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Advance Medical and Clinical Research

Case Report

Alcohol-related hepatic osteodystrophy presenting as an atraumatic femoral neck fracture: a diagnostic case report and mechanistic framework

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Received: 03 January 2026
Accepted: 15 January 2026
Published: 09 February 2026

Abstract

Background: Hepatic osteodystrophy (HOD) is an underrecognized metabolic bone disease that accompanies chronic liver disease and increases fragility-fracture risk. We report a case in which heavy alcohol use and biochemical features of liver involvement created a diagnostic pathway culminating in HOD as the proximate cause of an atraumatic femoral neck fracture, and we summarize a mechanistic framework linking liver injury to skeletal fragility.

Case: An adult man with long-standing alcohol use presented with acute right hip pain without high-energy trauma. Radiographs demonstrated a displaced femoral neck fracture. Systemic workup ruled out malignant and infectious etiologies and suggested chronic liver disease. He underwent hip arthroplasty for pain control and early mobilization.

Discussion: Contemporary evidence indicates that liver-derived signals (e.g., IGFBP1–FGF21 axis; PP2Ac–LCAT pathway) modulate bone turnover and that vitamin D deficiency, hypogonadism, cytokine-mediated osteoclast activation, and malnutrition converge to decrease bone mass and quality in chronic liver disease. We integrate this liver–bone axis with clinical clues from this patient to explain the insufficiency fracture and outline a diagnostic algorithm emphasizing early identification of HOD.

Conclusion: In patients with alcohol use disorder or suspected liver disease presenting with atraumatic fractures, clinicians should actively consider HOD, evaluate reversible contributors (vitamin D/K deficiency, hypogonadism), and address liver disease in parallel with fracture care.

Keywords: Hepatic osteodystrophy; liver–bone axis; alcohol; hip fracture; IGF-1; IGFBP1; LCAT; vitamin D

Introduction

Hepatic osteodystrophy (HOD) encompasses osteoporosis and osteomalacia that occur in the setting of chronic liver disease (CLD). Its prevalence varies with etiology and disease stage, reaching 20–75% across cohorts, and it confers a substantially increased risk of hip fracture. Although reduced physical reserve and falls contribute, mounting evidence supports intrinsic liver–bone crosstalk mediated by hepatokines and altered vitamin D and sex-steroid homeostasis, which together deteriorate bone microarchitecture and quality. Despite these risks, HOD remains underdiagnosed in orthopaedic pathways that focus on surgical repair rather than systemic contributors. We present a diagnostically focused case and a mechanistic schema [1–3].

Bone remodeling is a complex adaptive program that integrates endocrine, nutritional, inflammatory, and mechanical inputs over time. Accordingly, fracture risk in chronic liver disease cannot be explained by any single deficit (e.g., vitamin D) and may remain poorly predicted by areal BMD alone. We therefore frame the liver-bone axis as a coupled control system: the liver acts as an integrator that emits endocrine/hepatokine calibration signals, and the skeletal multicellular unit continuously updates its remodeling set-point in response. In this view, alcohol-related liver injury can reduce the amplitude and coherence of these calibration signals, shifting remodeling toward a resorption-dominant equilibrium (a 'fragility attractor') [1–6].

Case presentation

An adult male with a history of chronic heavy alcohol consumption presented with sudden right groin pain after a trivial twist while walking; there was no high-energy trauma. Examination showed limb shortening and external rotation without neurovascular deficit. Pelvic anteroposterior and lateral radiographs demonstrated a displaced intracapsular femoral neck fracture (Figure 1A–B). Initial laboratory testing showed mild transaminase elevation and cholestatic indices compatible with chronic liver involvement; inflammatory markers were not suggestive of infection. MRI of the lumbar spine, obtained because of concomitant radicular symptoms, revealed multilevel degenerative changes without compressive lesions (Figure 2A). Whole-body PET/CT excluded focal malignancy; no hypermetabolic primary or osseous lesions were identified (Figure 2B). Given the acute hip pain with minimal trauma, background of heavy alcohol use, and biochemical evidence of liver involvement, HOD was suspected as the underlying mechanism of skeletal fragility. The patient underwent arthroplasty for pain control and early mobilization, with an uneventful recovery (Figure 4A). Gross inspection of the excised femo-

ral head showed trabecular thinning and subchondral collapse consistent with fragility (Figure 3A–B).

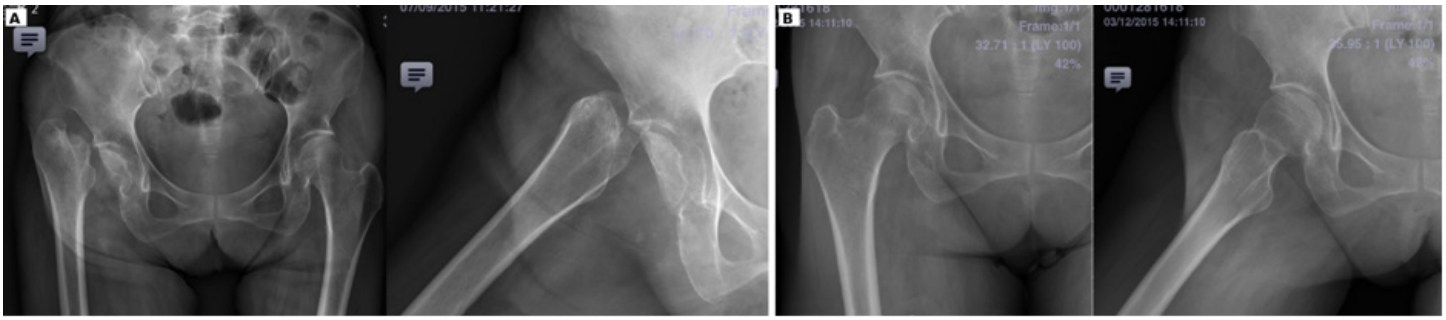

Figure 1. Diagnostic radiographs. (A) Pelvis AP demonstrating a displaced right femoral neck fracture; (B) cross-table lateral confirming intracapsular orientation.

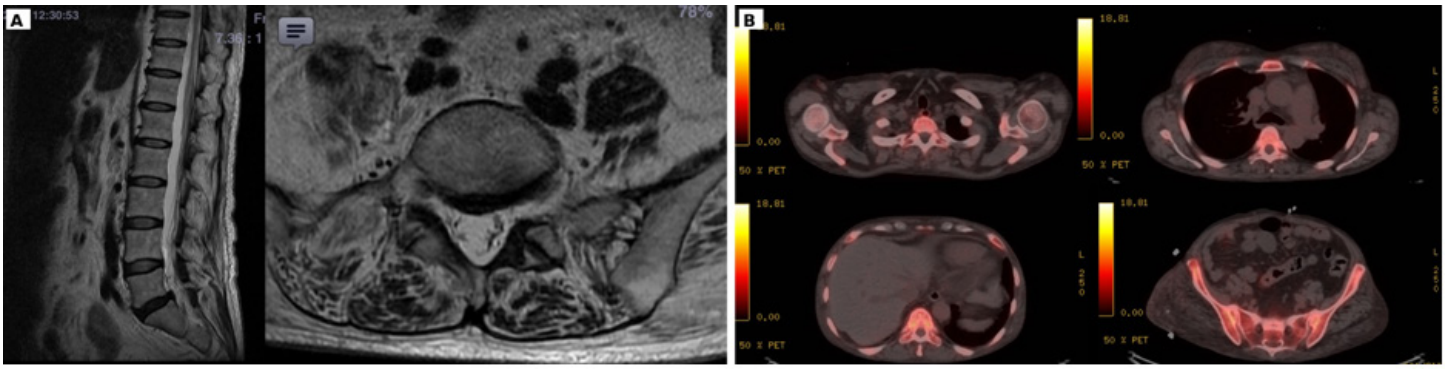

Figure 2. Systemic imaging used during the diagnostic work-up. (A) Lumbar spine MRI showing multilevel degenerative changes without compressive lesions; (B) whole-body PET/CT without focal hypermetabolic primary or osseous lesions, helping to exclude oncologic causes of a pathologic fracture.

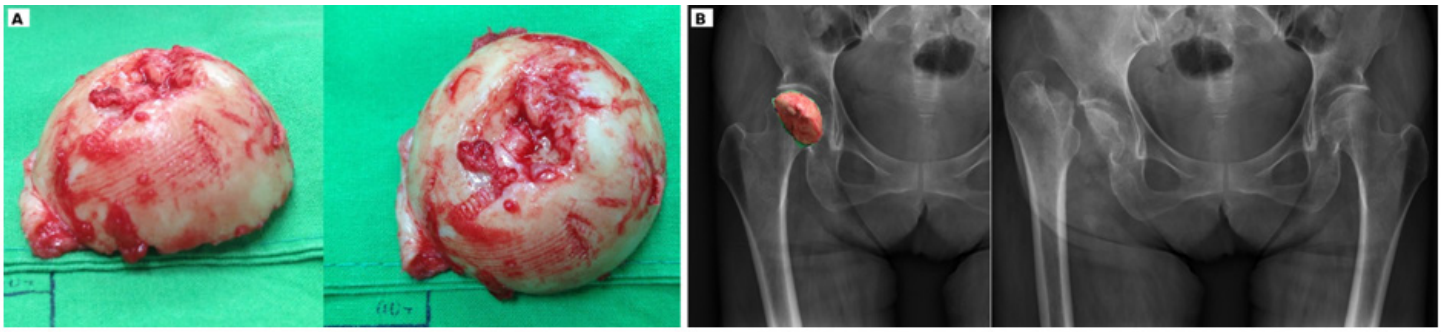

Figure 3. Gross pathology of the excised femoral head. (A–B) Two views reveal trabecular thinning and subchondral collapse consistent with fragility in metabolic bone disease.

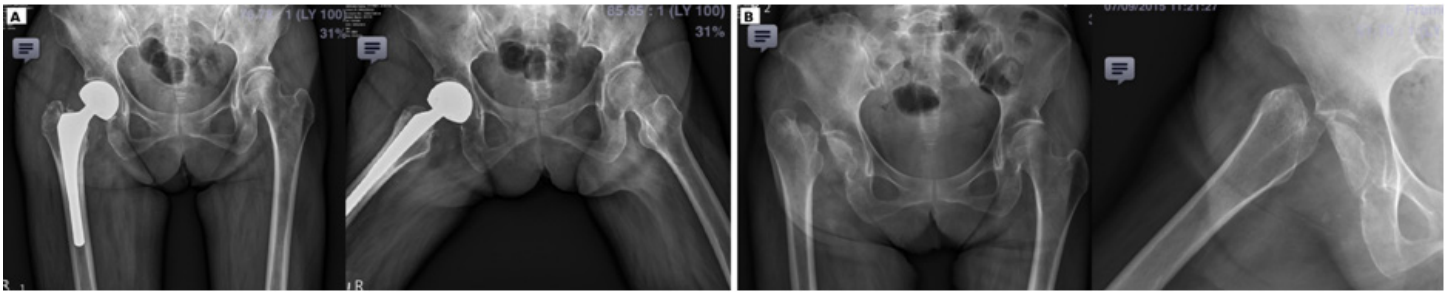

Figure 4. Post-operative radiographs. (A) Hip arthroplasty in satisfactory position; (B) original diagnostic AP film shown for reference.

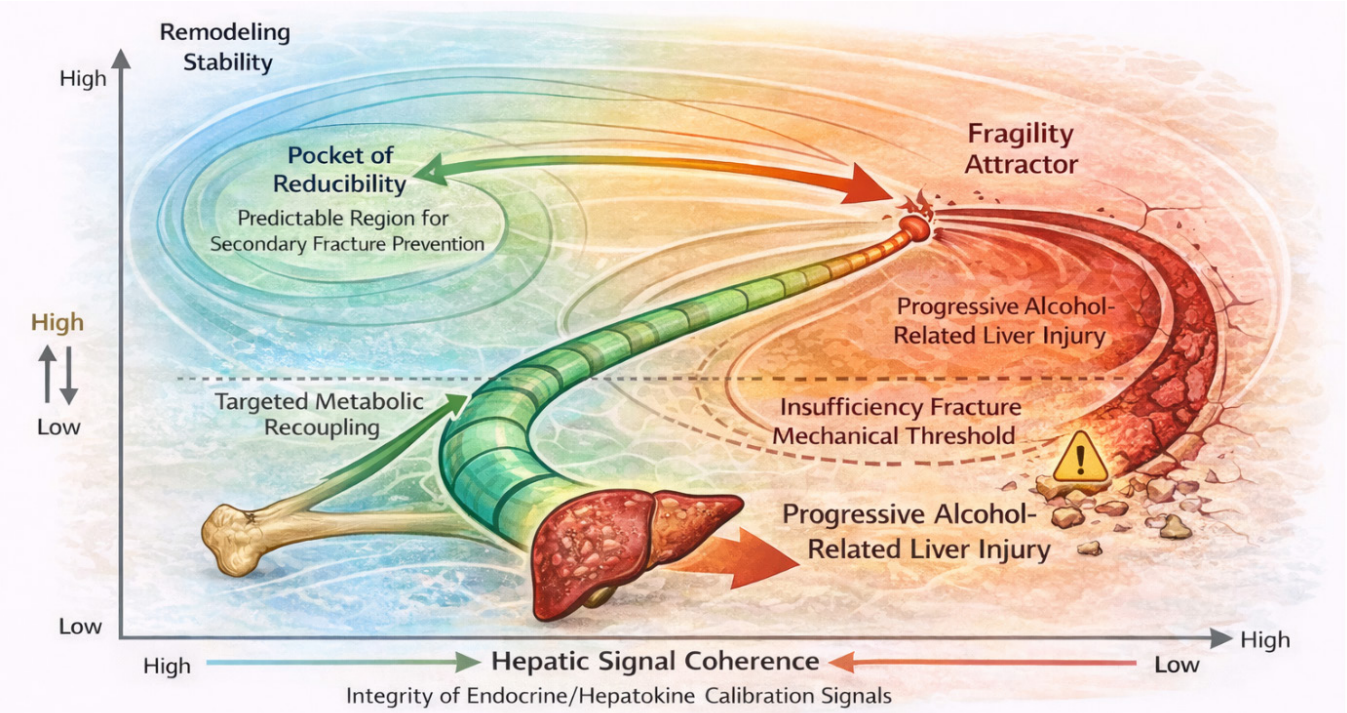

Figure 5. Conceptual phase portrait of metabolic decoupling in the liver–bone axis.

Figure 5. Conceptual phase portrait of metabolic decoupling in the liver-bone axis. The x-axis represents hepatic signal coherence (integrity of endocrine/hepatokine calibration signals), and the y-axis represents remodeling stability (formation/resorption balance). The trajectory illustrates how progressive alcohol-related liver injury can reduce signal coherence and shift the system toward a resorption-dominant 'fragility attractor,' lowering the mechanical threshold required to trigger an insufficiency fracture. Conversely, targeted metabolic recoupling may move the system toward a more predictable region (a practical 'pocket of reducibility') for secondary fracture prevention.

Systems-informed diagnostic planner: coarse-graining the liver-bone axis

This presentation raised a differential that included osteoporotic/insufficiency fracture due to HOD, alcohol-related osteonecrosis, metastatic pathologic fracture, and endocrine/metabolic bone disease. Negative systemic imaging lowered the likelihood of malignancy. The combination of alcohol use disorder, biochemical liver involvement, and atraumatic fracture supported HOD rather than primary osteonecrosis. We propose the following pragmatic algorithm for suspected HOD in orthopaedic settings: (i) screen for CLD risk (viral hepatitis, alcohol, cholestasis, fatty liver), falls, and hypogonadal symptoms; (ii) order baseline laboratories (25-hydroxyvitamin D, calcium, phosphate, alkaline phosphatase, albumin, bilirubin, AST/ALT, γ-GT, PTH, total testosterone where appropriate) and consider bone turnover markers; (iii) obtain DXA for BMD and trabecular bone score when available; (iv) evaluate secondary contributors (malnutrition, corticosteroid exposure, antiviral therapy, cholestyramine); and (v) institute early anti-fracture therapy alongside liver-directed care.

Because the liver-bone network is high-dimensional, the exact remodeling trajectory in an individual patient is difficult to predict a priori. The proposed planner can therefore be interpreted as a coarse-graining algorithm: a small set of accessible laboratory variables is used as a proxy for hepatic signal coherence and bone turnover state (e.g., 25-hydroxyvitamin D, calcium/phosphate, alkaline phosphatase, parathyroid hormone, albumin, bilirubin and transaminases, and testosterone in men; IGF-1 when available). Operationally, triggering this panel in the setting of an atraumatic fracture or unexpectedly severe fracture phenotype functions as a diagnostic noise filter, prioritizing reversible contributors that may 'recouple' the liver-bone axis while definitive fracture management proceeds [1–3,5,6].

Discussion: metabolic decoupling and the fragility attractor in hepatic osteodystrophy

Conceptual reframing: metabolic decoupling in the liver-bone axis

A purely linear causal narrative (alcohol → liver injury → nutrient/hormone deficiency → bone loss) captures important mechanisms but can understate the systems-level feature of hepatic osteodystrophy: the liver and skeleton form a coupled, feedback-regulated network. In this framework, the liver functions as a central metabolic integrator that packages systemic energy, lipid handling, and inflammatory states into endocrine and hepatokine outputs. These outputs act as calibration signals that tune the set-point of bone remodeling. When alcohol-related chronic liver disease degrades the amplitude and coherence of hepatic calibration signals, the remodeling program may drift toward a resorption-dominant equilibrium, creating a clinically observable state of fragility even in the absence of high-energy trauma [1–6].

Coarse-graining metabolic irreducibility: from biomarkers to state variables

At the level of the bone multicellular unit, remodeling emerges from nonlinear interactions among osteoblasts, osteoclasts, stromal cells, cytokines, and matrix cues; as a result, the precise time-course of bone quality decline and fracture threshold in a given individual is not readily derivable from first principles. Consistent with the broader notion of computational irreducibility in complex systems, clinically useful predictability is more plausibly achieved by coarse-graining: mapping the high-dimensional biology into a small number of macroscopic state variables that can be measured and acted upon. In practice, hepatic signal coherence can be approximated by liver function surrogates together with endocrine/nutritional markers (e.g., vitamin D/K status, testosterone where appropriate, albumin, and—

when available—IGF-1), while remodeling stability can be approximated by the formation–resorption balance using bone turnover markers and imaging. This perspective provides the conceptual basis for the diagnostic planner proposed above [1–3,8–11].

Molecular channels of calibration-signal loss: integrating IGFBP1-FGF21 and PP2Ac-LCAT

Mechanistically, hepatic osteodystrophy reflects both impaired bone formation and accelerated resorption. Impaired hepatic 25-hydroxylation and cholestasis-related fat-soluble vitamin malabsorption contribute to vitamin D deficiency, while reduced vitamin K-dependent osteocalcin carboxylation impairs mineralization. Chronic liver disease can reduce hepatic IGF-1 output and disrupt osteoblast anabolic signaling. More recently, hepatokine and lipid-handling pathways have strengthened the biological plausibility of a liver-bone axis beyond inactivity and nutrition: the IGFBP1–FGF21 axis has been linked to osteoclastogenesis, and the PP2Ac–LCAT pathway connects hepatic stress to reverse cholesterol transport and bone loss. Alcohol further suppresses osteoblastogenesis, shifts mesenchymal lineage allocation toward adipogenesis, and increases fracture risk in ways that may not be fully captured by areal BMD alone [1,2,5–8].

Phase-space intuition: a fragility attractor and a pocket of reducibility

Figure 5 visualizes this systems framing as a phase portrait: the x-axis represents hepatic signal coherence (integrity of calibration signals), and the y-axis represents remodeling stability (formation/resorption balance). Progressive alcohol-related liver injury can move patients along a trajectory toward a low-coherence, low-stability region—a 'fragility attractor' in which minimal mechanical perturbation can precipitate an insufficiency fracture. Conversely, coordinated metabolic and endocrine interventions that restore calibration signals and reduce inflammatory and nutritional noise may shift the system toward a more predictable region—a practical 'pocket of reducibility' in which secondary fracture prevention becomes tractable alongside definitive orthopaedic care [1–3,5,6,10,11].

Management implications: metabolic recoupling alongside fracture care

Management in suspected HOD is most effective when treated as two coupled tracks: definitive fracture care for pain control and early mobilization, and metabolic 'recoupling' aimed at restoring hepatic calibration signals and preventing recurrent fragility events. Orthopaedic management should be coupled with metabolic interventions, including vitamin D supplementation (with attention to fat-soluble vitamin kinetics in cholestasis), calcium repletion, and fall prevention; evaluation and treatment of hypogonadism in men; and antiresorptive therapy (e.g., bisphosphonates) or denosumab when appropriate after dental review. Equally important is coordinated hepatology care to address alcohol cessation and the underlying CLD. Post-arthroplasty follow-up should include secondary fracture prevention with laboratory monitoring and DXA when feasible [1,3,5].

Conclusion

First, hepatic osteodystrophy can be viewed not only as the downstream result of isolated deficiencies, but as a coupled-system failure in which loss of hepatic calibration signals shifts remodeling toward a resorption-dominant 'fragility attractor' redefining the clinical goal from simply fixing the broken bone to resetting the system's dynamic stability." Second, because the microdynamics of remodeling are complex, clinically useful action is best supported by coarse-grained state variables and a systems-informed diagnostic planner embedded in orthopaedic pathways for atraumatic or unexpectedly severe fractures. Third, effective care should integrate definitive fracture management with metabolic recoupling (vitamin D/K repletion, evaluation of hypogonadism, nutritional optimization, anti-fracture pharmacotherapy when indicated, and liver-directed care), and prospective studies should test whether these state-based tools improve early detection and secondary fracture prevention [1–8,10,11].

Ethics/Consent

According to the journal's guidance for de-identified case reports, individual patient consent is not required when no directly identifiable information is disclosed. All images and clinical details in this report are fully anonymized and presented solely for educational and scientific purposes. Per institutional policy, single anonymized case reports do not constitute human-subjects research and are exempt from ethics review.

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Cite this article: Chi-Ming Chiang. (2026) Alcohol-related hepatic osteodystrophy presenting as an atraumatic femoral neck fracture: a diagnostic case report and mechanistic framework. Advance Medical & Clinical Research. 7 (1): 251-254.