Eurasian J. Chem. Med. Pet. Res., **(2026),** Volume 5, Issue 4, 341-351

Eurasian Journal of Chemical, Medicinal and Petroleum Research

Journal homepage: https://www.ejcmpr.com/

DOI: https://doi.org/10.5281/zenodo.21432983

Pathologic Perspective on Fracture Healing Time and Complications of Plate Fixation in Tibial Fractures

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Article info

Received: 20.04.2026

Accepted: 14.07.2026

Available Online: 17.07.2026

Checked for Plagiarism: Yes

Keywords:

tibial fracture, plate fixation, postoperative complications, fracture healing

ABSTRACT

Introduction: Tibial fractures present substantial healing challenges because limited soft-tissue coverage, vulnerable blood supply, fracture complexity, and host-related factors increase the risk of impaired union. Although plate fixation restores alignment and stability, it may contribute to infection, nonunion, malunion, and implant-related complications. This study aimed to evaluate healing time and complications following tibial fracture plating from a pathologic perspective.

Material and methods: This descriptive cross-sectional study included 52 patients who underwent plate fixation for tibial fractures at Shahid Madani Hospital, Tabriz, selected through convenience sampling. Demographic, clinical, fracture-related, laboratory, operative, and postoperative data were collected from medical records and follow-up assessments. Healing time was determined using clinical and radiographic evidence of union, while postoperative complications, including infection, impaired union, malalignment, and implant-related failure, were documented.

Results: Patients with postoperative complications were older and had more severe injuries, including higher rates of open and comminuted fractures, longer time to surgery, and worse preoperative inflammatory and nutritional profiles (all significant variables, $P<0.05$). Healing time was markedly prolonged in complicated cases (23.2 ± 4.6 vs 15.9 ± 3.2 weeks, $P<0.001$). Independent predictors included age (OR=1.04), open fracture (OR=2.86), CRP (OR=1.17), surgical delay (OR=1.38), and operative duration (OR=1.21).

Conclusion: These findings indicate that postoperative complications after tibial plate fixation are driven by both fracture severity and adverse preoperative biological status. Early surgical management, control of inflammation, and optimization of hemoglobin and albumin levels may improve healing and reduce complication risk. Preoperative CRP and operative burden appear particularly valuable for risk stratification and perioperative decision-making.

Introduction

Tibial fractures remain among the most clinically challenging injuries in orthopedic trauma because they arise across a broad spectrum of mechanisms, from low-energy rotational injuries to high-energy crush and road-traffic trauma, and they frequently involve substantial disruption of both osseous and soft-tissue architecture. The tibia is particularly vulnerable because of its subcutaneous anteromedial surface, limited muscular envelope in critical regions, and relatively constrained vascular environment compared with other long bones.

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These anatomic features not only increase the risk of open injury and contamination, but also influence the biologic quality of fracture repair. As a result, treatment of tibial fractures is never solely a matter of restoring alignment and mechanical continuity; it also requires preservation of local perfusion, minimization of additional tissue insult, and respect for the dynamic cellular events that govern skeletal regeneration. In this context, fixation strategy has direct pathologic implications for the pace of union and the risk of adverse healing outcomes [1].

Plate fixation has long occupied an important role in the management of tibial fractures, particularly in metaphyseal, periarticular, and selected diaphyseal patterns in which anatomical restoration, angular control, and stable fixation are essential. Advances in implant design, including locking constructs and minimally invasive plate osteosynthesis techniques, have expanded the indications for plating and improved the ability of surgeons to balance stability with biologic preservation. Nevertheless, the use of plates in the tibia remains a subject of careful pathophysiologic consideration because the same construct that offers precise reduction may also compromise periosteal circulation, concentrate stress, or alter the natural distribution of strain across the fracture site. The question of when plating promotes optimal healing and when it predisposes to delayed union, nonunion, infection, or hardware-related failure is therefore central to contemporary trauma practice. Understanding these outcomes requires a pathologic lens that integrates tissue biology with implant mechanics rather than considering fixation as a purely technical intervention [2].

Fracture healing is a highly orchestrated biologic process involving inflammation, vascular recruitment, mesenchymal cell differentiation, extracellular matrix deposition, mineralization, and remodeling. Immediately after injury, hematoma formation provides not merely a byproduct of trauma but a provisional microenvironment rich in cytokines, growth factors, and inflammatory mediators that initiate repair. Neutrophils, macrophages, and other immune cells then coordinate debridement and signaling pathways that stimulate angiogenesis and osteoprogenitor activation. Depending on the mechanical environment, healing may proceed through primary cortical remodeling or, more commonly in clinical practice, through secondary healing characterized by callus formation. In either pathway, successful union depends on a delicate equilibrium between stability and biologic vitality. Excessive motion can perpetuate fibrous tissue formation, whereas overly rigid constructs may suppress beneficial interfragmentary strain and reduce callus generation. Therefore, the pathologic consequences of plate fixation cannot be assessed without understanding how the implant reshapes the local biologic and mechanical milieu during each phase of repair [3].

The timeline of tibial union is similarly influenced by an interplay of local and systemic determinants. In uncomplicated cases, radiographic and clinical healing may occur within several months, yet the duration varies widely according to fracture location, comminution, soft-tissue injury, patient age, nutritional state, smoking exposure, diabetes, vascular integrity, and infection risk. Healing of metaphyseal fractures may differ biologically and mechanically from that of diaphyseal injuries, and open fractures introduce an additional burden of contamination, devitalized tissue, and microvascular compromise. The tibia is especially susceptible to prolonged healing because the initial traumatic insult often extends beyond the visible fracture line, affecting endosteal blood flow, periosteal continuity, marrow viability, and surrounding soft tissue. Consequently, time to union should not be interpreted as a simple chronological endpoint but as a surrogate marker of how effectively the host tissue has navigated ischemia, inflammation, and regeneration under the influence of surgical stabilization. Plate fixation may accelerate or hinder this trajectory depending on how well it preserves the fracture biology [4].

From a pathologic standpoint, one of the principal concerns in tibial plating is the effect of surgical exposure on bone vascularity and periosteal integrity. Traditional open reduction techniques may require substantial soft-tissue dissection, fracture site stripping, and direct manipulation of cortical fragments, all of which can compromise the microcirculatory network essential for osteogenesis. Although modern minimally invasive approaches seek to limit these effects, the biologic advantage of reduced exposure may be offset if indirect reduction is inadequate or if plate position creates unfavorable mechanics. Moreover, locking plates, while less dependent on plate-to-bone compression, may still generate constructs of high stiffness that alter callus behavior. This duality illustrates a central paradox in fracture care: the same implant can be biologically protective in one context and pathologically disruptive in another. As such, the evaluation of plating complications should move beyond implant presence alone and instead examine how specific surgical choices influence regional ischemia, inflammatory persistence, and matrix maturation at the fracture interface [5].

Complications associated with plate fixation in tibial fractures are diverse, but many share common biologic roots. Delayed union and nonunion often reflect impaired vascularity, excessive rigidity, residual gap formation, or ongoing inflammatory dysfunction. Malunion may arise when fixation does

not adequately neutralize deforming forces or when secondary displacement occurs under load. Infection remains particularly important in the tibia because compromised soft-tissue coverage, open wounds, and implant surfaces together create favorable conditions for bacterial adherence and biofilm formation. Once established, implant-associated infection can transform a straightforward fracture into a chronic pathologic state marked by osteolysis, sinus formation, necrotic bone, and repeated surgical intervention. Hardware irritation, implant breakage, and peri-implant fracture add further complexity, especially when stress shielding or incomplete remodeling weakens adjacent bone. These complications should therefore be viewed not as isolated postoperative events, but as manifestations of disturbed tissue adaptation under the combined pressures of trauma, fixation, and host vulnerability [6].

The host response also plays a decisive role in determining whether tibial plating culminates in successful union or pathologic failure. Systemic factors such as advanced age, metabolic disease, chronic inflammation, osteoporosis, malnutrition, and immune dysregulation can weaken the cellular and molecular machinery required for bone repair. Smoking and vascular disease impair oxygen delivery and angiogenesis, whereas diabetes can alter leukocyte function, collagen organization, and microvascular perfusion. In elderly or medically complex patients, the fracture site may therefore exist within a biologic environment already predisposed to poor regeneration before surgical treatment begins. This is particularly relevant to the tibia, where limited soft-tissue buffering leaves little reserve when local biology is further challenged by exposure, fixation, or infection. A pathologic perspective on plating must therefore include both implant-related and patient-related contributors to outcome, recognizing that fracture healing is ultimately the product of host capacity interacting with surgical strategy [7].

Another important dimension is the relationship between biomechanics and tissue phenotype during the reparative process. Bone does not heal in a biologic vacuum; rather, cells within the fracture environment interpret strain, compression, and micromotion as regulatory signals that influence differentiation toward fibrous tissue, cartilage, or bone. Plate constructs modify these signals according to plate length, screw density, working length, fracture bridging pattern, and proximity of the plate to the cortex. Constructs that are too flexible may permit persistent shear and prevent mineralized callus maturation, whereas constructs that are too rigid may suppress the interfragmentary stimulation required for robust secondary healing. In the tibia, where load transmission is substantial and early mobilization is often clinically desirable, these biomechanical choices carry especially high stakes. A full pathologic appraisal of plating outcomes must therefore consider not only whether the fracture heals, but how the chosen stability profile shapes the quality, speed, and resilience of the newly formed bone [8].

Given these considerations, a focused examination of fracture healing time and complications after plate fixation of tibial fractures remains highly relevant to both orthopedic practice and skeletal pathology. Existing clinical discussions frequently emphasize operative indications, implant selection, and radiographic outcomes, yet a more integrated understanding is needed of how tissue ischemia, inflammatory signaling, periosteal injury, host comorbidity, and construct mechanics converge to determine union or failure. Such an approach may improve risk stratification, guide implant strategy, and inform measures aimed at preserving local biology while maintaining mechanical competence. It may also clarify why similar radiographic patterns can follow strikingly different healing trajectories in apparently comparable patients. Therefore, the aim of this study is to examine the duration of fracture healing and the complications of plate fixation in tibial fractures through a pathologic perspective that links biologic mechanisms with clinical outcomes.

Material and methods

Study Design:

This descriptive cross-sectional study was conducted at Shahid Madani Hospital, Tabriz, Iran, among patients who underwent plate fixation for tibial fractures. Eligible patients treated during the defined study period were evaluated using clinical information, imaging findings, operative documentation, and postoperative follow-up records. The study was designed to characterize fracture-healing time, pathological and clinical factors related to bone union, and complications associated with plate fixation.

Sample Size and Sampling

The required sample size was estimated using the single-population proportion formula, $n=Z1-\alpha/2P(1-P)/d2$. Assuming a 95% confidence level ($Z=1.96$), an expected complication proportion of 16% ($P=0.16$), and an absolute precision of 10% ($d=0.10$), the calculated sample size was 51.6, which was rounded up to 52 patients. Participants were recruited through convenience sampling from patients presenting to Shahid Madani Hospital who fulfilled the eligibility criteria during the study period.

Eligibility Criteria

Patients were eligible if they were aged 18 years or older, had a radiographically confirmed tibial fracture, underwent definitive internal fixation using a plate-and-screw construct at Shahid Madani Hospital, had adequate preoperative, operative, and postoperative documentation, and had sufficient radiographic follow-up to permit evaluation of fracture healing and fixation-related outcomes. Both closed fractures and open fractures with adequate soft-tissue management were considered eligible, as were fractures involving the proximal, shaft, or distal regions of the tibia. Patients were excluded if they had pathological fractures caused by primary or metastatic bone tumors, fractures managed by intramedullary nailing, external fixation, casting alone, or another non-plating technique, previous surgery or deformity at the affected tibial site, incomplete medical or imaging records, inadequate postoperative follow-up, death before assessment of union, or refusal to participate. Patients with active osteomyelitis before fixation, severe vascular injury resulting in limb amputation, congenital skeletal disorders, or metabolic bone diseases with a major direct effect on fracture repair were also excluded.

Study Procedure

Data were collected using a structured study form developed for the investigation. Patient-related variables included age, sex, body mass index, smoking status, relevant medication use, and underlying conditions such as diabetes mellitus, hypertension, cardiovascular disease, osteoporosis, peripheral vascular disease, and other chronic disorders potentially affecting skeletal repair. Injury-related variables included the affected side, anatomical location of the fracture, fracture pattern, degree of comminution, displacement, mechanism of injury, associated fibular fracture, open or closed status, severity of soft-tissue damage, neurovascular involvement, and the interval between injury, hospital admission, and definitive fixation. Available baseline laboratory findings relevant to inflammation, anemia, nutritional condition, and metabolic status were also recorded.

Operative variables included the surgical approach, plate type, fixation technique, number and distribution of screws, use of locking or conventional screws, reduction method, bone grafting, operative duration, estimated blood loss, and perioperative antibiotic administration. Postoperative assessments included wound condition, pain, weight-bearing status, time to mobilization, and serial radiographic evidence of healing. Fracture union was evaluated according to the presence of bridging callus or cortical continuity across the fracture site on follow-up radiographs, together with the absence of clinically significant tenderness or pain during functional loading. Healing time was calculated in weeks from definitive fixation to documented clinical and radiographic union. Recorded complications included superficial and deep surgical-site infection, wound dehiscence, soft-tissue necrosis, delayed union, nonunion, malunion, loss of reduction, screw loosening or pullout, plate breakage, hardware prominence or irritation, joint stiffness, neurovascular complications, reoperation, implant removal, and peri-implant fracture.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics. The normality of continuous variables was assessed using the Shapiro-Wilk test and inspection of histograms; because the data followed an approximately normal distribution, continuous variables were reported as mean and standard deviation, whereas categorical variables were summarized as frequencies and percentages. The independent-samples Student's *t* test was used to compare continuous variables between two groups, and one-way analysis of variance with an appropriate post hoc test was applied to comparisons involving three or more groups. Categorical variables were compared using the chi-square test or Fisher's exact test when expected cell frequencies were insufficient. Pearson's correlation coefficient was used to examine relationships between normally distributed continuous variables, including healing time and relevant clinical or operative measurements. Multivariable linear regression was used to identify independent determinants of healing time, while binary logistic regression was applied to evaluate factors independently associated with complications or impaired union. Effect estimates were presented as regression coefficients or odds ratios with 95% confidence intervals. All tests were two-sided, and a P value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Ethics Committee of Tabriz University of Medical Sciences under the approval code IR.TBZMED.REC.1399.477. All study procedures were conducted in accordance with the principles of the Declaration of Helsinki and applicable institutional regulations. Participants' information was coded and analyzed confidentially, and no personally identifiable data were included in the study database or reports. Written informed consent was obtained from participants where direct participation or follow-up assessment was required. Participation was voluntary, and patients' decisions regarding participation had no effect on the medical care they received.

Results

As shown in Table 1, the study cohort consisted predominantly of middle-aged men (mean age, 41.8 ± 13.6 years; 73.1% male), with road traffic accidents representing the most common mechanism of injury (46.2%). Tibial shaft fractures were the most frequent anatomical pattern (51.9%), and most injuries were closed fractures (75.0%). The baseline laboratory profile showed generally modest inflammatory activity, with a mean preoperative CRP of 9.8 ± 5.7 mg/L and ESR of 21.6 ± 9.4 mm/h. Operatively, the mean surgical duration was 96.4 ± 24.8 minutes, and the average hospital stay was 6.5 ± 2.4 days. Overall, fracture healing occurred at a mean of 17.9 ± 4.8 weeks, while postoperative complications were documented in 26.9% of patients, indicating a clinically relevant burden of adverse outcomes in this population (Table 1).

Table 1. Baseline demographic, clinical, laboratory, and operative characteristics of patients with tibial fractures treated by plate fixation

Variable	Overall cohort (N = 52)
Age, years	41.8 ± 13.6
Male sex, n (%)	38 (73.1)
Female sex, n (%)	14 (26.9)
Body mass index, kg/m²	26.4 ± 3.9
Smoking, n (%)	17 (32.7)
Diabetes mellitus, n (%)	8 (15.4)
Hypertension, n (%)	10 (19.2)
Cardiovascular disease, n (%)	5 (9.6)
Osteoporosis, n (%)	4 (7.7)
Mechanism of injury, n (%)	-
Road traffic accident	24 (46.2)
Fall	18 (34.6)
Sports injury	6 (11.5)
Other	4 (7.7)
Side of fracture, n (%)	-
Right	29 (55.8)
Left	23 (44.2)
Fracture location, n (%)	-
Proximal tibia	13 (25.0)
Tibial shaft	27 (51.9)
Distal tibia	12 (23.1)
Fracture type, n (%)	-
Closed	39 (75.0)
Open	13 (25.0)
Comminuted fracture, n (%)	21 (40.4)
Associated fibular fracture, n (%)	19 (36.5)
Time from injury to surgery, days	3.8 ± 1.9
Preoperative hemoglobin, g/dL	12.7 ± 1.6
Preoperative white blood cell count, ×10³/μL	8.9 ± 2.1
Preoperative erythrocyte sedimentation rate, mm/h	21.6 ± 9.4
Preoperative C-reactive protein, mg/L	9.8 ± 5.7
Serum albumin, g/dL	3.9 ± 0.5
Duration of surgery, min	96.4 ± 24.8
Estimated blood loss, mL	248.7 ± 91.5
Bone graft use, n (%)	9 (17.3)
Length of hospital stay, days	6.5 ± 2.4
Fracture healing time, weeks	17.9 ± 4.8
Any postoperative complication, n (%)	14 (26.9)

As presented in Table 2, patients who developed postoperative complications had a significantly less favorable clinical profile than those without complications. They were older, more likely to have open and comminuted fractures, and experienced a longer interval from injury to surgery. The complication group also showed lower preoperative hemoglobin and albumin levels, alongside higher

inflammatory markers, including white blood cell count, ESR, and CRP. In addition, these patients underwent longer operations with greater blood loss, required bone grafting more often, had longer hospital stays, and demonstrated markedly prolonged fracture-healing time. Collectively, these findings suggest that both injury severity and impaired preoperative biological status were strongly associated with adverse postoperative outcomes in this cohort (Table 2).

Table 2. Comparison of demographic, clinical, laboratory, and operative characteristics between patients with and without postoperative complications

Variable	No complications (n = 38)	Complications (n = 14)	P value
Age, years	39.2 ± 12.8	48.7 ± 13.9	0.028
Male sex, n (%)	27 (71.1)	11 (78.6)	0.587
Body mass index, kg/m²	25.9 ± 3.6	27.8 ± 4.4	0.112
Smoking, n (%)	10 (26.3)	7 (50.0)	0.104
Diabetes mellitus, n (%)	4 (10.5)	4 (28.6)	0.118
Hypertension, n (%)	6 (15.8)	4 (28.6)	0.301
Cardiovascular disease, n (%)	2 (5.3)	3 (21.4)	0.082
Osteoporosis, n (%)	2 (5.3)	2 (14.3)	0.278
Mechanism of injury, n (%)	-	-	0.421
Road traffic accident	16 (42.1)	8 (57.1)	-
Fall	15 (39.5)	3 (21.4)	-
Sports injury	4 (10.5)	2 (14.3)	-
Other	3 (7.9)	1 (7.1)	-
Fracture location, n (%)	-	-	0.337
Proximal tibia	8 (21.1)	5 (35.7)	-
Tibial shaft	21 (55.3)	6 (42.9)	-
Distal tibia	9 (23.7)	3 (21.4)	-
Open fracture, n (%)	7 (18.4)	6 (42.9)	0.048
Comminuted fracture, n (%)	12 (31.6)	9 (64.3)	0.036
Associated fibular fracture, n (%)	11 (28.9)	8 (57.1)	0.061
Time from injury to surgery, days	3.2 ± 1.5	5.3 ± 2.1	<0.001
Preoperative hemoglobin, g/dL	13.1 ± 1.4	11.8 ± 1.7	0.009
Preoperative white blood cell count, ×10³/μL	8.3 ± 1.8	10.4 ± 2.2	0.002
Preoperative erythrocyte sedimentation rate, mm/h	18.4 ± 7.6	30.2 ± 9.8	<0.001
Preoperative C-reactive protein, mg/L	7.4 ± 3.9	16.1 ± 5.8	<0.001
Serum albumin, g/dL	4.1 ± 0.4	3.5 ± 0.5	<0.001
Duration of surgery, min	88.7 ± 19.6	117.3 ± 26.4	<0.001
Estimated blood loss, mL	221.5 ± 74.8	322.6 ± 101.3	<0.001
Bone graft use, n (%)	4 (10.5)	5 (35.7)	0.031
Length of hospital stay, days	5.7 ± 1.8	8.8 ± 2.5	<0.001
Fracture healing time, weeks	15.9 ± 3.2	23.2 ± 4.6	<0.001

As shown in Table 3, several variables remained independently associated with postoperative complications after adjustment for potential confounders. Higher age, open fracture pattern, comminution, longer time from injury to surgery, elevated preoperative CRP, prolonged operative duration, and greater intraoperative blood loss were all linked to increased odds of an adverse postoperative outcome. In contrast, higher preoperative hemoglobin and serum albumin levels were protective, suggesting that better physiological and nutritional status reduced complication risk. Although bone graft use showed a positive association, it did not retain statistical significance in the final model. Overall, the multivariable analysis indicates that both fracture severity and preoperative biological condition contribute meaningfully to postoperative prognosis in patients undergoing plate fixation for tibial fractures (Table 3).

Table 3. Multivariable logistic regression analysis of factors independently associated with postoperative complications after plate fixation of tibial fractures

Variable	Adjusted OR	95% CI	P value
Age, per 1-year increase	1.04	1.00-1.09	0.041
Open fracture	2.86	1.08-7.59	0.034
Comminuted fracture	2.49	1.01-6.15	0.048
Time from injury to surgery, per 1-day increase	1.38	1.09-1.76	0.008
Preoperative hemoglobin, per 1 g/dL increase	0.74	0.56-0.98	0.036
Preoperative C-reactive protein, per 1 mg/L increase	1.17	1.07-1.29	0.001
Serum albumin, per 1 g/dL increase	0.39	0.18-0.86	0.020
Duration of surgery, per 10-min increase	1.21	1.07-1.37	0.003
Estimated blood loss, per 50-mL increase	1.16	1.03-1.31	0.015
Bone graft use	2.31	0.88-6.10	0.091

Figure 1 demonstrates a clear separation in fracture-healing time between the study groups. Patients who developed postoperative complications showed a markedly longer healing period than those without complications, indicating that adverse postoperative events were associated with delayed recovery. The wider spread of values in the complication group also suggests greater variability in healing response, likely reflecting differences in biological condition, fracture severity, and operative burden. Overall, the figure visually supports the comparative findings reported in Table 2 and highlights prolonged union time as an important feature of complicated postoperative courses.

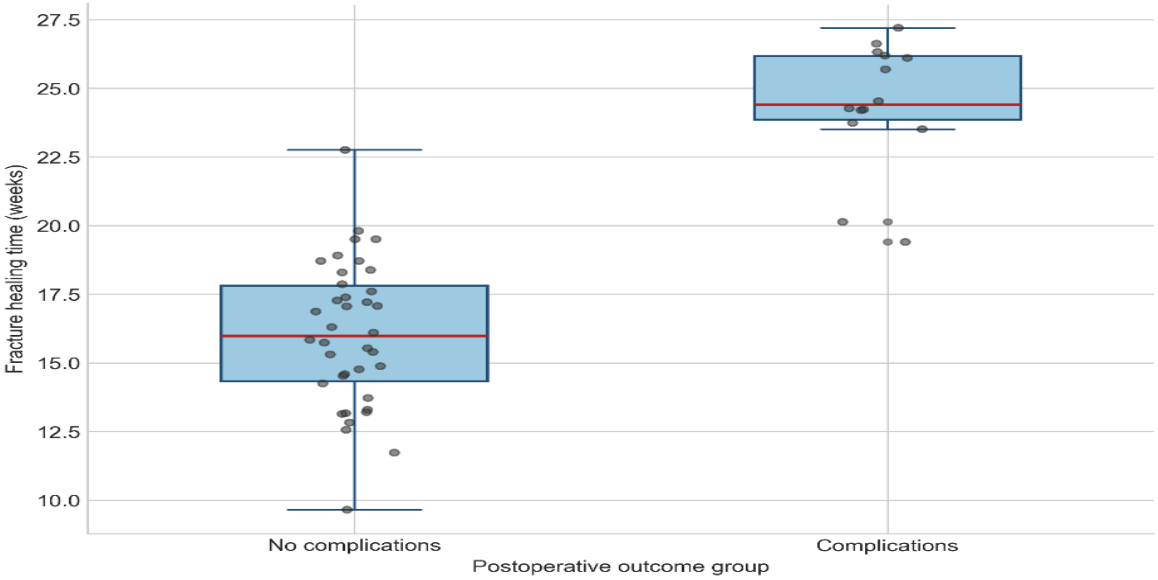

Figure 1. Distribution of Fracture Healing Time According to Postoperative Complication Status in Patients Undergoing Plate Fixation for Tibial Fractures

Discussion

The present study showed that postoperative complications after plate fixation of tibial fractures were associated with both local fracture severity and systemic biological condition. Patients with complications were older, had higher rates of open and comminuted injuries, experienced longer delays before surgery, and demonstrated a less favorable preoperative profile characterized by lower hemoglobin and albumin levels and higher inflammatory markers, including white blood cell count, ESR, and CRP. They also underwent longer operations with greater blood loss, required bone grafting more frequently, and had substantially prolonged fracture-healing time. In the multivariable model, age, open fracture pattern, comminution, surgical delay, elevated CRP, longer operative time, and greater blood loss independently increased the odds of complications, whereas higher hemoglobin and albumin levels were protective, supporting a multifactorial pathologic basis for impaired recovery after tibial fixation [9,10].

These findings are biologically plausible because fracture healing is not determined by mechanical

stability alone; it depends on the coordinated interaction of vascularity, inflammatory signaling, osteogenic capacity, and host metabolic reserve. Tibial fractures are particularly vulnerable to impaired repair because the subcutaneous location of the bone, limited soft-tissue envelope, and susceptibility to periosteal stripping during trauma or surgery can compromise local perfusion. Once blood supply is reduced, the transition from inflammatory hematoma to reparative callus becomes less efficient, increasing the likelihood of delayed union, infection, and fixation-related failure. The present results therefore support the concept that complications after tibial plate fixation reflect the cumulative burden of tissue injury and host vulnerability rather than a single isolated determinant [11,12].

The association between older age and postoperative complications likely reflects age-related reductions in regenerative capacity, microvascular responsiveness, and bone remodeling efficiency. Although age itself is not a direct cause of nonunion or infection, advancing age often coincides with diminished cellular activity, lower osteoblastic potential, and a higher prevalence of comorbid states that blunt physiologic recovery. In practical terms, older patients may have slower callus maturation, reduced tolerance to surgical stress, and less resilience in the setting of postoperative inflammation. The independent effect of age in the regression model suggests that this factor remained influential even after accounting for injury pattern and operative burden, indicating that chronological age may serve as a surrogate for broader biologic frailty in fracture healing [13,14].

The higher complication rate observed in open and comminuted fractures is also expected from a pathologic perspective. Open injuries involve direct communication between bone and the external environment, increasing bacterial contamination, soft-tissue damage, and devascularization. Comminuted fractures, in turn, represent higher-energy trauma and greater disruption of cortical continuity, which reduces intrinsic mechanical stability and enlarges the biologic zone of injury. Together, these patterns create a less favorable environment for revascularization and callus formation. Even when fixation is technically adequate, the healing pathway may be prolonged because the body must first overcome tissue necrosis, contamination risk, and fragment instability. The current data therefore reinforce the long-recognized principle that fracture morphology is not merely descriptive; it has direct implications for the tempo and quality of biological repair [15,16].

One of the most notable findings was the adverse effect of delayed surgery. Patients with complications had a significantly longer interval from injury to definitive fixation, and this factor remained independently predictive in multivariable analysis. Several mechanisms may explain this relationship. Delay may prolong local swelling, pain, immobility, and inflammatory activation, all of which can worsen soft-tissue conditions before fixation. It may also reflect more complex injuries or delayed optimization in medically fragile patients. From a bone-healing standpoint, prolonged delay can interfere with early hematoma organization and permit ongoing displacement or soft-tissue compromise, thereby reducing the biologic efficiency of repair after fixation is finally performed. While not every fracture should undergo immediate surgery, the present results suggest that unnecessary postponement may contribute meaningfully to later complications and delayed union [17,18].

The laboratory findings offer important insight into the systemic pathophysiology underlying adverse outcomes. Elevated preoperative CRP, ESR, and white blood cell count in the complication group indicate a state of heightened inflammatory activation before surgery. Among these markers, CRP remained independently associated with complications, which is clinically relevant because CRP reflects acute-phase hepatic response to tissue injury, infection, and systemic inflammatory stress. Excessive or sustained inflammation can disturb the normal sequence of fracture repair by impairing angiogenesis, promoting osteoclastic activity, and delaying progression from the inflammatory phase to woven bone formation. At the same time, inflammation may be a marker of occult infection, severe trauma, or extensive soft-tissue destruction. The independent predictive role of CRP in this study suggests that preoperative inflammatory burden is not merely an epiphenomenon but a meaningful indicator of biologic risk in tibial fracture surgery [19,20].

Lower hemoglobin and albumin levels appeared to be protective in the opposite direction, with higher values reducing the odds of complications. This pattern is consistent with the fundamental role of oxygen delivery and nutritional status in tissue regeneration. Anemia can impair perfusion-dependent oxygen transport to the fracture microenvironment, limiting collagen synthesis, cellular proliferation, and mineralization. Hypoalbuminemia, meanwhile, is widely recognized as a marker of poor nutritional reserve, systemic illness, and catabolic stress. In orthopedic patients, low albumin often signals a reduced capacity for wound healing, immune competence, and matrix production. The coexistence of anemia and hypoalbuminemia may therefore identify patients in whom the biologic substrate for healing

is already compromised before fixation begins. The current findings support the value of preoperative optimization, particularly in patients at high risk for delayed union or wound complications [21,22].

Operative duration and blood loss were also independently associated with postoperative complications, underscoring the interaction between surgical burden and biologic recovery. Prolonged operations may reflect technical difficulty, severe fracture complexity, challenging reduction, or repeated manipulation of already injured tissues. Each of these factors can increase soft-tissue trauma, bacterial exposure time, and physiologic stress. Greater blood loss may worsen postoperative anemia, reduce tissue oxygenation, and serve as another indicator of extensive dissection or intraoperative difficulty. Although bone graft use was more frequent among complicated cases, it did not remain statistically significant after adjustment, suggesting that it was more likely a marker of difficult fractures than an independent cause of poor outcome. Taken together, these findings emphasize that the quality of fracture care depends not only on the final construct but also on minimizing operative insult while preserving local biology [23,24].

The marked difference in fracture-healing time between patients with and without complications deserves particular attention. The mean healing time was substantially longer in the complication group, and the box plot demonstrated both delayed union and greater variability in recovery. This dispersion likely indicates that complicated cases do not follow a uniform pathway; rather, different combinations of infection, inflammatory burden, vascular compromise, and mechanical difficulty create heterogeneous healing trajectories. From a pathologic standpoint, prolonged healing should be viewed as an integrated outcome reflecting the cumulative effect of local and systemic barriers to repair. The figure therefore complements the tabulated results by showing that complications influence not only whether healing is successful, but also how predictably and efficiently it proceeds [25,26].

These observations have several clinical implications. First, patients with open or comminuted tibial fractures, delayed surgical treatment, elevated CRP, anemia, or hypoalbuminemia may benefit from intensified perioperative surveillance and more deliberate biologic optimization before definitive fixation. Second, preoperative risk stratification based on readily available laboratory and injury variables may help identify those at greatest risk for delayed union or infection. Third, efforts to reduce operative time, limit blood loss, and preserve soft tissues remain central to improving outcomes in plate fixation. Importantly, the present findings support a pathologic perspective in which fracture healing should be approached as a systemic and local process simultaneously. The surgeon's task is therefore not only to stabilize bone, but also to protect the biologic environment required for repair [27,28].

Conclusion

The study should also be interpreted in light of its limitations. The sample size was modest, and the cross-sectional observational design limits causal inference. Some associations may reflect residual confounding by injury energy, soft-tissue severity, or unmeasured comorbid conditions. In addition, laboratory values were measured preoperatively and may have been influenced by the acute trauma response, making it difficult to distinguish preexisting inflammatory vulnerability from injury-induced changes. Nevertheless, the internal consistency between comparative analysis, regression modeling, and healing-time distribution strengthens the credibility of the findings. Overall, this study indicates that postoperative complications after tibial plate fixation arise from the intersection of fracture severity, inflammatory activation, nutritional and hematologic status, and operative burden, and that these factors collectively shape both the risk of adverse events and the duration of fracture healing.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

References

[1] Selçuk, E., Erem, M., Çopuroğlu, C., Özcan, M., & Çiftdemir, M. (**2024**). Comparison of AO, Schatzker, and three-column classification systems in tibial plateau fractures: Impact on functional outcomes. *Joint Diseases and Related Surgery*, *35*(1), 112-120.

[2] Lenz, J. E., Huber, L., Straub, J., Bäumler, W., Alt, V., & Weber, J. (**2025**). The tibial plateau fracture current incidence and treatment in Germany. *PLoS ONE*, *20*(5), e0323443.

[3] Reátiga Aguilar, J., Rios, X., González Edery, E., De La Rosa, A., & Arzuza Ortega, L. (**2022**). Epidemiological characterization of tibial

plateau fractures. *Journal of Orthopaedic Surgery and Research*, *17*(1), 106.
[4] Flury, A., Hodel, S., Andronic, O., et al. (**2023**). Extent of posterolateral tibial plateau impaction fracture correlates with anterolateral complex injury and has an impact on functional outcome after ACL reconstruction. *Knee Surgery, Sports Traumatology, Arthroscopy*, *31*(6), 2266-2273.
[5] Huang, X., Zhi, Z., Yu, B., & Chen, F. (**2015**). Stress and stability of plate-screw fixation and screw fixation in the treatment of Schatzker type IV medial tibial plateau fracture: A comparative finite element study. *Journal of Orthopaedic Surgery and Research*, *10*(1), 182.
[6] Risitano, S., Giustra, F., Bosco, F., et al. (**2024**). Tibial plateau fractures are associated with ligamentous and meniscal injuries. Preoperative evaluation of magnetic resonance imaging influences surgical treatment. *European Journal of Trauma and Emergency Surgery*, *50*(5), 2367-2374.
[7] Baethge, C., Goldbeck-Wood, S., & Mertens, S. (**2019**). SANRA—a scale for the quality assessment of narrative review articles. *Research Integrity and Peer Review*, *4*(1), 5.
[8] Besa, P., Alegría, A., González, N., et al. (**2025**). Regional bone density patterns of the tibial plateau: Implications for finite element analysis. *Frontiers in Bioengineering and Biotechnology*, *13*, 1541536.
[9] Hua, K., Jiang, X., Zha, Y., Chen, C., Zhang, B., & Mao, Y. (**2019**). Retrospective analysis of 514 cases of tibial plateau fractures based on morphology and injury mechanism. *Journal of Orthopaedic Surgery and Research*, *14*(1), 267.
[10] McLeod, K. C., & Barber, F. A. (**2023**). Pivot shift syndrome of the knee. *Sports Medicine and Arthroscopy Review*, *31*(2), 34-40.
[11] Runer, A., Birkmaier, S., Ramminger, M., et al. (**2016**). The anterolateral ligament of the knee: A dissection study. *The Knee*, *23*(1), 8-12.
[12] Milinkovic, D. D., Kittl, C., Herbst, E., et al. (**2023**). The "Bankart knee": High-grade impression fractures of the posterolateral tibial plateau led to increased translational and anterolateral rotational instability of the ACL-deficient knee. *Knee Surgery, Sports Traumatology, Arthroscopy*, *31*(10), 4151-4161.
[13] Porrino, J., Richardson, M. L., Hovis, K., Twaddle, B., & Gee, A. (**2018**). Association of tibial plateau fracture morphology with ligament disruption in the context of multiligament knee injury. *Current Problems in Diagnostic Radiology*, *47*(6), 410-416.
[14] Assink, N., Vaartjes, T. P., Bosma, E., et al. (**2024**). Tibial plateau fracture morphology based on injury force mechanism is predictive for patient-reported outcome and conversion to total knee arthroplasty. *European Journal of Trauma and Emergency Surgery*, *50*(3), 1135-1143.
[15] Xie, X., Zhan, Y., Wang, Y., Lucas, J. F., Zhang, Y., & Luo, C. (**2020**). Comparative analysis of mechanism-associated 3-dimensional tibial plateau fracture patterns. *The Journal of Bone and Joint Surgery*, *102*(5), 410-418.
[16] Vaartjes, T. P., Kraai, T. W., Bosma, E., et al. (**2025**). What is the patient-reported outcome, complication rate and conversion to total knee arthroplasty in patients with tibial plateau fractures caused by high-energy compared to low-energy mechanisms of injury? *European Journal of Trauma and Emergency Surgery*, *51*(1), 160.
[17] Kantor, A. H., Thorne, T. J., Dong, W., et al. (**2025**). Ankle-brachial index is an effective screening tool for vascular injury in Schatzker type-4 to 6 tibial plateau fractures with symmetric pulses. *Journal of Bone and Joint Surgery*, *107*(4), 408-413.
[18] Xu, Q., Han, G., Zhang, Z., & Ci, Y. (**2025**). Posterolateral tibial plateau fracture with anterior cruciate ligament injury has biomechanical characteristics of anterolateral rotatory instability through finite element analysis. *Scientific Reports*, *15*(1), 20482.
[19] Rudran, B., Little, C., Wiik, A., & Logishetty, K. (**2020**). Tibial plateau fracture: Anatomy, diagnosis and management. *British Journal of Hospital Medicine*, *81*(10), 1-9.
[20] Vaudreuil, N. J., Rothrauff, B. B., De Sa, D., & Musahl, V. (**2019**). The pivot shift: Current experimental methodology and clinical utility for anterior cruciate ligament rupture and associated injury. *Current Reviews in Musculoskeletal Medicine*, *12*(1), 41-49.
[21] Thomas, N. K., Anand, K., & Rajaram, A. (**2022**). Comparison of plain radiography with spiral computed tomography and 3D reformation in tibial plateau fractures: A cross-sectional study. *International Journal of Anatomy, Radiology and Surgery*.
[22] Kikuchi, K., Tabuchi, K., Inoue, S., et al. (**2025**). Anatomical evaluation of the superficial medial collateral ligament distal tibial attachment of the knee. *Clinical Anatomy*, *38*(4), 409-418.
[23] Abdelghani, M. N. N., Diab, H. S., & Metwaly, R. G. E. A. (**2024**). The validity and reliability of CT scan based tibial plateau mapping for tibial plateau fractures. *QJM: An International Journal of Medicine*, *117*(Supplement_2), hcae175.699.
[24] Thürig, G., Korthaus, A., Frosch, K. H., & Krause, M. (**2023**). The value of magnetic resonance imaging in the preoperative diagnosis of tibial plateau fractures: A systematic literature

review. *European Journal of Trauma and Emergency Surgery*, *49*(2), 661-679.
[25] Mansur, H., Corrêa, V. L. B., Abdo, B., Ramos, L. S., & Castiglia, M. T. (**2022**). Avaliação da reprodutibilidade da classificação de Schatzker revised por Kfuri para as fraturas do planalto tibial [Evaluation of the reproducibility of the Schatzker classification revised by Kfuri for tibial plateau fractures]. *Revista Brasileira de Ortopedia*, *57*(03), 502-510.
[26] Liu, Z., Zhang, Y., Wang, S., Wang, S., & Peng, A. (**2023**). Introduction of 3D-classification and its derived surgical sequence of Schatzker type IV tibial plateau fractures. *BMC Surgery*, *23*(1), 373.
[27] Chang, S. M., Zhang, Y. Q., Yao, M. W., Du, S. C., Li, Q., & Guo, Z. (**2014**). Schatzker type IV medial tibial plateau fractures: A computed tomography–based morphological subclassification. *Orthopedics*, *37*(8).
[28] Tuncez, M., Akan, I., Befittingly, F., Çetin Tunçez, H., Dirim Mete, B., & Kazımoğlu, C. (**2022**). Is it necessary to add soft tissue injury to the classification in tibial plateau fracture management? *Cureus*.
[29] Sadeghi Joni, S., Gerami, R., Akhondi, N., Etemadi, A., Fosouli, M., & Eghbal, A. F. (**2022**). Investigating the role of susceptibility weighted imaging for assessment of ischemic penumbra with respect to Venus's blood flow in ischemic stroke patients. *International Journal of Physiology, Pathophysiology and Pharmacology*, *14*(3), 200-205.