




Aspartate Aminotransferase-to-Platelet Ratio Index (APRI) as a Predictor of Mortality in Patients with Femoral Fracture: A Retrospective Cohort Analysis

Pao-Jen Kuo ^{1,*}, Shao-Chun Wu ^{2,*}, Shiun-Yuan Hsu³, Ching-Hua Hsieh ¹

¹Department of Plastic Surgery, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, 83301, Taiwan; ²Department of Anesthesiology, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, 83301, Taiwan; ³Department of Trauma Surgery, Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, 83301, Taiwan

*These authors contributed equally to this work

Correspondence: Ching-Hua Hsieh, Email m93chinghua@gmail.com

Background: Femoral fractures carry high mortality in elderly patients. The aspartate aminotransferase-to-platelet ratio index (APRI), a noninvasive liver function marker, may reflect physiological vulnerability. Its prognostic role in trauma remains unclear.

Methods: We retrospectively analyzed 4,310 adult patients (2009–2023) with femoral fractures undergoing surgery. APRI was calculated on admission. The primary outcome was in-hospital mortality. Multivariable logistic regression and ROC analysis identified independent predictors and optimal APRI cut-off.

Results: Among 4,310 patients, 85 (2.0%) died in-hospital. Non-survivors had significantly higher APRI (1.4 ± 1.5 vs 0.7 ± 2.1 , $p = 0.008$), higher AST levels, and greater injury severity. APRI independently predicted mortality (adjusted OR 1.57 per unit, 95% CI 1.41–1.98, $p = 0.023$). ROC analysis yielded an AUC of 0.670. An $APRI \geq 0.74$ provided 55% sensitivity and 78% specificity. Patients with $APRI \geq 0.74$ had higher mortality (4.9% vs 1.1%, adjusted OR 2.82, $p < 0.001$) and longer hospital stays.

Conclusion: Given APRI's moderate predictive value for adverse outcomes, we cautiously recommend including APRI in pre-operative risk assessment for hip fracture patients. This approach may help identify high-risk cases for targeted interventions.

Keywords: APRI, femoral fracture, trauma, mortality prediction, liver function

Introduction

Femoral fractures in the elderly are a major public health concern, associated with substantial morbidity and mortality despite advances in care. With 1-year mortality rate of 29% for all femoral fractures, approximately 26% of women and 37% of men suffering a femoral fracture die within one year.¹ Early identification of patients at elevated risk of mortality could enable targeted perioperative management and potentially improve outcomes. Traditional risk factors for post-fracture mortality include advanced age, male sex, medical comorbidities, and delays in surgery.^{2–6} However, laboratory markers reflecting a patient's physiological reserve and organ function may provide additional prognostic insight.

One emerging prognostic indicator is the aspartate aminotransferase-to-platelet ratio index (APRI), originally developed as a noninvasive marker of hepatic fibrosis in chronic hepatitis.⁷ APRI is calculated from routine blood tests (AST and platelet count) and correlates with the degree of liver fibrosis or dysfunction.⁷ Even mild underlying liver disease has been linked to worse surgical outcomes; in orthopedic patients, comorbid cirrhosis is associated with higher postoperative complications and mortality.^{8,9} APRI offers a convenient proxy for liver health: it has been validated as a cost-effective alternative to liver biopsy and has shown prognostic value in other surgical fields.^{10–14} For example, elevated APRI has

been associated with increased postoperative morbidity and mortality in vascular surgery¹⁵ and general surgery populations.^{16,17} In addition, orthopedic patients with higher preoperative APRI have significantly increased risks of postoperative complications, such as infections, pneumonia and transfusions.^{18,19} These findings in elective arthroplasty cohorts support the plausibility that APRI could be relevant in hip fracture patients. Elderly femoral fracture patients often have multiple comorbidities such as subclinical liver impairment and chronic inflammation, so an elevated APRI may similarly flag those with diminished physiologic reserve who are prone to fractures and adverse outcomes. This provides a biological rationale that systemic inflammation and hepatic dysfunction, captured by APRI, might contribute to complications in fragile hip fracture populations.

Unlike previous studies in other populations (eg elective joint arthroplasty cohorts),¹⁹ no prior research has evaluated APRI's prognostic role in acute hip fracture patients. To our knowledge, this investigation is the first to examine APRI in a femoral fracture cohort, making our findings novel for this high-risk patient population. In this retrospective cohort study, we evaluated the association between preoperative APRI and postoperative mortality in patients undergoing surgery for femoral fractures. Our primary objective was to determine whether APRI is an independent predictor of mortality after adjusting for other known risk factors. A secondary aim was to explore an optimal APRI cutoff value for risk stratification and to compare outcomes between patients with elevated versus normal APRI.

Methods

Study Design and Setting

We conducted a retrospective cohort study at a tertiary care academic medical center. This retrospective cohort study was conducted in compliance with the regulations of the Chang Gung Memorial Hospital Institutional Review Board (IRB number 202401700B0). After Institutional Review Board approval (with waiver of informed consent), we identified all adult trauma patients (age ≥ 20) who sustained femoral fractures between January 2009 and December 2023 from the registered data from the Trauma Registry System of the hospital. Femoral fracture was defined as any fracture of the proximal femur (including intertrochanteric, femoral neck, or femoral head), shaft, and distal femur. Patients with pathological fractures (eg, metastatic disease) were excluded to maintain a homogeneous cohort of acute trauma cases. Those trauma patients by burn, hanging, drowning, and those patients without completed registered data were excluded.

Data Collection and Outcome Measures

Demographic information including age, sex, comorbid conditions, injury severity, and laboratory data from medical records. Laboratory data were collected based on the data at the arrival to the emergency room. APRI was calculated for each patient using the formula: $APRI = AST (U/L) \times 100/\text{platelet count } (10^9/L)$. In outcome assessment, the primary outcome was in-hospital mortality. In-hospital deaths were identified from hospital records. Secondary outcome is the length of stay in hospital.

Statistical Analysis

We first performed descriptive analyses of the cohort stratified by survival status. The homogeneity of variances is tested with the Levene's test. Continuous variables were compared using the Student's *t*-test or Mann–Whitney *U*-test as appropriate for distribution, and categorical variables were compared with Chi-square or Fisher's exact tests. All statistical tests were two-sided with a significance level set at $p < 0.05$. Analyses were conducted using SPSS (v26, IBM Corp).

To identify independent predictors of mortality, we employed multivariable logistic regression for in-hospital mortality. The model's covariates included APRI, particular comorbidities, and injury severity, all of which showed a significant difference between mortality and survival individuals. We checked for multicollinearity among covariates and reported adjusted odds ratios (OR) with 95% confidence intervals.

Further, we performed a receiver operating characteristic (ROC) curve analysis to assess the performance of APRI in discriminating in-hospital mortality. The area under the ROC curve (AUC) was calculated. We determined the optimal APRI cutoff that maximized the Youden index (sensitivity + specificity – 1) for predicting mortality, and we report the

corresponding sensitivity and specificity for that threshold. The patients in the study cohort were divided into two groups based on the identified optimal APRI cutoff value to compare the first outcome (in-hospital mortality) and secondary outcome (length of hospital stay). Adjusted odd ratios of mortality was calculated after adjusting the baseline characters including gender, age, comorbidities, and injury severity. Kaplan–Meier survival curves were generated for patients with admission APRI ≥ 0.74 versus < 0.74 , and differences between groups were compared using the Log rank test.

Results

Patient Enrollment

Between 2009 and 2023, a total of 54,313 trauma patients were registered, of whom 47,922 were adults (age ≥ 20). After excluding patients with burn injuries ($n = 1,192$), hanging ($n = 19$), or drowning incidents ($n = 4$), those without femoral fractures ($n = 21,148$), and cases with missing laboratory data ($n = 21,249$), a final cohort of 4,310 patients with femoral fractures was included (Figure 1). The trauma mechanisms for these patients comprised road accidents ($n = 1,558$), falls from walking level (height < 1 m, $n = 2,481$), falls from heights of 1–6 m ($n = 170$), falls from heights > 6 m ($n = 27$), and being struck by or against an item ($n = 74$). The study population comprised 85 in-hospital deaths and 4,225 survivors.

Patient Characteristics and Mortality Outcomes

Baseline characteristics differed between survivors and non-survivors (Table 1). Age and sex distributions were similar in the two groups (mean age ~ 69 vs 66 years, $p = 0.182$; male 51.8% vs 42.7%, $p = 0.096$). Non-survivors had a significantly higher admission APRI (mean 1.4 ± 1.5 vs 0.7 ± 2.1 , $p = 0.008$), driven by elevated AST levels (mean 95.8 vs 47.6 IU/L, $p < 0.001$), while platelet counts did not differ. End-stage renal disease was more frequent among non-survivors (8.2% vs 3.8%, $p = 0.035$), whereas a history of stroke (CVA) was less common (2.4% vs 9.4%, $p = 0.027$). Injury severity was markedly higher in non-survivors: median ISS 13 (IQR 9–34) vs 9^(*), $p < 0.001$. Notably, 41% of non-survivors had ISS ≥ 25 , compared to only $\sim 5\%$ of survivors. Neurologic status on admission was worse in non-survivors, with 35% presenting in severe coma (GCS 3–8) versus 1.9% of survivors. Consistent with more severe injuries, non-survivors had longer hospital stays on average (16.6 ± 21.2 vs 10.6 ± 10.4 days, $p < 0.001$).

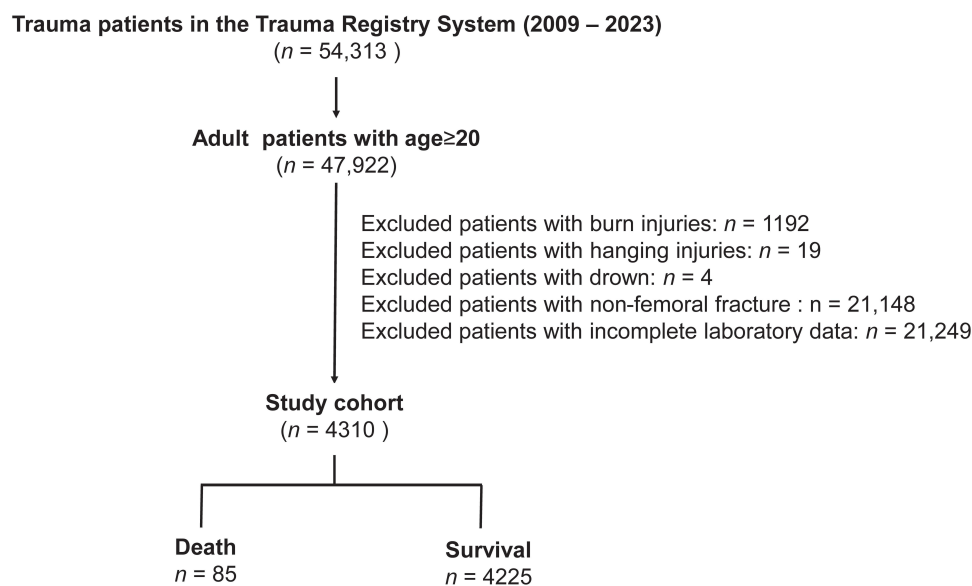


Figure 1 Flowchart illustrating patient inclusion and exclusion criteria from trauma registry, resulting in 4,310 femoral fracture cases analyzed.

Table 1 Comparison of Baseline Characteristics, Comorbidities, and Injury Severity Between Survivors and Non-Survivors with Femoral Fractures

Variables	Death n = 85	Survival n = 4,225	OR (95% CI)	P
Gender				0.096
Male, n (%)	44(51.8)	1806(42.7)	1.44(0.94–2.21)	
Female, n (%)	41(48.2)	2419(57.3)	0.70(0.45–1.07)	
Age, years	69.3±18.9	66.3±20.0	-	0.182
APRI	1.4±1.5	0.7±2.1	-	0.008
GOT (IU/L)	95.8±123.1	47.6±69.6	-	<0.001
Platelets (10 ⁹ /L)	209.8±71.5	218.4±71.7	-	0.273
Comorbidities				
CVA, n (%)	2(2.4)	397(9.4)	0.23(0.06–0.95)	0.027
HTN, n (%)	45(52.9)	2052(48.6)	1.19(0.78–1.83)	0.424
CAD, n (%)	8(9.4)	299(7.1)	1.36(0.65–2.85)	0.407
CHF, n (%)	3(3.5)	75(1.8)	2.02(0.63–6.55)	0.230
DM, n (%)	27(31.8)	1094(25.9)	1.33(0.84–2.11)	0.222
ESRD, n (%)	7(8.2)	160(3.8)	2.28(1.04–5.02)	0.035
GCS, median (IQR)	15(7–15)	15(15–15)	-	<0.001
3-8, n (%)	30(35.3)	81(1.9)	27.91(17.00–45.84)	<0.001
9-12, n (%)	6(7.1)	133(3.1)	2.34(1.00–5.46)	0.043
13-15, n (%)	49(57.6)	4011(94.9)	0.07(0.05–0.11)	<0.011
ISS, median (IQR)	13(9–34)	9(9–9)	-	<0.001
1–15, n (%)	43(50.6)	3836(90.8)	0.10(0.07–0.16)	<0.001
16–24, n (%)	7(8.2)	181(4.3)	2.01(0.91–4.41)	0.077
≥25, n (%)	35(41.2)	208(4.9)	13.52(8.59–21.28)	<0.001
Hospital stay (days)	16.6±21.2	10.6±10.4	-	<0.001

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; CI, confidence interval; CVA, cerebral vascular accident; DM, diabetes mellitus; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; GOT, aspartate aminotransferase; HTN, hypertension; IQR, interquartile range; ISS, injury severity score; OR, odds ratio.

Multivariable Analysis

On multivariable logistic regression (Table 2), APRI emerged as an independent predictor of in-hospital mortality. Each unit increase in APRI was associated with a ~57% increase in the odds of death (adjusted odds ratio [aOR] 1.57, 95% confidence interval [CI] 1.41–1.98, $p = 0.023$). Other independent predictors of mortality included pre-existing end-stage renal disease (aOR 3.90, 95% CI 1.72–8.88, $p = 0.001$), lower Glasgow Coma Scale on admission (aOR 0.81 per point, 95% CI 0.75–0.87, $p < 0.001$), and higher injury severity score (aOR 1.07 per ISS point, 95% CI 1.04–1.10, $p < 0.001$). While a history of CVA appeared protective in univariate analysis, it was not statistically significant after adjustment (aOR 0.35, $p = 0.144$).

Table 2 Multivariable Regression to Identify Independent Mortality Predictors

Variables	Univariate Analysis		Multivariate Analysis	
	ORCI	P	ORCI	P
APRI	1.37 (1.38–1.93)	0.008	1.57(1.41–1.98)	0.023
CVA	0.23 (0.06–0.95)	0.027	0.35 (0.08–1.44)	0.144
ESRD	2.28 (1.04–5.02)	0.035	3.90 (1.72–8.88)	0.001
GCS	0.72 (0.68–0.75)	<0.001	0.81 (0.75–0.87)	<0.001
ISS	1.12 (1.10–1.14)	<0.001	1.07 (1.04–1.10)	<0.001

Abbreviations: CI, confidence interval; CVA, cerebral vascular accident; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; ISS, injury severity score; OR, odds ratio.

Discriminative Ability of APRI

APRI showed modest discriminative power for predicting mortality. The receiver operating characteristic curve for APRI had an area under the curve (AUC) of 0.670, indicating fair accuracy. Using the optimal cutoff value of 0.74 (determined by Youden's index), the sensitivity was 55% and specificity 78% for in-hospital mortality prediction. In practical terms, $APRI \geq 0.74$ correctly identified 47 of 85 patients who died (sensitivity 55%), while $APRI < 0.74$ correctly identified 3,314 of 4,225 survivors (specificity 78%). These data suggest APRI has a significant but moderate ability to distinguish survivors from non-survivors (Figure 2).

Comparison by APRI Level

When stratified by APRI level, patients with high APRI (≥ 0.74) differed notably from those with low APRI (Table 3). The high-APRI group was younger (mean 56.5 ± 22.0 vs 69.2 ± 18.5 years) and more often male (53.2% vs 40.0%; both $p < 0.001$). They also had significantly fewer comorbidities – for example, lower rates of hypertension (34.8% vs 52.6%) and diabetes (15.9% vs 28.9%) – consistent with a generally healthier baseline. However, trauma severity was higher in the high-APRI cohort: 8.6% sustained severe head injury (GCS 3–8) compared to $<1\%$ of low-APRI patients, and 17.2% had ISS ≥ 25 (vs 2.3% in the low-APRI group). Accordingly, high-APRI patients required longer hospital stays (mean 15.3 ± 14.1 vs 9.4 ± 9.1 days, $p < 0.001$). In-hospital mortality in the high-APRI group was over four-fold higher than in the low-APRI group (4.9% vs 1.1%, OR 4.50, $p < 0.001$). Even after adjusting for age, sex, comorbidities, GCS, and ISS, an elevated APRI (≥ 0.74) remained associated with significantly higher mortality risk (adjusted OR 2.82, 95% CI 1.64–4.85, $p < 0.001$). As shown in Figure 3, patients with $APRI \geq 0.74$ had significantly lower survival probabilities than those with $APRI < 0.74$, as illustrated by early and sustained divergence of the Kaplan–Meier curves (log-rank $p < 0.001$). For the first time, our results demonstrate that an elevated APRI is significantly associated with worse outcomes in hip fracture patients – an association not previously reported in this patient cohort. This underscores the prognostic value of APRI in identifying trauma patients with femoral fractures at increased risk of poor outcomes.

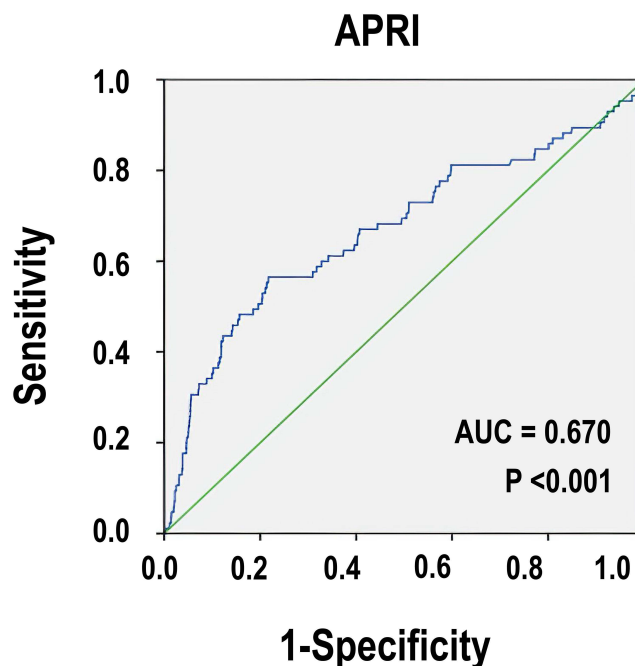


Figure 2 Receiver operating characteristic (ROC) curve analysis showing APRI's predictive accuracy for mortality. AUC = 0.670 with an optimal cutoff at 0.74.

Table 3 Comparison of Demographics, Comorbidities, Trauma Severity, and Outcomes Between Those Patients with High and Low Levels of APRI (≥ 0.74 Vs < 0.74)

Variables	APRI			P
	≥ 0.74 n = 958	< 0.74 n = 3,352	OR (95% CI)	
Gender				<0.001
Male, n (%)	510(53.2)	1340(40.0)	1.71(1.48–1.98)	
Female, n (%)	448(46.8)	2012(60.0)	0.59(0.51–0.68)	
Age, years (SD)	56.5 \pm 22.0	69.2 \pm 18.5	-	<0.001
Comorbidities				
CVA, n (%)	49(5.1)	350(10.4)	0.46(0.34–0.63)	<0.001
HTN, n (%)	333(34.8)	1764(52.6)	0.48(0.41–0.56)	<0.001
CAD, n (%)	49(5.1)	258(7.7)	0.65(0.47–0.89)	0.006
CHF, n (%)	8(0.8)	70(2.1)	0.40(0.19–0.82)	0.010
DM, n (%)	152(15.9)	969(28.9)	0.46(0.38–0.56)	<0.001
ESRD, n (%)	28(2.9)	139(4.1)	0.70(0.46–1.05)	0.083
GCS, median (IQR)	15(15–15)	15(15–15)	-	<0.001
3–8, n (%)	82(8.6)	29(0.9)	10.73(6.98–16.49)	<0.001
9–12, n (%)	55(5.7)	84(2.5)	2.37(1.67–2.36)	<0.001
13–15, n (%)	821(85.7)	3239(96.6)	0.21(0.16–0.27)	<0.001
ISS, median (IQR)	9(9–18)	9(9–9)	-	<0.001
1–15, n (%)	683(71.3)	3196(95.3)	0.12(0.10–0.15)	<0.001
16–24, n (%)	110(11.5)	78(2.3)	5.45(4.03–7.35)	<0.001
≥ 25 , n (%)	165(17.2)	78(2.3)	8.73(6.60–11.56)	<0.001
Hospital stay (day)	15.3 \pm 14.1	9.4 \pm 9.1	-	<0.001
Mortality, n (%)	47(4.9)	38(1.1)	4.50(2.92–6.94)	<0.001
Mortality AOR*	-	-	2.82(1.64–4.85)	<0.001

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; CI, confidence interval; CVA, cerebral vascular accident; DM, diabetes mellitus; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; HTN, hypertension; IQR, interquartile range; ISS, injury severity score; OR, odds ratio. AOR*, adjusted by Gender, age, CVA, HTN, CAD, CHF, DM, GCS, and ISS.

Discussion

In this retrospective cohort of patients with femoral fractures, we found that the APRI is a significant independent predictor of mortality. Patients with elevated APRI values on admission had substantially higher odds of dying during the hospitalization, even after accounting for age, comorbidities, and injury severity. Our findings suggest that APRI may capture an element of patient frailty or physiologic compromise and demonstrate the prognostic value of APRI in the trauma patients with femoral fracture.

The association between high APRI and mortality in femoral fracture patients aligns with emerging evidence from other clinical contexts. APRI was initially introduced as a surrogate marker for liver fibrosis, but it has since been linked with outcomes beyond liver disease. For instance, McLellan et al¹⁹ analyzed 104,633 total hip arthroplasty patients from the NSQIP database from 2007 to 2020 and found that abnormal APRI was independently associated with higher rates of major and minor complications, bleeding requiring transfusion, readmission, and non-home discharge following total hip arthroplasty. Although their primary focus was not mortality, the implication is that even mild liver dysfunction or elevated liver enzymes can adversely affect surgical outcomes. Our study extends these observations to the trauma population, indicating that the physiologic state captured by APRI is relevant in the acute femoral fracture setting as well.

Notably, APRI's prognostic role appears to differ between trauma patients and elective arthroplasty patients due to the acute trauma response. In hip fractures, injury severity can acutely influence APRI: AST levels rise from skeletal muscle damage and shock-related hepatic injury, while platelet counts often fall from consumption and dilution in trauma-induced coagulopathy.²⁰ Consistently, the AST/ALT (De Ritis) ratio correlates with trauma severity, and an early post-

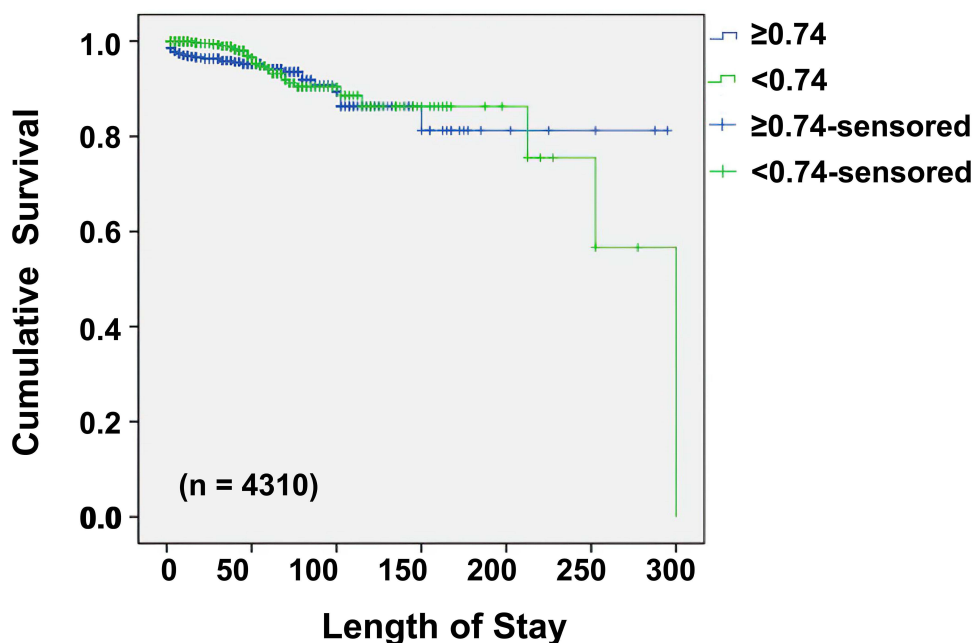


Figure 3 The Kaplan–Meier curves comparison between those patients with APRI ≥ 0.74 vs those with APRI < 0.74 .

trauma drop in platelets is linked to higher mortality.^{21,22} Thus, an elevated APRI on admission in a trauma setting may partly reflect the acute inflammatory/coagulopathic response rather than solely underlying liver dysfunction. In contrast, elective orthopedic patients manifest high APRI primarily due to chronic liver disease or systemic inflammation, which directly predisposes them to complications. Indeed, in elective joint surgery, preoperative APRI-defined fibrosis or cirrhosis has been associated with increased postoperative complications and poorer outcomes.^{18,19} In trauma patients, the predictive power of APRI may be blunted by confounding injury effects. This mechanistic difference explains why APRI showed only modest prognostic utility in trauma contexts²³ but is a significant risk marker in elective orthopedic cohorts. These distinctions underscore the importance of considering the trauma-induced physiological changes when interpreting APRI in hip fracture patients.

It is notable that we observed an APRI threshold (0.74) above which mortality risk rose markedly. This threshold is consistent with definitions of significant hepatic fibrosis in the hepatology literature,¹⁰ and it appears to have prognostic significance in our cohort as well. Even patients without known liver disease might have an elevated APRI due to subclinical liver steatosis, chronic alcohol use, or other systemic conditions. An elevated AST level in trauma patients could reflect a combination of factors: direct liver injury (eg, due to heart failure, medications, or alcohol), muscle breakdown from trauma, or global tissue ischemia in shock states,^{24–28} and a low platelet count might indicate chronic liver disease (due to hypersplenism or reduced thrombopoietin production) or acute consumptive processes (sepsis, disseminated intravascular coagulation).^{29–32} Therefore, a high APRI may signal a patient who either has chronic organ impairment or is manifesting an intense acute phase response. Both scenarios align with increased vulnerability. Thus, a high APRI may also be deemed as a composite marker of diminished physiological reserve. In elderly femoral fracture patients, such diminished reserve likely contributes to an inability to recover from the stress of surgery and injury.³³ In line with this reasoning, a related study found that a high AST/ALT ratio (another liver-related index) was independently predictive of in-hospital mortality in femoral fracture patients.³⁴ Both AST/ALT ratio and APRI leverage liver enzyme levels, supporting the concept that liver-related biomarkers reflect systemic frailty or inflammation influencing outcomes after fracture.^{34,35}

Importantly, APRI remained predictive in our multivariable model alongside established risk factors. Consistent with prior literature, we confirmed that advanced age and a higher burden of comorbidities significantly increase mortality risk after femoral fracture. These findings mirror the basis of clinical risk scores like the Nottingham Hip Fracture Score,

which emphasize age and comorbidity in mortality prediction.^{36,37} However, those conventional models do not include laboratory indices. Our results suggest that incorporating an objective lab marker such as APRI could further refine risk stratification. Because APRI is inexpensive and routinely obtainable, it could easily augment existing risk assessment protocols. For example, an elderly patient with a seemingly moderate risk profile based on age and comorbidities might be reclassified as high-risk if they present with an elevated APRI, alerting clinicians to potential occult issues like liver dysfunction or severe inflammation.

From a clinical standpoint, the ability to predict mortality risk early has practical implications. If a high APRI identifies a patient as high-risk, multidisciplinary interventions could be employed. These might include prompt management to mitigate ongoing catabolism or associated complications, intensive perioperative monitoring, and aggressive management of medical issues, and discussion of goals of care with patients and families. In settings where resources allow, involving a hepatologist or geriatrician in preoperative optimization might be beneficial for patients flagged by a high APRI. Additionally, APRI could be considered for inclusion in future risk prediction models or scoring systems for femoral fracture.

Limitations

We acknowledge several limitations in our study. First, the retrospective design makes it susceptible to unmeasured confounding and limits our ability to infer causality. We attempted to adjust for key confounders, but there may be other factors (eg, nutritional status, frailty scores, prior medicine drug) that correlate with both APRI and mortality, which we did not capture. Second, our study was conducted at a single center, and the relatively low number of occurrences may undermine the statistical analysis's power, which was conducted without controlling surgical procedures. Thus, the findings may not generalize to all settings. Third, while we focused on mortality, we did not extensively analyze other complications or long-term mortality in this report. It remains possible that APRI is actually a stronger predictor of specific postoperative complications, which in turn contribute to mortality. However, our analysis did not investigate the connection of APRI and complications like sepsis and pneumonia. Fourth, APRI can be influenced by acute muscle injury, a condition that may be frequently encountered in trauma patients—in theory, a patient with a very high AST due to muscle trauma could have a high APRI not truly reflecting liver function. We attempted to mitigate this by using the laboratory data from patients visits to the emergency room. Furthermore, the indication for resuscitation management and surgery may vary among different attending physicians or surgeons, and we can only assume the intervention outcome is eventful among different care staff. Finally, the mortality benefit of any interventions guided by APRI remains unproven. Whether modifying management based on a high APRI (eg, delaying surgery for optimization or providing more intensive postoperative care) actually improves outcomes would require prospective investigation.

Conclusion

In summary, our study demonstrates that the APRI is an independent predictor of mortality in patients undergoing surgery for femoral fractures. An elevated APRI identifies patients at significantly higher risk of postoperative death. This suggests that subclinical hepatic dysfunction or related systemic factors captured by APRI play a meaningful role in recovery after femoral fracture. APRI is an inexpensive and readily available metric that could be easily obtained upon hospital admission for a fracture patient. Incorporating APRI into risk assessments may improve the early identification of high-risk patients, enabling tailored management strategies to potentially reduce mortality. Future prospective studies and clinical trials should evaluate whether interventions guided by APRI can improve outcomes in this population. Ultimately, the inclusion of biomarkers like APRI might provide a better risk stratification tool to support more informed clinical decision-making and allocation of resources, with the goal of improving survival of trauma patients with femoral fractures.

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Disclosure

The authors report no conflicts of interest in this work.

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