratio	0.39 mg/mg	< 0.20 mg/mg	Confirms severe hypercalciuria independent of total urine volume.
Urine Phosphorus	12.0 mg/dL	7 – 148 mg/dL	Must be interpreted dynamically via fractional excretion.
Urine Creatinine	16.0 mg/dL	16 – 327 mg/dL	Baseline for index calculation.
Serum Creatinine	1.02 mg/dL	0.5 – 1.1 mg/dL	Normal glomerular filtration rate (GFR), confirming the defect is probably tubular, not glomerular failure.
Fractional Excretion of Phosphorus	27.2 %	< 20 %	Diagnostic of Renal Phosphate Wasting. Indicates a proximal tubular defect in phosphate reabsorption.
Spot Albumin to Creatinine (ACR)	135 mg/g	< 30 mg/g	Microalbuminuria.

The most critical diagnostic calculation was the fractional excretion of phosphorus, calculated using the standard physiological formula:

Using the patient's values (dated 14/6/25), the calculation yielded:

This highly elevated result of 27.2% decisively and definitively localized a severe structural and functional defect to the proximal renal tubule, where the vast bulk of filtered phosphate is normally reabsorbed by sodium-phosphate cotransporters. Combined with the massive urinary calcium leak (237 mg/day in a severely underweight 35 kg patient) and the irrefutable clinical history of hypokalemic paralysis responsive to potassium chloride, the diagnostic picture solidified: the patient was suffering from a devastating, generalized autoimmune attack on her entire renal tubular system, encompassing both proximal and distal architectures.

Autoimmune and Systemic Profiling

With the renal tubular failure definitively mapped, the investigation turned to identifying the systemic etiology driving the destruction.

Table 3: Autoimmune and Systemic Profiling

Parameter	Patient Result	Clinical Interpretation
Extractable Nuclear Antigen (ENA) - SSA / Ro-52	3+ (Strongly Positive)	specific for Sjögren's Syndrome. Strongly associated with aggressive extraglandular and neurological manifestations.
Extractable Nuclear Antigen (ENA) - SSB / La	3+ (Strongly Positive)	Confirms Sjögren's syndrome as both antibodies are positive
dsDNA / Anti-Smith / Anti-Histone	All Negative	Effectively and definitively rules out Systemic Lupus Erythematosus and drug-induced lupus erythematosus.
Complement C3	96.40 mg/dL (Normal)	The alternative complement pathway is intact and unconsumed (Reference: 82.0 to 160.0).
Complement C4	8.80 mg/dL (Low)	Classical pathway consumption. Highly prevalent and prognostic in pSS with severe systemic/extraglandular involvement (Reference: 12.00 to 36.00).
Schirmer Test	Positive (+ve)	Objective, physical confirmation of profound xerophthalmia (lacrimal gland failure), validating the sicca symptoms
Hemoglobin / Protein Electrophoresis	Normal	Absolutely rules out hemoglobinopathies (HBF, HBA2, HB Adult all normal) and monoclonal gammopathies/myeloma.

The profound, overwhelming positivity for anti-SSA/Ro (3+) and anti-SSB/La (3+) autoantibodies, coupled with the objectively positive Schirmer test, provided the undeniable root etiology. The diagnosis was conclusively established as Primary Sjögren's Syndrome presenting as severe osteomalacia secondary to acquired distal and proximal Renal Tubular Acidosis.The evaluation of a young adult

presenting with a low-trauma fragility fracture, severe generalized physical wasting, and a documented history of self-managed episodic weakness is inherently complex. The clinical picture demands a rigorous, structured exclusion of primary nutritional deficiencies, autonomous endocrinological hyperplasias, occult malignancies, and complex systemic autoimmune etiologies.

Table 4: Differential Diagnosis and Pathophysiological Matrix

Differential Diagnosis	Supportive Cues	Why it was rejected	Pathophysiological Reality in Case
Nutritional Vitamin D Deficiency (Osteomalacia)	Severe skeletal fragility, multiple fractures, profound subjective weakness, history of poor general nutrition, and a measured low Serum Vitamin D3 level (18 ng/mL).	Profound phosphaturia (Fractional Excretion of Phosphorus at 27.2%) and massive hypercalciuria (237 mg/day) entirely contradict simple nutritional deficiency. Pure nutritional osteomalacia typically features profound hypocalciuria as the kidneys desperately attempt to retain filtered calcium.	The moderate Vitamin D deficiency (18 ng/mL) is merely a compounding environmental variable, exacerbated by the failure of the severely acidotic, inflamed kidneys to perform optimal 1-alpha-hydroxylation. It is not the primary mechanism of skeletal failure.
Primary Hyperparathyroidism	Severe hypophosphatemia, significant hypercalciuria, pathological bone fractures, and generalized skeletal pain.	Intact Parathyroid Hormone (iPTH) was paradoxically normal (31.4 pg/mL) in the face of severe hypocalcemia (7.2 mg/dL). Primary hyperparathyroidism strictly demands significantly elevated PTH driving hypercalcemia.	The normal iPTH despite severe hypocalcemia indicates a functional parathyroid response failure, while the serum calcium and phosphate are being obligatorily wasted through the damaged proximal and distal renal tubules.
Isolated Distal Renal Tubular Acidosis (Type 1 RTA)	Seven-year history of hypokalemic paralysis (responsive to potassium chloride), chronic metabolic acidosis, alkaline urine, and generalized skeletal demineralization.	The presence of massive renal phosphate wasting (Spot Calcium:Creatinine ratio 0.39, highly elevated Fractional Excretion of Phosphorus) indicates severe proximal tubule involvement, not just a distal intercalated cell defect.	The autoimmune tubulointerstitial nephritis has progressed extensively to involve both the proximal architecture (causing phosphaturia) and the distal architecture (causing acidosis and hypokalemia).
Multiple Myeloma / Monoclonal Gammopathy	Unexplained pathological bone fractures in an adult, generalized weakness, renal dysfunction, and profound hypocomplementemia (low C4).	Completely normal Serum Protein Electrophoresis and normal Hemoglobin Electrophoresis. Lack of hypercalcemia (the patient was profoundly hypocalcemic).	Bone destruction was driven by biochemical acid buffering and the accumulation of unmineralised osteoid (osteomalacia), rather than neoplastic plasmacyte proliferation and localized osteolytic lesions.
Systemic Lupus Erythematosus (SLE)	Alopecia, malar hyperpigmentation, severe chronic fatigue, low C4 complement levels, and generalized joint/bone pain.	Negative Anti-dsDNA and negative Anti-Smith antibodies. The predominant clinical finding was massive sicca complex markers and selective tubular damage, rather than classic SLE immune-complex glomerulonephritis.	Primary Sjögren's Syndrome frequently shares overlapping autoimmune cutaneous features with SLE.

CONCLUSION

While the general incidence of osteomalacia in primary sjogren syndrome patients who already have established renal tubular acidosis has been reported in historical cohorts to range between 25% and 45%, these statistics apply almost exclusively to patient populations already identified and monitored for advanced renal disease.[11,12] Conversely, when analyzing cases where osteomalacia was the primary, inaugural manifestation that eventually led to the delayed diagnosis of Sjögren's syndrome, the epidemiological numbers plummet drastically. In the Indian subcontinent, the diagnostic reality is sparse, creating a significant void in clinical awareness. As meticulously detailed by Khandelwal et al, metabolic bone disease acting as the initial presenting manifestation of pSS is exceedingly rare. Their seminal study identified only three such cases at a major tertiary center, noting that out of the roughly 50 global cases, only 6-9 similar cases of metabolic bone disease acting as the primary manifestation have been formally reported in India to date.[2] Furthermore, the evaluation of bone pain in the Indian population is heavily confounded by endemic, population-wide nutritional deficiencies. This baseline demographic reality was firmly established by Shivane et al,[17]which demonstrated that an overwhelming 70% of totally healthy young adults in India exhibit 25-hydroxyvitamin D3 levels below the 20 ng/mL threshold. Consequently, when a patient present with generalized bone pain and a mildly reduced Vitamin D level (such as our patient's 18 ng/mL), the default, almost automatic clinical reflex is to diagnose nutritional osteomalacia and prescribe high-dose cholecalciferol. However, as highlighted in studies specifically addressing sjogrens induced skeletal disease, this absolute reliance on Vitamin D levels is highly paradoxical and dangerous; in an analysis of pSS patients with proven osteomalacia, only 38.8% exhibited Vitamin D levels strictly below the 20 ng/mL cut-off.[11,12,13] This stark statistical disparity conclusively proves that the severe bone destruction in pSS is driven primarily by acidotic dissolution and massive renal phosphate wasting, rather than simple avitaminosis D. The therapeutic implications

of recognizing this pathophysiological distinction are massive and life-altering. The foundational treatment protocol was established decades ago by Richards et al,[18] demonstrating that the severe osteomalacia of renal tubular acidosis could be successfully and rapidly treated by the aggressive administration of sodium bicarbonate alone. By neutralizing the systemic acid load, the skeletal buffering response is immediately halted, allowing the bone to remineralize. Furthermore, the complexity of proximal involvement was illuminated by Yang et al,[16]confirming that generalized proximal tubular defects in Sjögren's syndrome can present exclusively and dangerously as acquired hypophosphatemic osteomalacia. In such complex cases, without meticulously correcting the underlying systemic acidosis with alkali therapy and aggressively replacing the wasted serum phosphate, massive pharmacological doses of Vitamin D and calcium will simply be flushed uselessly through the damaged kidneys, leaving the skeleton entirely vulnerable and ensuring the continued progression of the disease.

This comprehensive case report details a catastrophic, late-stage manifestation of an autoimmune disease that effectively hid behind the benign mask of chronic fatigue, hypokalemia, and trivial fragility fractures. Primary sjogrens syndrome must be aggressively uncoupled from the benign, localized perception of a simple "dry eye and dry mouth" disease; it is a pervasive, highly destructive systemic autoimmune condition capable of stealthily dismantling the entire renal and skeletal systems over years of subclinical progression. The fundamental clinical message derived from this exhaustive physiological and epidemiological analysis is clear: in any adult patient presenting with unexplained osteomalacia, recurrent hypokalemia, and signs of renal tubular acidosis, clinicians must universally suspect, investigate, and systematically rule out primary sjogrens syndrome, even in the absolute absence of volunteered sicca symptoms. Early recognition, specific serological profiling (ENA Ro/La and complement C3/C4 analysis), and aggressive, targeted alkaline and electrolyte replacement are paramount to halting the autoimmune destruction before permanent, irreversible skeletal and renal devastation occurs.

INVESTIGATION DETAILS OF THE PATIENT

Complete Blood Count

Test	20/05	26/05
TC	6500	7800
DC	59/29/13	60/31/9
RBC	3.12	3.24
Hb	9.3	9.5
PCV	30.1	31.2
MCV	96.5	99.6
Platelet	1.56	1.59

Renal Profile

Test	20/05	26/05	03/06
Urea	32.5	34.4	33.9
Creatinine	1.02	0.91	0.98
Sodium	138	139	136
Potassium	4.2	3.7	3.9
RBS	90	87	96

Liver Function Test (LFT)

LFT	20/5	26/5	30/5
Total	0.5	0.4	0.6
Ind bil	0.3	0.1	0.4
Dir bil	0.2	0.3	0.2
SGOT	39	43.8	34.8
SGPT	23	20.5	15.5
ALP	155	160	156
Prot	6.9	6.2	6.3
Alb	2.8	2.7	2.2
Glob	4.1	3.5	4.1

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