Predicting the Presence of Compressive Conditions After Distal Radius Fractures

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Introduction: Distal radius fractures are among the most common orthopedic injuries. Despite adequate fracture management, some patients experience delayed onset of compressive conditions, increasing the overall treatment burden. Identifying risk factors for these complications is important to improve patient outcomes. We sought to identify independent preoperative factors associated with the delayed development of compressive conditions following distal radius fracture management.
Methods: A retrospective review was conducted of patients with distal radius fractures over a decade. Data collected included demographic information, fracture characteristics, treatment modality (operative vs. nonoperative), and the subsequent incidence of compressive pathologies for 1 year after injury. Multivariate regression was performed to isolate the most predictive factors.
Results: Of 462 patients, 15.4% developed a compressive condition, with carpal tunnel syndrome being the most common (11%). Significant predictors for delayed development of compressive condition were diabetes (odds ratio [OR], 3.34) and increasing age (OR, 1.02). Complex fractures and operative treatment of fractures predicted treatment for a compressive condition, whereas increasing age was the only factor associated with requiring a secondary surgery (OR, 0.92). Preoperative diagnoses were not found to be a significant risk factor.
Conclusion: Diabetes and increasing age were the primary predictors for development of delayed onset of compressive neuropathy or tendinopathy following distal radius fracture treatment, with age being the only factor associated with increased need for surgical intervention. Although operative treatment of distal radius fracture was not correlated with development of delayed compressive conditions, it was associated with an increased need for subsequent interventions.
Level of Evidence: III.

Key Words: distal radius fracture, nerve compression, tendon compression

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Distal radius fractures are among the most common fractures to occur in nearly all age groups.[1,2] Management of distal radius fractures can include both operative or nonoperative treatments, typically based on both patient characteristics and fracture patterns.[3] Although distal radius fractures are sometimes associated with acute injury to neurovascular or tendinous structures, both surgical and nonsurgical management can be associated with a sequela of delayed-onset compressive conditions, including carpal tunnel syndrome,[4] mechanical flexor tenosynovitis,[5] or de Quervain's tenosynovitis.[6] These complications impair hand function, delay recovery, and potentially necessitate further intervention, increasing overall treatment burden.

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Questions regarding potential risk factors that may impact the risk of developing compressive conditions after the completion of treatment for the fracture remain. Factors such as age, bone quality, fracture type, socioeconomic status, and medical comorbidities all may play a role.[4–6] Several studies have investigated the impact of operative versus closed management, especially regarding the development of carpal tunnel syndrome.[4,7,8] Although operative intervention has been shown to correlate with increased rates of carpal tunnel syndrome, it is unclear if this is from the fracture itself or from secondary edema and inflammation caused by operative dissection and placement of volar implants. Furthermore, few studies exist describing the effects of distal radius fractures on other inflammatory and compressive pathologies, such as de Quervain's tenosynovitis and flexor tenosynovitis, as the relevant tendons are situated in the immediate vicinity of the fracture site. These tendons are often visualized during the volar approach to the distal radius, especially upon release of the brachioradialis tendon.

To answer these questions, we sought to perform a review of patients who sustained distal radius fractures, treated at our institution, to determine which preoperative factors might be independent predictors of the delayed development of compressive neuropathies and tendinopathies. Our primary outcome measure was rate of compressive conditions (defined as carpal tunnel, stenosing flexor tenosynovitis, and/or de Quervain's tenosynovitis) associated with operative versus nonoperative management of distal radius fractures. Secondary outcomes were defined as the rates of both surgical and nonsurgical treatments for these conditions. We hypothesized that there would be a correlation between increasing fracture complexity and development of compressive conditions, as well as patient factors that "predispose" to these conditions, such as diabetes. We intended to determine specific preoperative factors to allow for better counseling of patients with distal radius fractures regarding their anticipated posttreatment course specific to their risk profile.

METHODS

This Study Was Approved by Our Institutional Review Board

Patient Selection and Preoperative Data

A retrospective review was performed of all patients diagnosed with distal radius fracture over a 10-year period, from 2013 to 2022, at our institution. Inclusion criteria included age greater than 18 years, with at least a 1-year follow-up after either operative or nonoperative management for distal radius fracture. Detailed demographic, clinical, and procedural data were collected, including fracture pattern, age, occupation, and history of compressive neuropathy or tendinopathy. Fracture pattern classification was done by the lead author. Patients with prior distal radius or multiple fractures of the relevant arm, devastating soft tissue injury requiring tendon, nerve, vessel, or flap-based reconstruction, or fracture fixation using a dorsal approach were excluded. Additionally, patients with incomplete or missing critical data, or those outside predefined criteria, were excluded from the analysis. As we were focused on delayed development of compressive conditions,

TABLE 1. Demographics

Characteristic	Value (% or Average ± Standard Deviation)
Age, y	53.5 ± 20.8
Fracture class	
AO A	3.7
AO B	19.7
AO C	76.6
Associated ulnar fracture	40.5
Intra-articular fracture	70.8
Number of parts	
1	45.7
2	25.5
3	26.0
4 or more	2.8
Diabetes	15.4
Previous compressive disorder	2.2
Operative intervention	63.2

patients with acute carpal tunnel syndrome associated with distal radius fracture were also excluded. Patients who were treated for a compressive condition before fracture treatment was initiated (defined as either surgery or casting) were excluded.

Fracture Care

Patients chose either a nonoperative or operative course based on fracture pattern, patient goals, and desire for operative intervention. The recommendation for operative intervention was made by the senior surgeon based on standard parameters. One of 2 fellowship-trained surgeons performed the operation through the standard modified Henry approach. The brachioradialis was released in all patients.

Postoperative Data Collection

Charts were reviewed for up to 1 year postoperatively to monitor the development of compressive pathologies, including carpal tunnel symptoms, trigger fingers, or de Quervain tenosynovitis, in a delayed fashion (defined as presenting after initial immobilization of the fracture, 6 weeks for closed and 2 weeks for open, was complete). Any required treatments, such as steroid injections or surgical release, were also documented.

Statistical Analysis

All statistical analyses were done on SPSS (IBM, Armonk, NY). ANOVA was performed for continuous variables; chi-squared tests were performed for discrete variables. Multivariable analysis was done to identify individual predictors of compressions. A P value cutoff of 0.05 was used to define statistical significance.

RESULTS

Patient Demographics

A total of 462 patients met the inclusion criteria and were included in the final analysis, with a mean age of 53.5 ± 20.8 years. Of these, 17 patients (3.7%) had a simple fracture pattern (AO Class A), 91 patients (19.7%) had a moderate fracture (AO Class B), and 354 (76.6%) had a complex fracture (AO Class C). Less than half (40.5%) had an associated ulnar styloid fracture. The majority had intra-articular fracture extension, with the most common being a 1-part (211 patients, 45.7%), 2-part (118 patients, 25.5%), or 3-part (120 patients, 26%) fracture. Of these patients, 292 were treated with surgical fixation (63.2%). Diabetes was present in 71 patients (15.4%), and 10 patients (2.2%) had a diagnosis of a compressive condition by a hand surgeon prior to injury (Table 1).

Among the 462 patients who met the inclusion criteria, only 71 developed a delayed compressive condition (15.4%), with 51 developing carpal tunnel syndrome (11%), 8 developing de Quervain tenosynovitis (1.7%), and 43 developing trigger fingers (9.3%), of which 35 cases involved multiple digits concurrently. The most common fingers involved were the index finger (31 patients, 72%) and the middle finger (30 patients, 70%). Of these patients diagnosed with a compressive condition, 56 patients had severe-enough symptoms to require nonoperative management (78.9%), of which 39 patients (70%) required operative intervention (Table 2).

The rate of compressive conditions in operative and nonoperative patients was not significantly different for all compressions (15.3% vs15.4%, $P > 0.05$), carpal tunnel syndrome (11.1% vs 10.9%, $P > 0.05$), or trigger fingers (8.8% vs 9.6%, $P > 0.05$). However, the percentage of patients who underwent further nonoperative treatment for a compressive condition was higher if prior operative intervention was pursued (89% vs 62%, $P < 0.01$), although the rate of patients progressing to surgical intervention for compressive issues was equivalent regardless of the intervention for the distal radius fracture. These data are summarized in Table 3.

Predictors of Compression

Given the correlation between fractures and compressive conditions, multivariate regression was performed to isolate the factors most predictive of developing a delayed compressive condition. Of the factors studied, diabetes mellitus (odds ratio [OR], 3.34; $P < 0.01$) and increased age (OR, 1.02; $P = 0.01$) were the only significant risk factors

TABLE 2. Compressive Disorders

Disorder	%
Any disorder	15.4
Multiple disorders	
0	84.6
1	9.5
2	5.0
3	0.9
Carpal tunnel syndrome	11.0
De Quervain syndrome	1.7
Trigger fingers	
0	90.7
1	2.2
2	1.9
3	2.2
4	1.3
5	1.7
Multiple	7.6
Thumb	4.1
Index	6.7
Middle	6.5
Ring	5.6
Small	3.5
Needing treatment	12.1
Needing surgery	8.4

TABLE 3. Diagnoses and Interventions Based on Mode of Fracture Management

Characteristic	% of Operative Patients	% of Nonoperative Patients	P
Any compression	15.3	15.4	>0.05
Carpal tunnel	11.1	10.9	>0.05
Trigger finger	9.6	8.8	>0.05
Treatment needed	89.0	62.0	<0.01
Surgery needed	44.0	56.0	>0.05

predictive of delayed compressive condition. In terms of predictors for compressive conditions requiring intervention, more complex fractures required treatment for compressive conditions (OR, 12.5; P = 0.04), whereas need for operation for a distal radius fracture correlated with more interventions for compressions (OR, 12.7; P < 0.01). The only factor predicting the need for surgical intervention, however, was the patient age (OR, 0.92; P = 0.01). Importantly, preoperative diagnoses by a hand surgeon of these conditions were not predictive of postoperative development or treatment. These data are summarized in Table 4.

Carpal Tunnel Syndrome

Among CTS patients, only diabetes was predictive of a CTS diagnosis (OR, 3.2; P < 0.01). The only factor associated with the need for treatment was operative intervention for a distal radius fracture (OR, 8.6; P = 0.02). No factors were identified as predictors for those requiring surgical intervention for carpal tunnel. Preoperative diagnoses were also not found to be significant predictors. These data are summarized in Tables 4 and 5.

Trigger Fingers

Similarly, subgroup analysis was performed on those diagnosed with trigger finger. Predictors for the diagnosis of trigger finger included diabetes (OR, 2.5; P = 0.03), age (OR, 1.0; P < 0.01), and multifragmentary fractures (OR, 1.5; P = 0.03). Predictors of multiple trigger fingers included age (OR, 1.0; P = 0.01) and diabetes (OR, 3.5; P < 0.01). Operative intervention of distal radius fracture predicted a need for further nonoperative treatment of trigger fingers (OR, 50.9; P < 0.01), as did age (OR, 1.35; P = 0.04). However, there were no predictors identified for surgical intervention for trigger fingers. Preoperative diagnoses were again not found to be statistically significant predictors. These data are summarized in Tables 4 and 6.

de Quervain Tenosynovitis

Given the low frequency of de Quervain tenosynovitis, it was not included for subgroup analysis.

DISCUSSION

Distal radius fractures remain a common pathology seen by hand surgeons. Therefore, providing thorough counseling on the management of these fractures, particularly regarding their potential risk profile for delayed development of compression syndromes, is essential to ensure that patients have accurate information to make an informed decision regarding surgical versus nonoperative management.

Despite adequate treatment of distal radius fracture, development of a compressive syndrome (trigger finger, carpal tunnel syndrome, or de Quervain tenosynovitis) is not uncommon.[4–6] This may be due to intraoperative irritation of the flexor tendons, median nerve, and/or first dorsal compartment from surgical dissection and resultant edema, as they all exist near the distal radius fracture.

Based on the above data, we found that the primary patient factors predicting the development of a compressive condition after a distal radius fracture were patient age and a history of diabetes. Importantly, neither a prior diagnosis of a compressive condition nor undergoing operative treatment for distal radius fracture was correlated with the future development of a compressive condition. For carpal tunnel syndrome, diabetes alone was a predictor, whereas for trigger fingers, multifragmentary fractures were also a predictive factor. Complex fractures and operative

TABLE 4. Regression for Total Data Set

Factor	Compression OR	Compression P	Carpal Tunnel OR	Carpal Tunnel P	Trigger Finger OR	Trigger Finger P	Multiple Triggers OR	Multiple Triggers P	Treatment OR	Treatment P	Surgery OR	Surgery P
Age	**1.02** **1.00–1.04**	**0.01**	1.01 0.99–1.02	0.40	**1.04** **1.01–1.06**	**<0.01**	**1.03** **1.01–1.05**	**0.01**	0.99 0.95–1.02	0.49	**0.92** **0.87–0.98**	**0.01**
Dominant hand	0.25 0.14–1.68	0.25	0.99 0.23–4.33	0.99	0 0–0	1.00	9 9–9	1.00	0.29 0–	1.00	490,000 0–	1.00
Work Intensity	1.08 0.62–1.86	0.79	1.34 0.73–2.48	0.35	0.89 0.44–1.79	0.74	1.12 0.52–2.42	0.78	1.45 0.34–6.12	0.61	0.70 0.11–4.44	0.71
Pattern AO	1.25 0.68–2.28	0.48	1.31 0.67–2.58	0.43	0.84 0.42–1.70	0.63	0.90 0.42–1.93	0.78	**12.5** **1.6–101**	**0.04**	2.04 0.50–8.22	0.32
Ulnar fracture	1.35 0.79–2.31	0.28	1.41 0.77–2.58	0.26	1.28 0.66–2.49	0.47	1.12 0.54–2.33	0.77	1.54 0.36–6.54	0.56	0.69 0.15–3.25	0.64
Intra-articular	1.27 0.65–2.47	0.48	1.08 0.52–2.26	0.84	1.07 0.48–2.37	0.88	0.77 0.34–1.76	0.54	1.00 0.17–5.9	1.00	1.33 0.20–8.94	1.33
No. parts	1.30 0.98–1.74	0.07	1.13 0.80–1.59	0.48	**1.48** **1.05–2.10**	**0.03**	1.34 0.91–1.97	1.34	1.04 0.45–2.39	0.93	0.43 0.15–1.18	0.10
Diabetes	**3.34** **1.63–6.84**	**<0.01**	**3.19** **1.45–7.03**	**<0.01**	**2.49** **1.07–5.81**	**0.03**	**3.47** **1.46–8.26**	**<0.01**	4.82 0.65–36.0	0.13	1.03 0.19–5.64	0.98
ORIF	0.94 0.52–1.70	0.84	0.97 0.50–1.87	0.92	1.16 0.55–2.46	0.70	1.34 −.58–3.06	0.49	**12.7** **2.6–62.8**	**<0.01**	0.47 0.08–2.83	0.41
Preoperative diagnosis	1.96 0.56–6.86	0.29	2.51 0.57–11.0	0.22	9400 0–	1.00	7600 0–	1.00	0 0–	1.00	0 0–0	1.00

Significant values are in bold font.

OR, odds ratio with 95% confidence interval. *P*, *P* value.

TABLE 5. Regression for Carpal Tunnel Patients

	Treatment		Surgery	
Factor	**OR**	***P***	**OR**	***P***
Age	0.98 0.94–1.02	0.41	0.68 0–0	1.00
Dominant hand	0.29 0–0	1.00	0 0–0	1.00
Work intensity	0.86 0.18–4.20	0.85	0 0–0	1.00
Pattern AO	0.27 0.02–2.94	0.28	0 0–0	1.00
Ulnar fracture	0.82 0.14–4.67	0.82	0 0–0	1.00
Intra-articular	0.32 0.03–3.56	0.35	180 0–0	1.00
No. parts	1.21 0.46–3.20	0.70	0 0–0	1.00
Diabetes	10.8 0.78–150.3	0.08	0 0–0	1.00
ORIF	**8.64** **1.40–53.4**	**0.02**	0 0–0	1.00
Preoperative diagnosis	0 0–00	1.00	0 0–0	1.00

Significant values in bold font.

OR, odds ratio with 95% confidence interval; P, *P* value.

management of the fracture were found to correlate with compressive conditions that required treatment and surgical intervention, specifically.

These data do contradict some of the previously published literature, specifically regarding carpal tunnel syndrome. In a recent database study, the rate of carpal tunnel syndrome after a distal radius fracture was found to be 9.1% regardless of intervention, with nearly half requiring surgery.[4] These numbers are lower than those seen in our study, likely due to the nature of database studies relying on diagnosis codes, not symptomatology, and detailed chart review. Similarly, our analysis showed that operative repair of a distal radius fracture did not predict the development of posttreatment carpal tunnel syndrome. Those who did pursue surgery for their fracture did have a higher incidence of carpal tunnel symptoms necessitating intervention, suggesting that the severity of symptoms is higher in that population, potentially due to the increased inflammation and edema from surgery. This is similar to previous studies, which identified risk factors including multiple surgeries and multipart fractuers.[5,7–11]

However, our study is novel in several important ways. By providing details on fracture pattern, number of parts, and specifics of patient history unattainable from database sources, we showed that there was no correlation between fracture severity and incidence of delayed carpal tunnel symptoms. Previous studies have shown that open fractures and complex fractures are correlated with the development of acute carpal tunnel syndrome; however, acute carpal tunnel syndrome is a different pathological process from the delayed carpal tunnel, as we discussed herein.[8] Those with preoperative findings of carpal tunnel were treated at the time of presentation and excluded from this study. Further, we extended the time frame to 1 year after injury, which may account for the increase in overall incidence in this cohort. Also, the decision for surgery and surgical technique may be subject to less variance, as only 2 senior surgeons were included.

We also were able to describe the incidence of other compressive pathologies, specifically de Quervain tenosynovitis and trigger fingers. Notably, the frequency of de Quervain tenosynovitis was quite low—this is likely because the first dorsal compartment is rarely affected by closed management and typically exposed in our operative approach of a brachioradialis tenotomy with visualization of the tendons of the first compartment. Although not a formal release, this does completely open the floor of the compartment. Although it is difficult to say that this is preventative in regard to de Quervain tenosynovitis, our rates are much lower than those seen in previous studies, suggesting a potential role.[6] Identifying specific factors that lead to de Quervain is also difficult, as our small cohort prevents subgroup analysis.

Trigger fingers are much more common than de Quervain tenosynovitis after distal radius fractures. However, the thumb is not the most common digit to be affected. This is counterintuitive, as the flexor pollicis longus is necessarily manipulated during operative intervention of distal radius fracture via a volar approach. Multifragmentary fractures were predictive of trigger finger symptoms, along with a diagnosis of diabetes, whereas operative intervention of the fracture did result in trigger fingers that needed intervention.

Few studies exist on trigger fingers after a distal radius fracture in the hand surgery literature. In 2 previous population studies, the most predictive factor for the development of trigger finger after a distal radius fracture was the presence of a comorbidity such as diabetes with an OR of 6.5 in one study with a rate of 1%, much lower than our study[5]; factors such as surgical intervention and hypertension are not significant.[5,12] Again, these studies are limited as granular data on fracture pattern were not included, which does impact the potential development of the diagnosis.

Thus, we do demonstrate that there is a role for discussing the development of compressive pathology after distal radius fractures, with “red flag” factors being diagnosis of diabetes, older patients, and those with complex fracture patterns who are undergoing operative fixation. This does not mean that prophylactic release is mandated, as less than 10% of patients develop symptoms requiring surgery. Further, the role of prophylactic release remains a matter of debate. A recent review suggested no benefit of release; however, this conclusion is based on

TABLE 6. Regression for Trigger Finger Patients

	Treatment		Surgery	
Factor	**OR**	***P***	**OR**	***P***
Age	**1.35** **1.01–1.81**	**0.04**	0.95 0.87–1.03	0.18
Dominant hand	0 0–0	1.00	0.80 0.11–5.83	0.83
Work intensity	54.3 0.05–	0.26	0.25 0.02–3.09	0.28
Pattern AO	0.00 0–0	1.00	1.60 0.29–8.95	0.59
Ulnar fracture	0 0–0	1.00	0.97 0.11–8.39	0.98
Intra-articular	0 0–0	1.00	1.33 0.12–14.17	0.82
No. parts	220 0–0	0.17	0.24 0.05–1.08	0.06
Diabetes	0.08 0–67	0.46	1.15 0.13–9.90	0.90
ORIF	**50.9** **3.07–843**	**<0.01**	1.01 0.11–9.60	1.00
Preoperative diagnosis	0 0–0	1.00	0.57 0.01–28.3	0.78

Significant values in bold font.

OR, odds ratio with 95% confidence interval; P, *P* value.

persistent symptoms after release and the development of symptoms despite release in the surgical cohort without a matched fracture pattern and preoperative symptomatology in the no release segment.[13] Thus, the validity of this conclusion is debatable, and patients should be informed of the potential development of these conditions and counseled on the possible need for intervention.

Despite these findings, the true underlying mechanism for the development of these conditions is still unclear. Likely, complex fracture patterns correlate to more perifracture inflammation that affects the flexor tendons and median nerve overlying the distal radius, which in turn causes inflammation and edema and subsequent compression.[8,10,14] This may be amplified by surgical intervention, which does manipulate the flexor tendons and median nerve.

However, as described above, the rate of carpal tunnel symptoms does not seem to change whether operative intervention for the fracture is pursued or not, only the need to intervene on the carpal tunnel symptoms. In other words, distal radius fixation does not increase the chances of developing symptoms but may increase the severity of symptoms. Although the exclusion of acute carpal tunnel patients removes the highest energy fractures, this does not affect our outcomes, as the acute carpal tunnel cohort cannot be accurately compared to the nonoperative cohort.

Similarly, operative intervention on a fracture predicted the need for intervention for trigger fingers, but not the diagnosis. Again, fixation may not increase the chances of developing a trigger finger but may increase the severity. It is unclear why complex fractures were only predictive of severe symptoms in trigger fingers, not carpal tunnel syndrome. Perhaps, this is due to acute carpal tunnel syndrome patients not being included in the study. In general, diabetes and older age are intuitive risk factors in these patients, as they are major risk factors in several complications and are strongly associated with a variety of compressive pathologies, with diabetes providing a 2× higher rate of carpal tunnel diagnosis, diffuse trigger finger 1.5× more likely in diabetics, and patients with de Quervain having poor outcomes if diabetic.[15–18]

There are some important limitations to this study. First, there are well known risks of bias associated with retrospective review. Further, although we claim that carpal tunnel and trigger finger interventions were based on symptom severity, it is difficult to ascertain whether patients had not desired to pursue further surgery, despite the indication for it, as opposed to having self-limiting symptoms. Our cohort does have a high proportion of low-energy fractures, which may lower the rates of compressive conditions; however, this represents a more typical range of distal radius fractures rather than the high-energy trauma. Additionally, as this was a single-institution study, generalizability may be limited, especially as the diagnostic criteria of the participating surgeons may not be the same used by other surgeons. Also, we attempted to exclude patients with acute carpal tunnel symptoms to focus on the delayed development of these compressive conditions, but this may not have been completely accurate as certain acute conditions may have developed further delayed complications. Finally, subgroup analysis had small numbers, which may have contributed to the wide confidence intervals.

In conclusion, we found that the factors associated with distal radius fractures that result in delayed compressive pathology were primarily the diagnosis of diabetes and the age of the patient. Operative intervention for fractures and increasing complexity of fractures were associated with the need for subsequent nonoperative intervention, although multifragmentary fractures were only correlated with trigger fingers, not carpal tunnel syndrome. Based on these data, surgeons should observe for carpal tunnel and trigger finger symptoms after distal radius fracture, especially in older patients with diabetes. It is important to counsel patients that the need for surgical release after distal radius fixation is increased.

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