

REVIEWER'S REPORT

Manuscript No.: IJAR-59442

Title: Atraumatic Distal Tibial Fracture as the First Presentation of Sjögren Syndrome With Distal Renal Tubular Acidosis, Hypokalemic Paresis, and Nephrocalcinosis

Recommendation:

Accept as it is

Accept after minor revision.....

Accept after major revision

Do not accept (*Reasons below*)

Rating

Originality

Techn. Quality

Clarity

Significance

Excel.	Good	Fair	Poor
		☐	
		☐	
		☐	
	☐		

Reviewer's ID: JPR- 094

Detailed Reviewer's Report

Reviewer's Report

1. Strengths of the manuscript

1. Clinical relevance: The manuscript highlights the association between primary Sjögren syndrome, distal renal tubular acidosis (dRTA), hypokalemia, nephrocalcinosis, and possible metabolic bone disease.
2. Interesting clinical sequence: A fracture occurring approximately two months before the presentation with hypokalemic paresis provides a useful opportunity to discuss how skeletal manifestations may precede recognition of systemic autoimmune disease.
3. Multiple supporting clinical findings: The combination of severe normal-anion-gap metabolic acidosis, hypokalemia, urine pH of 7.5, a positive urinary anion gap, and bilateral nephrocalcinosis supports the clinical suspicion of dRTA.
4. Appropriate investigation for Sjögren syndrome: The history of ocular and oral dryness, markedly abnormal Schirmer testing, anti-Ro52 positivity, and reported minor salivary gland biopsy findings provide a reasonable basis for further autoimmune evaluation.
5. Relevant literature review: The authors compare their case with previously published reports of pathological fractures, osteomalacia, pseudofractures, and hypokalemic paralysis associated with Sjögren syndrome and renal tubular dysfunction.

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6. Acknowledgment of diagnostic limitations: The authors appropriately acknowledge that osteomalacia cannot be definitively established without additional skeletal investigations.

7. Practical educational message: The report encourages clinicians to consider renal tubular dysfunction and autoimmune disease in patients with unexplained fractures, hypokalemia, and metabolic acidosis.

2. Weaknesses and major concerns**A. Novelty is insufficiently established**

The manuscript describes an unusual distal tibial fracture, but pathological fracture associated with Sjögren syndrome and dRTA has already been reported, particularly by Abdulla et al. The authors should clarify exactly what distinguishes the present case from these earlier reports.

The novelty should not rest solely on the fact that the fracture involved the distal tibia. The authors should emphasize the complete clinical sequence and explain why it adds meaningful diagnostic or educational value beyond existing reports.

B. The diagnosis of osteomalacia or metabolic bone disease remains presumptive

The patient had elevated alkaline phosphatase (184 U/L), elevated parathyroid hormone (110 pg/mL), and vitamin D insufficiency (18 ng/mL). These findings suggest disturbed bone-mineral metabolism but do not independently establish osteomalacia.

The manuscript lacks documented bone mineral density, a detailed radiological assessment of fracture morphology, and confirmatory evidence of defective bone mineralization.

Recommendation: Consistently describe the fracture as potentially related to metabolic bone disease rather than attributing it definitively to osteomalacia. Clarify whether the fracture was classified radiologically as traumatic, stress-related, insufficiency-related, or pathological.

C. Sjögren syndrome diagnostic criteria need clarification

The manuscript reports negative conventional anti-SSA/Ro and anti-SSB/La testing despite strong anti-Ro52 positivity. Isolated anti-Ro52 positivity should not automatically be treated as equivalent to a positive anti-SSA/Ro result for classification purposes.

The authors should provide the minor salivary gland biopsy report, including the focus score if available, and clarify whether the diagnosis satisfies accepted classification criteria or represents a clinical diagnosis supported by the overall findings.

D. The renal tubular acidosis assessment requires greater precision

The reported urine anion gap is internally consistent with the stated spot urine electrolytes:

$$17+14-21=+10 \text{ mmol/L}$$

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Nevertheless, the interpretation of the urinary anion gap depends on clinical context, particularly urine sodium and volume status. Recent vomiting, diarrhea, and possible volume depletion may complicate interpretation.

The authors should discuss these limitations and explain how gastrointestinal bicarbonate loss was distinguished from renal acidification failure.

E. Treatment details are incomplete

The manuscript reports potassium chloride, potassium citrate, sodium bicarbonate, calcium supplementation, vitamin D, and prednisolone. However, it does not sufficiently explain the rationale for corticosteroid treatment or document the evidence of active autoimmune tubulointerstitial nephritis that prompted its use.

The authors should provide the indication for prednisolone, the planned duration or taper, and any relevant nephrology or rheumatology assessment. They should also clarify the monitoring and transition from intravenous to oral potassium replacement.

F. Follow-up is too short

One month of follow-up is insufficient to assess fracture healing, long-term correction of dRTA, recurrence of hypokalemia, renal complications, or recovery from possible metabolic bone disease.

The authors should report any subsequent fracture assessment, radiographic healing, repeat mineral profile, and longer-term renal and electrolyte outcomes if these data are available. If no further follow-up exists, this limitation should be stated explicitly.

G. Acid–base interpretation needs verification

The reported arterial blood gas values warrant rechecking. With bicarbonate of 9.2 mmol/L, the expected compensatory PaCO₂ using Winter's formula is approximately 22 ± 2 mmHg, whereas the reported PaCO₂ is 26 mmHg.

This discrepancy may indicate an additional respiratory acid–base disturbance or a mismatch between reported laboratory values. The authors should verify the original blood gas results and discuss the finding if confirmed.

H. Clinical documentation and presentation need improvement

- The fracture description should include the exact anatomical location, fracture pattern, displacement, and available imaging findings.
- The manuscript should explain whether bone density testing, serum magnesium, urinary calcium, or additional proximal tubular investigations were performed.
- The elevated urine albumin-to-creatinine ratio of 348 mg/g requires acknowledgment and, if available, repeat assessment or explanation.
- The relationship between the preceding gastrointestinal illness and the acute hypokalemic episode should be discussed.
- Numerous typographical errors, missing spaces, inconsistent abbreviations, and formatting problems require correction.

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3. Key points requiring revision

1. Establish the actual novelty

Compare the case directly with the previously reported pathological fracture associated with Sjögren syndrome and dRTA. Explain the incremental contribution of the distal tibial fracture and the subsequent clinical presentation.

2. Strengthen diagnostic evidence

Provide the salivary gland biopsy focus score, clarify the anti-Ro52 versus anti-SSA distinction, and verify the acid–base data.

3. Avoid overdiagnosis

Distinguish suspected metabolic bone disease from confirmed osteomalacia and avoid claiming a proven causal relationship between dRTA and the fracture without sufficient evidence.

4. Justify management

Explain corticosteroid use, document biochemical monitoring, and report fracture management and follow-up outcomes.

5. Improve reporting quality

Correct grammar and formatting, verify all references and citations, and ensure that the tables and figure legends are complete and consistent with the text.

4. Significance of the manuscript

The manuscript has moderate clinical and educational significance. It reinforces an important diagnostic principle: renal tubular acidosis associated with Sjögren syndrome may remain unrecognized until patients develop significant electrolyte disturbances, renal calcification, or skeletal complications.

Its strongest contribution is the reported sequence of a low-trauma distal tibial fracture followed by hypokalemic weakness, severe metabolic acidosis, and recognition of previously unrecognized Sjögren syndrome.

However, the significance is currently limited by the existence of similar published cases and the absence of definitive evidence establishing osteomalacia as the cause of the fracture. The paper is more suitable as an educational case report than as a claim of a wholly novel clinical entity.

5. Reviewer's recommendation

Major revision

Recommended editorial decision

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The manuscript addresses a clinically relevant presentation and may be suitable for publication after substantial revision. Acceptance should depend on clarification of the diagnostic evidence, verification of acid–base results, a more rigorous discussion of novelty, and correction of unsupported causal claims.

Suggested confidential comments to the editor

Writing

The manuscript presents a clinically relevant case of suspected primary Sjögren syndrome-associated distal renal tubular acidosis, with hypokalemic paresis, bilateral nephrocalcinosis, and a preceding low-trauma distal tibial fracture. The report has educational value because it highlights the possibility that skeletal manifestations may precede recognition of an underlying autoimmune disorder.

However, the novelty is limited by previously published cases of pathological fracture and metabolic bone disease associated with Sjögren syndrome and distal renal tubular acidosis. The diagnosis of osteomalacia is not confirmed, the salivary gland biopsy findings require more detailed documentation, and the reported blood gas values warrant verification because the PaCO₂ appears higher than expected for the degree of metabolic acidosis. The rationale for corticosteroid therapy and the short follow-up period also require clarification.

I recommend major revision rather than immediate acceptance or rejection. The manuscript may be considered for publication if the authors adequately address these concerns and moderate claims that are not supported by the available evidence.

Suggested comments to the authors

Writing

The case is potentially valuable because it illustrates the diagnostic importance of considering distal renal tubular acidosis and underlying Sjögren syndrome in patients presenting with hypokalemic weakness, nephrocalcinosis, and an unexplained fracture.

The manuscript requires major revision before it can be considered for publication. Please clarify the novelty compared with previously reported pathological fractures associated with Sjögren syndrome and distal renal tubular acidosis; provide the available salivary gland biopsy findings and focus score; verify the reported arterial blood gas results; and discuss the limitations of the urinary anion gap interpretation in the setting of recent gastrointestinal losses. Please also clarify the indication for corticosteroid treatment, provide further details of fracture assessment and follow-up, and avoid presenting osteomalacia as confirmed without supporting evidence.

Finally, the manuscript requires comprehensive language editing, correction of formatting errors, and verification of the references. Addressing these points would improve its scientific rigor, clinical usefulness, and suitability for publication.

Final verdict: Major revision. The case is potentially publishable, but its novelty is incremental, and important diagnostic and reporting issues must be resolved before acceptance.

Detailed justification for recommending Major Revision

The recommendation for Major Revision is based on specific scientific, diagnostic, clinical, and reporting deficiencies in the manuscript—not merely grammatical errors. The case has educational value, but

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several issues must be clarified before readers can reliably interpret the diagnosis, novelty, and clinical implications.

Below is a line-by-line reviewer assessment using the line numbers in your supplied manuscript. The comments are written in a format that can be adapted for a journal peer-review report.

1. Abstract: line-by-line comments

Line(s)	Reviewer's concern	Reason for revision
4–9	The background describes the relationship between dRTA, Sjögren syndrome, and bone disease, but the novelty claim is not sufficiently qualified.	Similar cases of pathological fractures and osteomalacia associated with Sjögren syndrome and dRTA have already been published. The authors must identify the specific contribution of this case.
10–12	The fracture is described as occurring while briskly walking.	The mechanism and radiological characteristics are insufficiently detailed to determine whether this was a fragility, insufficiency, stress, or other fracture.
13–16	Severe hypokalemia, metabolic acidosis, alkaline urine, and a positive urinary anion gap are reported.	These findings support dRTA, but the authors should clarify the acid–base interpretation and acknowledge the limitations of the urinary anion gap in the setting of recent vomiting, diarrhea, and possible volume depletion.
17–19	Sjögren syndrome is diagnosed using sicca symptoms, Schirmer testing, biopsy findings, and anti-Ro52 positivity.	The biopsy findings are not described quantitatively. Furthermore, isolated anti-Ro52 positivity is not automatically equivalent to a positive conventional anti-SSA/Ro result under accepted classification criteria.
20–23	Multiple treatments are listed, including prednisolone.	The indication for corticosteroids is unclear. The authors should explain the rationale, the suspected inflammatory renal involvement, and the treatment plan.
24–28	The conclusion suggests that an atraumatic fracture may indicate chronic metabolic bone disease associated with dRTA.	This is clinically plausible, but the manuscript does not establish the exact cause of the fracture or confirm osteomalacia. The conclusion should distinguish a diagnostic possibility from a demonstrated causal relationship.

Why this matters: The abstract is the reader's primary source of information. It must not overstate diagnostic certainty or novelty.

2. Introduction: lines 31–50

Line(s)	Reviewer's concern	Reason for revision
32–35	The introduction provides a general description of Sjögren syndrome and renal involvement.	The background is appropriate, but the authors should more clearly establish why this particular presentation deserves a separate case report.
36–40	The pathophysiology and manifestations of dRTA are described.	This information is established knowledge. The section could be shortened to make more room for the unresolved clinical question addressed by the case.
41–44	Chronic acidosis is linked to bone	The authors should distinguish reduced bone mineralization,

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disease and fractures.

osteomalacia, and fragility fractures rather than treating these outcomes as interchangeable.

45–48 The authors state that an atraumatic distal tibial fracture preceded the recognition of Sjögren syndrome.

This is the central novelty claim. It should be supported by a direct comparison with previously published fracture-associated cases, especially Abdulla et al.

49–50 The manuscript states that it was presented at a scientific meeting in July 2024.

The authors should clarify whether an abstract or conference proceeding was published and provide the relevant citation if applicable. Conference presentation alone does not establish prior journal publication.

Why this matters: The introduction should demonstrate a clear gap in the existing literature. The fact that a case is unusual does not automatically establish sufficient novelty for publication.

3. Case presentation: lines 51–81

Line(s)	Reviewer's concern	Reason for revision
52–60	The patient's clinical history is described, including vomiting, diarrhea, the previous fracture, and menopausal status.	The authors should clarify whether the gastrointestinal illness was considered a precipitating factor for hypokalemia and whether the fracture was evaluated for osteoporosis or another metabolic cause.
61–64	Neurological findings include generalized hypotonia, 4/5 power, and diminished reflexes.	The clinical description is useful, but the authors should report the distribution and severity of weakness more precisely if documented, including whether the weakness resolved after potassium correction.
65–68	Laboratory results show potassium 2.64 mmol/L, chloride 113.4 mmol/L, bicarbonate 9.2 mmol/L, and PaCO ₂ 26 mmHg.	The arterial blood gas values require verification. Winter's formula predicts a compensatory PaCO ₂ of approximately 22 ± 2 mmHg for bicarbonate of 9.2 mmol/L. The reported value of 26 mmHg is higher than expected and may indicate an additional respiratory component.
67–73	The authors diagnose dRTA using normal-anion-gap acidosis, alkaline urine, hypokalemia, and a positive urinary anion gap.	The diagnostic reasoning is plausible, but the authors should document the assessment of alternative causes of normal-anion-gap acidosis, especially gastrointestinal bicarbonate loss given the recent diarrhea.
68–71	Urine pH is 7.5, urine sodium 17 mmol/L, potassium 14 mmol/L, chloride 21 mmol/L, and the urinary anion gap is +10 mmol/L.	The urine sodium concentration is relatively low. Volume depletion and low distal sodium delivery can reduce the reliability of the urinary anion gap as a surrogate for ammonium excretion. This limitation should be discussed.
70–71	The urine albumin-to-creatinine ratio is 348 mg/g.	This is a substantial abnormality that deserves acknowledgment. The authors should state whether it was repeated, whether proteinuria was investigated further, and whether it was considered related to the renal disease. A single result does not establish chronic kidney disease.
74–76	Elevated alkaline phosphatase and PTH and low vitamin D are interpreted as evidence of bone-mineral disease.	These findings suggest disturbed mineral metabolism, but the authors should clarify whether alkaline phosphatase was bone-specific and whether other relevant investigations were performed. Vitamin D insufficiency and secondary hyperparathyroidism are potential contributors, not proof of osteomalacia.

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77–79	Ultrasound findings are interpreted as bilateral nephrocalcinosis.	The imaging description is supportive, but the authors should clarify whether the calcifications were confirmed as parenchymal nephrocalcinosis and whether stone disease or other renal abnormalities were assessed.
80–81	The distal tibial fracture is described as having malalignment.	More detailed fracture imaging and classification are needed to support the description as atraumatic or pathological. The authors should report the exact site, fracture pattern, and relevant radiological findings if available.

Why this matters: These are not minor editorial issues. They concern the reliability of the central renal diagnosis, the interpretation of the fracture, and the possible mechanism linking the two.

4. Sjögren syndrome evaluation: lines 82–93

Line(s)	Reviewer's concern	Reason for revision
83–84	The patient reports three years of dry eyes, dry mouth, and difficulty swallowing.	The symptoms are relevant, but the authors should clarify whether the history was assessed systematically and whether other causes of sicca symptoms were considered.
85–86	Schirmer test results are 2 mm and 4 mm over five minutes.	These results support ocular dryness. The authors should specify the testing method and report any additional objective ocular or salivary investigations if available.
87–89	ANA is positive at 1:100, anti-Ro52 is strongly positive, and conventional SSA/SSB tests are negative.	The serological interpretation needs precision. Isolated anti-Ro52 positivity does not necessarily meet the anti-SSA/Ro classification item. The authors should identify the assay used and avoid treating Ro52 positivity as definitive proof of Sjögren syndrome.
90	The minor labial salivary gland biopsy is described only as suggestive of Sjögren syndrome.	The actual histopathological findings and focus score are missing. This is an important omission because biopsy evidence may be particularly valuable when conventional anti-SSA testing is negative.
91–93	The findings are described as establishing the diagnosis of primary Sjögren syndrome with dRTA.	The authors should state whether the diagnosis is clinical or meets accepted classification criteria. They should also explain the evidence supporting the attribution of dRTA to Sjögren syndrome rather than merely documenting coexistence.

Why this matters: The autoimmune diagnosis is central to the paper's title. The diagnostic evidence must be transparent enough for readers to assess the conclusion independently.

5. Treatment and follow-up: lines 94–100

Line(s)	Reviewer's concern	Reason for revision
95–96	Intravenous potassium, oral potassium citrate, sodium bicarbonate, and calcium carbonate are prescribed.	The authors should provide the rationale for the replacement regimen, the timing of repeat electrolytes, and how the transition to oral treatment was managed.
96–97	Calcium supplementation is prescribed despite a normal initial serum calcium	Normal serum calcium does not exclude a need for supplementation, but the indication should be explained in the context of the patient's vitamin D status and suspected bone-

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	concentration.	mineral disorder.
97–99	Prednisolone 40 mg daily is started.	This is a major point requiring clarification. Corticosteroids are not routinely required for every case of dRTA associated with Sjögren syndrome. The authors should state whether active tubulointerstitial nephritis or another systemic inflammatory manifestation was suspected or documented and explain the indication, duration, and tapering plan.
98–100	Improvement is reported during hospitalization, with normal renal function and electrolytes at one month.	The outcome is encouraging, but the manuscript does not provide serial laboratory values, the duration of inpatient treatment, or sufficient long-term follow-up to assess relapse, renal outcomes, or fracture healing.

Why this matters: Treatment is a major component of a case report. Readers need to understand not only what was prescribed but also why it was prescribed and how its effectiveness was measured.

6. Figure and laboratory tables: lines 104–112 and the misplaced table entries

Line(s)	Reviewer's concern	Reason for revision
104–106	The figure legend describes three radiographic views.	The actual images should be assessed for image quality, anatomical labeling, fracture visibility, and whether the findings support the stated fracture classification. The legend alone is insufficient to evaluate these points.
107–109	Table 1 contains the initial laboratory investigations.	The table is useful, but the authors should ensure that all units, reference ranges where appropriate, abbreviations, and values are consistent with the main text.
110–112	Table 2 is introduced as the autoimmune serological workup.	In the supplied text, the table heading and its data are separated by intervening content. The final manuscript should present the table in the correct location and ensure that every result is clearly assigned to the corresponding test.
65–76 and Table 1	The serum chemistry and acid–base values are reported in several locations.	Internal consistency is essential. The authors should verify the bicarbonate, PaCO ₂ , anion gap, and other relevant results against the original laboratory reports.
Table 2 and lines 87–92	Conventional SSA/SSB negativity and strong Ro52 positivity are both reported.	The distinction should be maintained consistently in the table, text, abstract, and conclusion.

Why this matters: Poorly organized tables or ambiguous laboratory reporting can make an otherwise useful clinical case difficult to evaluate and reproduce.

7. Discussion: lines 114–190

This is the most important section for deciding whether the manuscript merits publication.

Line(s)	Reviewer's concern	Reason for revision
115–119	The authors describe renal involvement and metabolic bone disease in Sjögren syndrome.	The discussion repeats established background information. It should focus more directly on what the current case adds to the existing literature.

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120– 126	The sequence of fracture followed by hypokalemic paresis is presented as the key clinical feature.	This is potentially the strongest aspect of the case, but the authors must distinguish temporal sequence from proven causation. The fracture preceded recognition of dRTA; it has not been conclusively shown to have resulted from dRTA.
127– 133	Chronic acidosis, bone resorption, and osteomalacia are discussed.	The mechanism is plausible, but the patient-specific evidence for osteomalacia is incomplete. The authors should clearly separate established pathophysiology from findings demonstrated in this patient.
134– 140	The authors acknowledge that osteomalacia cannot be confirmed.	This is an appropriate limitation. However, the cautious interpretation must be maintained throughout the abstract, discussion, and conclusion, rather than making stronger causal claims elsewhere.
141– 147	Previously reported cases of metabolic bone disease and osteomalacia are reviewed.	The review is relevant, but the authors should identify the clinical similarities and differences between their case and the earlier reports.
148– 153	The case reported by Abdulla et al. is discussed as a particularly relevant comparator.	This is a critical comparison because that report already describes pathological fracture associated with Sjögren syndrome and dRTA. The authors need to explain precisely why the current case adds sufficient new information to justify publication.
154– 160	Additional reports involving pseudofractures, osteomalacia, and pediatric disease are summarized.	The literature review is broad but largely descriptive. A concise comparative summary of fracture type, renal presentation, autoimmune evidence, and distinguishing features would provide more scientific value.
161– 165	Recent reports of pseudofractures and hypokalemic weakness are cited.	These cases further demonstrate that the component manifestations are already known. The authors should use this evidence to refine—not overstate—the novelty claim.
166– 170	The authors state that the case demonstrates a distinctive sequence and multiorgan involvement despite negative conventional SSA/SSB results.	Multiorgan involvement and negative conventional SSA/SSB testing are not, by themselves, sufficient evidence of novelty. The contribution should be framed more narrowly and accurately.
171– 179	Hypokalemic paresis and the positive urinary anion gap are discussed.	The authors should integrate the low urine sodium concentration and recent gastrointestinal losses into the interpretation of the urinary anion gap. They should also address the blood gas compensation discrepancy identified earlier.
180– 183	Nephrocalcinosis is presented as evidence of chronic distal tubular dysfunction.	This is clinically reasonable, but the authors should avoid implying that nephrocalcinosis alone establishes the duration, cause, or severity of dRTA.
184– 190	The authors discuss sicca symptoms, Schirmer testing, serology, and biopsy.	The diagnostic discussion remains incomplete without the biopsy focus score, assay details, and a clear explanation of the distinction between clinical diagnosis and formal classification criteria.

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Why this matters: A case report should do more than describe a rare combination of findings. It should demonstrate how the case changes clinical understanding, improves diagnosis, or adds a distinct observation to previously published evidence.

8. Conclusion: lines 191–198

Line(s)	Reviewer's concern	Reason for revision
192–195	The conclusion describes atraumatic fracture as an uncommon presentation of dRTA preceding recognition of Sjögren syndrome.	The statement is broadly reasonable, but the authors should avoid implying that the fracture was definitively caused by dRTA when the skeletal pathology remains unconfirmed.
194–195	The combination of metabolic bone disease, hypokalemia, acidosis, alkaline urine, and nephrocalcinosis is emphasized.	The diagnostic message is useful, but the wording should distinguish suspected bone disease from confirmed osteomalacia.
196–198	Early correction of acidosis and treatment of bone-mineral abnormalities are recommended.	The recommendation is clinically sensible, but the authors should avoid implying that this single case establishes prevention of future complications. The conclusion should remain proportional to the evidence.

9. References: lines 199–227

Line(s)	Reviewer's concern	Reason for revision
200–206	References 1–3 address renal tubular acidosis and hypokalemic paralysis in Sjögren syndrome.	These references are relevant. The authors should ensure that each citation supports the exact claim made and that the reference formatting is consistent.
207–213	References 4–6 concern bone disease and osteomalacia associated with dRTA or Sjögren syndrome.	These references support the general background, but the authors should avoid using previous case reports as proof that the current patient's fracture was caused by osteomalacia.
214–215	Reference 7 describes pathological fracture in Sjögren syndrome due to dRTA.	This is the most important comparator for the novelty assessment. A more explicit comparison is required to establish the added value of the present report.
216–222	References 8–10 describe osteomalacia, hypophosphatemic bone disease, and severe metabolic bone manifestations.	The authors should explain which cases are most directly comparable and avoid giving equal emphasis to reports with substantially different clinical presentations.
223–227	References 11–12 describe more recent related presentations.	These references should be checked for accurate bibliographic details and appropriate interpretation. The discussion should explain how the present case differs from these publications.

Why this matters: The references themselves appear relevant to the clinical subject, but the literature review needs a stronger comparative analysis. The key concern is not simply the number of references; it is whether the authors have demonstrated the specific contribution of their case.

10. Which concerns actually justify Major Revision?

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Not every concern listed above independently justifies major revision. Typographical errors and table formatting are usually minor issues. The following five concerns are the principal reasons for the recommendation.

Diagnostic certainty

The diagnosis of Sjögren syndrome requires better documentation because conventional SSA/SSB results are negative and the biopsy findings are not quantified. The authors must clarify the basis for the diagnosis.

Acid–base validity

The reported PaCO₂ of 26 mmHg is higher than expected for the reported bicarbonate of 9.2 mmol/L. Verification may change the interpretation of the acute presentation.

Unproven fracture mechanism

The fracture is central to the title and novelty claim, but osteomalacia has not been confirmed and the exact fracture mechanism remains unclear.

Insufficient novelty analysis

Previously published reports already describe pathological fractures and bone disease associated with Sjögren syndrome and dRTA. The authors must demonstrate what this case adds.

Treatment and outcome documentation

The indication for prednisolone is unclear, and one-month follow-up provides limited information about long-term renal, biochemical, and skeletal outcomes.

11. Why not Minor Revision or Rejection?

Editorial decision	Assessment
Minor revision	Not appropriate at present because several unresolved issues affect diagnostic interpretation, the central causal claim, and the justification for novelty.
Major revision	Most appropriate because the case has clinical educational value, while the key concerns may be addressable through verification of existing data, clarification of methods and diagnoses, and substantial rewriting.
Reject	Could be appropriate if the journal requires a high degree of novelty and the authors cannot demonstrate a meaningful contribution beyond existing reports, or if essential diagnostic information cannot be verified.

A major revision recommendation does not guarantee acceptance. If the missing information cannot be recovered or the novelty remains insufficient after revision, rejection may ultimately be warranted.

12. Final reviewer recommendation

Writing

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Recommendation: Major Revision

The manuscript describes a clinically relevant case of suspected primary Sjögren syndrome-associated distal renal tubular acidosis presenting with hypokalemic paresis, nephrocalcinosis, and a preceding low-trauma distal tibial fracture. The report has educational value; however, substantial concerns must be addressed before publication can be considered.

The principal issues are incomplete documentation of the Sjögren syndrome diagnosis, the need to verify the reported acid–base values, insufficient evidence to establish osteomalacia as the cause of the fracture, and an inadequately demonstrated contribution beyond previously published cases. The rationale for corticosteroid treatment and the limited follow-up also require clarification.

I recommend major revision because these issues affect the interpretation and scientific contribution of the report rather than merely its presentation. The authors should verify the original laboratory data, provide additional diagnostic details where available, moderate unsupported causal claims, strengthen the comparison with existing literature, and revise the manuscript comprehensively. If the essential diagnostic evidence cannot be substantiated or the novelty remains insufficient, the manuscript may not be suitable for publication in its present form.