Success of scaphoid nonunion surgery is independent of proximal pole vascularity

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Abstract

We followed 35 consecutive patients with scaphoid nonunions in a prospective longitudinal registry. All nonunions were treated with curettage, non-vascularized autogenous grafting and headless screw fixation. Preoperative magnetic resonance imaging, intraoperative bleeding points and histopathological analysis of cancellous bone in the proximal pole were recorded as measures of viability. Healing was categorized as ≥50% bony bridging on computed tomographic images in the plane of the scaphoid. Nine of 23 proximal poles demonstrated ischaemia on magnetic resonance imaging but none were interpreted as infarcted. Twenty-eight of 33 were found to have impaired vascularity as assessed by intraoperative bleeding. Fourteen of 32 demonstrated ≥50% trabecular necrosis and four of 33 demonstrated ≥50% tissue necrosis on histopathological analysis. Thirty of 33 demonstrated focal or robust remodelling activity. Despite pathological evidence of impaired vascularity in over half of the patients, 33 of the 35 scaphoids had healed by 12 weeks. We conclude that proximal pole infarction is decidedly rare and that vascularized bone grafting is seldom required.

Level of evidence: IV

Keywords

Scaphoid nonunion, proximal pole, avascular necrosis, vascularity, perfusion, non-vascularized, vascularized, bone graft

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Introduction

When intraosseous blood flow from the distal to proximal pole of the scaphoid is disrupted after a fracture, the proximal pole is separated from its primary blood supply owing to the axial structure of the vascular network supplying the scaphoid (Büchler and Nagy, 1995; Russe, 1960) and is predisposed to ischaemia and avascular necrosis (AVN). Proximal pole vascularity is thought to correlate with likelihood of success when treating scaphoid nonunion and is therefore of considerable concern when planning operative treatment (Büchler and Nagy, 1995; Green, 1985; Russe, 1960; Trumble, 1990). Based on statements by Green (1985) and Russe (1960) that Russe bone grafting ‘will not be successful if the proximal pole is completely avascular’, many authors today believe that proximal pole AVN is a contraindication to non-vascularized surgery (Derby et al., 2013; Jones et al., 2012; Steinmann et al., 2002; Zaidemberg et al., 1991).

Previously reported criteria for assessing proximal pole vascularity include changes in radiographic density (Bervian et al., 2015; Büchler and Nagy, 1995; Green, 1985; Russe, 1960; Trumble, 1990). Based on statements by Green (1985) and Russe (1960) that Russe bone grafting ‘will not be successful if the proximal pole is completely avascular’, many authors today believe that proximal pole AVN is a contraindication to non-vascularized surgery (Derby et al., 2013; Jones et al., 2012; Steinmann et al., 2002; Zaidemberg et al., 1991).
Table 1. Vascularity terminology.

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tr>
<td>Ischaemia</td>
<td>Blood supply insufficient to support physiological function</td>
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<tr>
<td>Ischaemic</td>
<td>Pathological changes reflecting ischaemia</td>
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<tr>
<td>Necrosis/osteonecrosis/AVN</td>
<td>Bone death resulting from the absence of blood supply; can be focal</td>
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<tr>
<td>Infarction</td>
<td>Diffuse osteonecrosis with empty lacunae and granular degeneration of marrow fat</td>
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AVN: avascular necrosis.

1995; Perlik and Guilford, 1991; Sakuma et al., 1995), T1- and/or T2-weighted magnetic resonance imaging (MRI) signal intensity (Fox et al., 2010; Morgan et al., 1997; Perlik and Guilford, 1991; Sakuma et al., 1995; Trumble, 1990), magnetic resonance contrast enhancement (Megerle et al., 2011; Singh et al., 2004), non-quantitative and quantitative perfusion MRI (Donati et al., 2011; Ng et al., 2013) and intraoperative punctate bleeding (Bervian et al., 2015; Büchler and Nagy, 1995; Green, 1985; Perlik and Guilford, 1991; Trumble, 1990). However, studies have shown inconsistent results in establishing definitive relationships between the various diagnostic modalities for vascularity and outcomes of surgery for scaphoid nonunion (Fox et al., 2010; Günel et al., 1999; Morgan et al., 1997; Perlik and Guilford, 1991; Schmitt et al., 2011; Singh et al., 2004; Trumble, 1990).

The difficulty in establishing prognostic relationships may in part arise from imprecise definitions of AVN. Although Russe (1960), Green (1985) and others (Büchler and Nagy, 1995; Trumble, 1990) have commented that a ‘completely necrotic’ and ‘totally avascular’ proximal pole is unlikely to successfully heal, there is a lack of consensus on what defines AVN. This has resulted in disparity over the incidence and effect of AVN on healing (Büchler and Nagy, 1995; Green, 1985; Krimmer, 2002; Malizos et al., 2007; Slade et al., 2008; Steinmann and Adams, 2006). Consequently, there are wide differences in opinion on the necessity of vascularized versus non-vascularized bone grafting (Büchler and Nagy, 1995; Chantelot et al., 2005; Ferguson et al., 2016; Jones et al., 2008; Kapoor et al., 2008; Merrell et al., 2002; Pinder et al., 2015; Stark et al., 1988).

The purpose of this study was to determine whether proximal pole vascularity affects the likelihood of healing or the time to union in patients with scaphoid nonunions treated by non-vascularized autogenous bone grafting and rigid fixation.

Methods

This prospective longitudinal study was approved by our Institutional Review Board. Thirty-five patients with a confirmed diagnosis of scaphoid nonunion (≥3 months from injury) who underwent surgical reconstruction at our hospital from November 2014 to September 2016 were consented and enrolled in a service-wide scaphoid nonunion registry. Six patients who presented following failed surgical intervention elsewhere included. Definitions for the vascular terminology as used in this study are detailed in Table 1.

Preoperative wrist MRIs were obtained at the surgeon’s discretion. MRI was carried out using a standardized wrist protocol on a 1.5T clinical magnet with a dedicated surface wrist coil. Multiplanar fluid-sensitive short tau inversion recovery (STIR) and proton density (PD) sequences were obtained (Figures 1 and 2). Metal artefact reduction sequences were also used in five patients after failed surgery who had fixation devices in place. Preoperative MR studies were blindly interpreted by a single musculoskeletal MRI radiologist for proximal pole signal intensity relative to normal marrow fat of the surrounding carpal bones on STIR and PD sequences, according to a 4-point vascularity scale (Table 2).

All nonunions were treated with thorough curettage of the proximal and distal poles down to healthy appearing bone, non-vascularized autogenous bone grafting [34 ipsilateral distal radius, one iliac crest] and rigid internal fixation. A volar approach was used in 23 cases and a dorsal approach in 12. All curetted material from the nonunion sites and from both scaphoid poles were sent as separate specimens for pathological analysis. Intraoperative punctate bleeding of the proximal and distal poles was graded as ‘good’, ‘fair’ or ‘poor’ using the classification system established by Green (1985). When present (11 patients), dorsal intercalated segment instability (DISI) was reduced and the lunate was pinned in neutral to the distal radius. Sixteen patients were treated with standard internal fixation with a ‘hump-back’ collapse deformity of the scaphoid (Amadio et al., 1989) were treated with a hybrid Russe technique, using a cancellous bone graft from the anterior aspect of the ipsilateral distal radius, a cortical strut of bone and fixation with a headless screw (Lee et al., 2015). All 35 scaphoid constructs were fixed with a cannulated headless screw.
bone quality and/or previous screw tracks, the optimal screw insertion point and trajectory was determined by the surgeon. The screw was placed retrograde (distal to proximal) in 17 cases and antegrade in 18. Screw manufacturer varied and included TriMed (Santa Clarita, CA, USA), Acumed (Hillsboro, OR, USA), Medartis (Exton, PA, USA) and Synthes (Paoli, PA, USA).

Figure 1. Representative coronal proton density (left) shows a nearly isointense marrow signal in the scaphoid relative to the other carpal bones, with corresponding STIR signal hyperintensity (right), probably reflecting stress reaction and/or mucinous changes.

Figure 2. Representative coronal proton density MRI (left) shows diffuse hypointense marrow signal in the scaphoid pole and signal hyperintensity on the corresponding STIR sequence (right). The findings suggest ischaemic but not completely devitalized marrow.
All specimens were fixed and decalcified before haemotoxylin and eosin (H&E) staining and examination by light microscopy. Histopathological assessment was performed by a board-certified musculoskeletal pathologist, without knowledge of the intraoperative and MR findings. Trabecular viability was assessed by the percentage of osteocytic lacunae and necrotic trabeculae according to a 4-point scale: Grade 1 (<20% viability) (Figure 3), Grade 2 (20%–50% viability), Grade 3 (50%–90% viability), or Grade 4 (>90% viability) (Figure 4). Preservation of marrow fat, fibrochondral, trabecular, vascular and loose connective tissue (tissue viability) throughout the proximal pole was also assessed to assign a viability score according to the same 4-point scale. Each specimen was separately assessed for the degree of bone repair (‘remodelling’) activity based on the presence or absence of osteoclastic and osteoblastic activity and classified as ‘none’, ‘focal’ or ‘robust’. The first 15 samples were blindly evaluated by a second independent pathologist from an outside institution for analysis of reliability.

Healing in all patients was assessed by the radiologist with computed tomography (CT) using contiguous 0.625 mm cuts along the longitudinal axis of the scaphoid in both the oblique coronal and sagittal planes. CT scans were usually ordered at 10 to 12 weeks postoperatively; when there was incomplete healing, repeat CT scans were usually done at 4–8 week intervals. A nonunion was classified as successfully healed if sequential CT slices confirmed osseous bridging across ≥50% of the fracture gap (Figure 5). Union was classified as ‘delayed’ if healing was progressive but not achieved until after 6 months. ‘Non-union’ was classified as failure to reach 50% bony bridging by 1 year after surgery.

**Statistical methods**

Histopathological trabecular and tissue viability scores were assessed by individual category and then merged into binary groupings (grades 1/2 = ≥50% necrotic; grades 3/4 = ≤50% necrotic) for analysis. Chi-square tests were used to compare the following: MRI signals with punctate bleeding, trabecular viability, tissue viability, remodelling activity and fracture location; punctate bleeding with trabecular viability, tissue viability and remodelling

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**Table 2.** Vascularity scale used to grade magnetic resonance findings.

<table>
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<th>Category</th>
<th>Description</th>
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<tr>
<td>Grade 1</td>
<td>Vascular compromise concerning for necrosis (STIR hypointense and PD hypointense)</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Any focal areas of ischemia but with retained vascularity (STIR hyperintense and PD hypointense)</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Largely normal but with oedema pattern (STIR hyperintense and PD isointense)</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Entirely normal (STIR isointense and PD isointense)</td>
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</table>

STIR: multiplanar fluid-sensitive short tau inversion recovery sequence; PD: proton density imaging sequence.

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activity; and trabecular viability with tissue viability, remodelling activity and fracture location. Non-parametric Mann–Whitney U-tests were used to compare vascular assessments (MRI signal, punctate bleeding, trabecular viability, tissue viability, remodelling activity and fracture location) with time to union. Fisher’s exact tests were used to compare each vascular assessment with union rate. Inter-reader reliability analysis of histopathological trabecular viability grading was assessed with Cohen’s kappa. Although no formal power analysis was done, we estimated that 200 patients would have been needed to detect significant associations. A $p$-value of 0.05 was used to designate statistical significance.

Results

Demographics

Five board-certified orthopaedic hand surgeons contributed 35 patients (six women) (mean age 23 years; range 14–49) to the registry. These were unselected consecutive cases. No vascular bone grafting procedures were done at our institution during this collection period. The mean interval between injury and surgery was 32 months (range 3–288). There were five distal pole, 21 waist and nine proximal pole nonunions.

Magnetic resonance imaging

Twenty-three patients underwent preoperative MRI. Nine were classified as ischaemic (Grade 2). The remaining 14 were judged mostly normal with oedema (Grade 3). There were no cases in which the proximal pole demonstrated abnormal signal intensity on both the STIR and PD sequences to suggest complete necrosis. There was no significant association between MRI signal intensity and punctate bleeding ($p=0.77$), trabecular viability ($p=0.35$), tissue viability ($p=0.71$), remodelling activity ($p=0.60$) or fracture location ($p=0.13$).

Intraoperative bleeding

Punctate bleeding data were complete on 33 patients. The tourniquet was inflated in 28 of the patients during bleeding assessment. Five proximal poles had ‘poor’ bleeding intraoperatively and were judged totally avascular. Nineteen had ‘fair’ bleeding and nine had ‘good’. Of the 22 patients with both MRI and punctate bleeding data, five had ‘poor’ bleeding, 13 had ‘fair’ and four had ‘good’. There was no significant association between punctate bleeding and MRI signal intensity ($p=0.77$), trabecular viability ($p=0.90$), tissue viability ($p=0.88$), remodelling activity ($p=0.49$) or fracture location ($p=0.051$).
Histopathological analysis

Trabecular viability data were available on 32 patients. In two cases, there was not enough tissue to assess and in one case occult infection was suspected, which limited the examination of viability. Fourteen showed ≥50% necrotic trabeculae under microscopic examination and seven of these cases had more than 80% necrotic trabeculae (Grade 1). Of the 20 patients with both MRI and percentage data of necrotic trabeculae, seven showed ≥50% necrotic trabeculae. There was no significant association between the percentage of necrotic trabeculae and MRI signal \((p=0.35)\), punctate bleeding \((p=0.90)\) or fracture location \((p=0.24)\). On subgroup analysis of the seven patients with >80% necrotic trabeculae, no significant association was found with MRI signal \((p=0.76)\), punctate bleeding \((p=0.94)\) or fracture location \((p=0.53)\). Inter-observer reliability between the two musculoskeletal pathologists for the first 15 samples was ‘substantial’ \((K=0.667)\) according to the grading of Cohen [1960].

Tissue viability data were available on 33 patients. Under microscopic examination, four patients had ≥50% necrotic marrow fat, fibrochondral, trabecular, vascular and loose connective tissue, and two cases had more than 80% necrotic tissue (Grade 1). Of the 21 patients with both MRI and necrotic tissue data, two patients showed ≥50% necrotic tissue. There was no significant association between the percentage of necrotic tissue and trabecular viability \((p=0.06)\), MRI signal \((p=0.71)\), punctate bleeding \((p=0.88)\) or fracture location \((p=0.83)\).

Histopathological remodelling data were available on 33 patients; in two cases, there was not enough tissue to assess. Twenty-four had robust remodelling activity, six demonstrated focal activity and three showed no activity. Of the three cases with no remodelling activity, two had 50%–90% viable trabeculae (Grade 3) and one had less than 20% viable trabeculae (Grade 1). Of the 21 patients with MRI and remodelling data, 16 showed robust remodelling, three showed focal remodelling and two showed no remodelling. There was no significant association between remodelling activity and punctate bleeding \((p=0.49)\) or MRI signal intensity \((p=0.60)\). However, there was a positive association with trabecular viability \((p=0.01)\) and tissue viability \((p=0.01)\).

Healing

Thirty-three scaphoids healed by an average of 12 weeks (range 6–22) and 34 of 35 healed overall. One patient was initially lost to follow-up at 8 weeks when her cast was removed and did not return until 11 months postoperatively, at which time healing was confirmed by CT; she was not included in the time-to-union analysis. One patient had failure of fixation at 14.5 weeks; the fracture was healed 18 weeks after revision by further internal fixation with bone graft. There was one delayed union in a patient who was non-compliant and returned to swimming and mountain biking shortly after the index procedure while still in a cast. He developed a superficial infection and did not reach 50% bony bridging on CT until 38 weeks.

There was no significant correlation between union rate and trabecular viability \((p=0.21)\), tissue viability \((p=0.85)\), remodelling activity \((p=1.00)\), punctate bleeding \((p=0.62)\) or fracture location \((p=0.69)\). All patients with preoperative MRI healed and thus no analyses could be carried out to assess correlation with union rate. There was no significant correlation between time to union and trabecular viability \((p=0.35)\), tissue viability \((p=0.56)\), remodelling activity \((p=0.70)\), fracture location \((p=0.97)\), punctate bleeding \((p=0.96)\) or MRI signal intensity \((p=0.56)\).

Discussion

Non-vascularized autogenous bone grafting and rigid internal fixation led to successful healing in this series of scaphoid nonunions with a high incidence of compromised vascularity, as demonstrated by accepted preoperative, operative and histopathological assessments.

Accurate assessment of vascularity is considered important when planning scaphoid nonunion reconstruction. In our study, MRI, intraoperative punctate bleeding and histopathological examination all demonstrated substantial degrees of ischaemia, but none correlated with another, nor with time to healing or union. The lack of correlation between high resolution imaging or intraoperative bleeding points and histopathological viability suggests that our ability to predict scaphoid vascularity is poor.

Green [1985] concluded that poor punctate bleeding was predictive of surgical failure when all five patients in his case series with ‘complete’ AVN failed to heal with cortico-cancellous grafting. It should be noted that internal fixation was not used in these patients. Although magnetic resonance findings have been compared with punctate bleeding in the diagnosis of AVN, few studies since Green’s original series have examined the association between bleeding points and prognosis (Güral et al., 1999; Megerle et al., 2011; Robbins et al., 1995). Furthermore, despite concluding poor punctate bleeding was predictive of failure to heal, Green [1985] noted that bleeding points did not always
agree with identification of avascular trabeculae on histological sectioning. Based on the lack of evidence for punctate bleeding in previous publications and our inability to correlate punctate bleeding with any outcome factors, we feel that this parameter is unreliable as an indicator of scaphoid vascularity or healing potential.

Trumble (1990) reported a correlation between diminished signal intensity on T₁- and T₂-weighted MRI and histopathological confirmation of necrotic trabeculae in six patients. He proposed that AVN may signal poor prognosis with screw or K-wire fixation when three of these six patients failed to unite. In a randomized clinical trial, Ribak et al. (2010) found a significantly greater percentage of scaphoid non-unions achieved healing with vascularized (89.1%) versus non-vascularized (72.5%) bone grafting, but used percutaneous K-wire instead of compression screw fixation. Robbins et al. (1995) reported successful complete or partial (~50%) bony union after non-vascularized bone grafting with screw fixation in ten of 17 patients diagnosed with AVN by punctate bleeding, but CT was not performed.

Although MRI has been used to assess scaphoid viability, the variability of technique limits definitive conclusions. The reported utility of intravenous contrast varies by study. Fox et al. (2010) calculated that decreased signal intensity on unenhanced T₁-weighted magnetic resonance images had 79% (23/29) accuracy for proximal pole vascularity when compared with intraoperative punctate bleeding, whereas Günal et al. (1999) found unenhanced MRI and punctate bleeding agreed in only 59% (19/32) of cases. Using either histological analysis or punctate bleeding, two other studies have reported an improved sensitivity of contrast-enhanced MRI compared with non-contrast in diagnosing proximal pole AVN (Cerezal et al., 2000; Schmitt et al., 2011). A separate study reported superior sensitivity and accuracy for non-contrast over contrast-enhanced MRI (Fox et al., 2015).

Donati et al. (2011) reported contrast-enhanced perfusion analysis did not correlate with histological examination and was diagnostically inferior to unenhanced MRI. Conversely, Ng et al. (2013) found dynamic perfusion analysis was diagnostically superior to both unenhanced and contrast-enhanced magnetic resonance when vascularity was defined by punctate bleeding. Sakuma et al. (1995) suggested that low intensity on unenhanced T₁- and T₂-weighted images may be associated with poor prognosis when both scaphoids with these features failed to unite after bone grafting with Herbert screw fixation. However, two independent series reported that contrast-enhanced MRI vascularity assessment was not predictive of surgical outcome (Megerle et al., 2011; Singh et al., 2004).

Histopathological analysis can be considered the current reference standard for scaphoid vascularity; unfortunately, it cannot be used preoperatively to guide treatment. However, despite the fact that nearly half of the patients in this series demonstrated greater than 50% trabecular necrosis of the proximal pole histologically, and nearly a quarter had >80% trabecular necrosis, 94% of our patients healed within 12 weeks and there was no correlation between histological assessment of necrosis and healing rate or time to union. The findings in this study of 91% active trabecular remodelling and 88% viable connective tissue and marrow fat indicates that true vascular infarction of the scaphoid is probably rare.

It is surprising that MRI, intraoperative punctate bleeding and histopathological examination all demonstrated substantial degrees of ischaemia in our surgical cohort, but none correlated with another, nor with time to healing or union. It is likely that each modality examines different aspects of vascular response to injury. Punctate bleeding may be an inaccurate vascular assessment as osteonecrosis may be variable within the proximal pole (Büchler and Nagy, 1995; Trumble, 1990). It is also possible that although decreased signal intensity on MRI may reflect loss of bone marrow fat, it cannot discriminate whether loss is due to necrosis, fibrosis or a decreased inter-trabecular space resulting from reparative activity (Simmons et al., 1989).

A limitation of this study is the paucity of nonunion and delayed unions, which may contribute to a statistical beta error. We estimated that 200 patients would be required to provide sufficient power for the statistical tests. Dynamic contrast-enhanced MRI to assess perfusion and collect quantitative data was not used, although in our experience it has not been successful in differentiating between viable and non-viable bone and the studies referred to above have disputed its utility for viability assessment. We were also unable to biopsy the entire proximal pole for histological examination. However, we argue that histological analysis is the most physiologically accurate vascular assessment and that future studies of vascular grafting should incorporate historical studies of the excised bone. Moreover, our cohort size is similar or larger than that in most studies examining diagnostic accuracy for scaphoid vascularity (Cerezal et al., 2000; Dawson et al., 2001; Donati et al., 2011; Fox et al., 2010; Günal et al., 1999; Morgan et al., 1997; Ng et al., 2013; Perlk and Guilford, 1991; Singh et al., 2004; Trumble, 1990).

Many authors have reported on the necessity of vascularized bone grafting in the setting of...
compromised scaphoid vascularity, citing proximal pole AVN as the primary concern (Chang et al., 2006; Fernandez and Eggli, 1995; Fox et al., 2015; Henry, 2007; Jones et al., 2008; Merrell et al., 2002; Steinmann et al., 2002). Our overall union rate of 97% matches that recently reported in a systematic review of 1600 patients that concluded there was no statistical benefit of vascular over non-vascular grafts for scaphoid nonunion (Pinder et al., 2015). Our data suggest that surgical curettage of sclerotic and necrotic bone, replacement with fresh non-vascularized autogenous graft and rigid fixation are highly effective in attaining scaphoid union, despite a high incidence of vascular ischaemia and histopathological osteonecrosis, and that vascularized bone grafting is seldom required for internal fixation of a scaphoid nonunion.

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