

Risk Factors for AVN After Talar Fractures

Maxwell C Alley MD; Paul Tornetta MD

Boston University Medical Center, Boston, MA, United States

Purpose: Our purpose was to identify the patient, injury, and treatment factors associated with development of AVN (avascular necrosis) following talar fractures. In particular we were interested in whether any modifiable factors were present.

Methods: We retrospectively reviewed consecutive talar neck and/or body fractures treated surgically at 20 institutions from 2008 to 2019. Demographics, social, comorbidity, injury, treatment (surgical approach and timing), and follow-up data were collected. Radiographs and clinic notes were reviewed to identify cases of AVN. AVN at 1 year post-injury was the primary outcome; infection, nonunion, and malunion were secondary outcomes. We performed both univariate and regression analysis to identify those factors associated with AVN. We separately evaluated the timing of reduction after injury.

Results: 1237 talus fractures were reviewed. We included only those with ≥ 1 year follow-up and excluded those without information about AVN, and those with minor fractures. This left 494 patients (254 M; 240 F; age 18-93 years, mean 37.3) with 499 (335 R; 164 L) fractures. These were classified as Hawkins grade I (55), IIa (47), IIb (59), III (82), IV (38), neck + body (90), and body (128). 105/499 developed AVN (21%). Univariate analysis showed that more severe Hawkins type ($P < 0.001$), neck + body ($P = 0.01$), BMI (body mass index) ($P = 0.002$), smoking ($P = 0.017$), MVA (motor vehicle accident) ($P < 0.0001$), right-sided fractures ($P = 0.045$), open fracture ($P = 0.005$), and combined medial and lateral approaches ($P = 0.018$) were associated with AVN. After multivariate regression, only fracture type (odds ratio [OR] = 2.7; $P = 0.013$), smoking (OR = 1.7; $P = 0.034$), open fractures (OR = 1.7; $P = 0.05$), and BMI (OR = 1.04; $P = 0.004$) remained significant. Talar body fractures in addition to neck fractures increased the risk of AVN in types I to IIb, but not in type III and IV injuries. Excluding late cases (> 7 days), the time to joint reduction for type IIb to IV injuries was not different between those who developed AVN (22.9 ± 31 hours) and those who did not (26.4 ± 34 hours); $P = 0.47$. The AVN rates for reduction < 6 hours vs > 6 hours were 26.4% and 25.3% ($P = 0.85$). Complications included 22 infections (4%), 29 nonunions (5.8%), and 10 malunions (2%). Infection was more common in open fractures (13.9% vs 4.8%, $P = 0.001$).

Conclusion: The rate of AVN after talar fractures was 21%, with worse injuries (worse fractures and open injuries) having higher rates of AVN. No surgeon-controlled factor including the timing of the reduction or surgical approach was associated with AVN. In type I to IIb injuries the addition of a talar body fracture increased the rate of AVN, but this was not true in type III or IV injuries. AVN is more related to the injury than the treatment.