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Arthroscopic evaluation of the rotator cuff vasculature: inferences into the pathogenesis of cuff tear and re-tear

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Background: Little is known about alterations of the rotator cuff (RC) macroscopic vasculature associated with medical conditions and/or habits that predispose a person to diseases of the peripheral microcirculation. The high frequency of cuff tear and re-tear in patients with diabetes, hypercholesterolemia, uncontrolled arterial hypertension, or metabolic syndrome may be due to tissue hypovascularity.
Methods: The macroscopic vasculature of both the articular and bursal sides of the posterosuperior RC was evaluated arthroscopically in 107 patients (mean age, 58.2 years) with no RC tear. Patients were divided into three groups according to medical comorbidities and lifestyle factors (group I, none; group II, smokers and/or drinkers and one comorbidity; and group III, two or more comorbidities). Pulsating vessels originating from both the myotendinous and osteotendinous junctions were assessed as "clearly evident," "poorly evident," or "not evident."
Results: Groups I, II, and III comprised 36, 45, and 26 patients, respectively. Within the myotendinous junction, vessels were visualized in 22 group I patients (61%), 25 group II patients (55%), and 6 group III patients (23%) (P=0.007). Pulsating arterial vessels originating from the osteotendinous junction were seen in 42%, 36%, and 0% of patients, respectively (P<0.001). Within the bursal side of the RC, a dense anastomotic network was visualized (either clearly or poorly) in 94% (34), 80% (36), and 35% (9) of patients, respectively (P<0.001).
Conclusions: The macroscopic vasculature of the RC is influenced by pre-existing diseases and lifestyle factors, which may impair peripheral microcirculation.
Level of evidence: III.

Keywords: Rotator cuff injuries; Vasculature; Microvasculature

INTRODUCTION

Degeneration and consequent tearing of the rotator cuff (RC) have been largely attributed to the tissue's physiological senescence and intrinsic factors. Intrinsic factors fall into two categories: medical conditions (comorbidity) and lifestyle factors. The

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comorbidities thought to be associated with RC tear include diabetes [1-3], hypercholesterolemia [4-6], uncontrolled arterial hypertension [7], metabolic syndrome, and obesity [8,9]. Additional lifestyle factors that may predispose an individual to RC tear include smoking [10-12], disordered eating (eating disorders) [8,9], and excessive alcohol consumption [13], each of which may contribute to peripheral microcirculatory disease. The microcirculation of the RC is of particular interest with respect to the etiology of RV tear [14].

The tendons of the RC are supplied by the suprascapular, thoracoacromial, scapular circumflex, and anterior and posterior humeral circumflex arteries [15-19]. The posterior humeral circumflex artery supplies the teres minor tendon [20]; however, its role in the vascularization of the supraspinatus tendon is still a matter of debate, whereby this role is accepted by Determe et al. [21] but denied by Põldoja et al. [15]. The anterior and posterior circumflex humeral arteries cross the osteotendinous junction to provide most of the blood supply to the posterosuperior cuff [20,22]. Brooks et al. [23] found no significant difference between the vascularity of the supraspinatus and infraspinatus tendons from their humeral insertions. The supraspinatus muscle receives its blood supply from the suprascapular and dorsal scapular arteries, while the infraspinatus muscle is supplied by the suprascapular and circumflex scapular arteries [17]. However, the subscapular artery has been found to supply the greater portion of the infraspinatus muscle circulation through its dorsal or circumflex scapular branch [24]. The blood supply of the teres minor muscle derives from several vessels, typically including the branch arising from the posterior humeral scapular circumflex artery [24].

In this study, we performed arthroscopic visualization of the distribution of visible vessels on the articular and bursal sides of the posterosuperior cuff to investigate whether there were any macroscopic differences between subjects with and without the above-noted conditions and lifestyle factors. Our hypothesis was that these conditions compromised the vascular supply of the RC.

METHODS

The study protocol was approved by the Institutional Review Board of Sapienza University of Rome, which waived the requirement for informed consent since a standard surgical procedure was performed and the anonymity of the patients was ensured during data collection.

This prospective study initially included 128 patients aged 50 to 72 years at Istituto Chirurgico Ortopedico Traumatologico (ICOT) who underwent shoulder arthroscopy from January 2017 to December 2022 for examination of the macroscopic vasculature of the posterosuperior RC. Twenty-one patients diagnosed with RC tears, frozen shoulder, rheumatic, autoimmune diseases, and fracture sequelae were excluded. Patients who had undergone injections (intra-articular or subacromial) of aspirin, antiplatelet agents, steroids, or any other viscosupplementary substance were excluded [25].

Arthroscopic Evaluation

All procedures were performed by the senior author (SG). All arthroscopic procedures were performed with the patient under general anesthesia in the beach-chair position. The irrigation pump (Crossflow Arthroscopy Pump, Stryker) was set to 60 mmHg. Systolic blood pressure was maintained around 100 mmHg.

The conventional arthroscopic procedure [26] was augmented with additional steps to evaluate the macroscopic vascularization of the posterosuperior cuff from both the articular and bursal sides (through anterior and posterior portals). The arthroscope was inserted close to the tendons in an anteroposterior and mediolateral direction. The subacromial bursa was resected to expose the cuff on the bursal side.

Vessels originating from the myotendinous junction were "clearly evident," "poorly evident," or "not evident" when the anteroposterior extension was at least 1–3 cm, 0.5–1 cm, or <0.5 cm, respectively. The same criteria were adopted to evaluate the vascular extension of the vessels at the osteotendinous junction. The vessels on the bursal side were considered "well represented," "poorly represented," or "not represented" when the extension was 1–3 cm, 0.5–1 cm, or <0.5 cm, respectively. All measurements were performed using a millimeter-scale graduated probe.

Groups

For comparison of variables, the patients were divided into three groups. In group I, the patients had no comorbidities (diabetes, hypercholesterolemia, uncontrolled arterial hypertension, metabolic syndrome, or obesity) or unhealthy lifestyle factors (smoking, eating disorder, or excessive alcohol intake). Group II comprised patients who were smokers (defined as any patient who smoked at least 10 cigarettes per day at the time of surgery or with a smoking history of more than 40 pack-years [11,12]), drinkers (defined as consuming more than 24 g alcohol per day for at least 2 years [13]), and had one of the following comorbidities (diabetes, hypercholesterolemia, uncontrolled arterial hypertension, or body mass index >30 kg/m^2). In group III, the patients were smokers and/or drinkers and had two or more of the above comorbidities.

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Statistical Analyses

Statistical analyses of categorical data were performed using the chi-square test to identify associations between vascular type and patient group (as described above). In addition, Cramer's V test was used to measure effect size for tests of association for nominal variables. The Cramer's V statistic varies from 0 to 1, with 0 indicating no association and 1 indicating a perfect association. Cohen indicates the interpretation of V based on the k value, where k is the minimum number of categories in either rows or columns. For k = 3, Cramer's V values ranging from 0.07 to 0.20 indicate a weak association, values ranging from 0.20 to 0.35 indicate a moderate association, and values greater or equal to 0.35 indicate a strong association. Significance levels for multiple comparisons were adjusted using the Bonferroni procedure to maintain control of the false discovery rate. Calculated P-values were two-tailed with a 95% confidence interval, where appropriate, and a P-value less than 0.05 was considered significant.

RESULTS

The final study group comprised 107 patients (59 male and 48 female). The mean age was 58.2 ± 4.1 years. Groups I, II, and III comprised 36, 45, and 26 patients, respectively (Table 1). These patients underwent arthroscopic examination to assess glenohumeral instability without RC tear (28 patients), acromioclavicular injury (type III and V) with possible concomitant pathologies (e.g., superior labrum anterior to posterior 32 patients), acute rupture of the biceps long head tendon with healthy cuff (29 patients), and low-grade (Samilson-Prieto 1) glenohumeral arthropathy without synovitis after undergoing chondroplasty (18 patients).

The vessels identified in the myotendinous junction after arthroscopic examination of the articular side of the posterosuperior cuff are shown in Fig. 1. Differences between the three patient groups were significant ($\chi^2 = 14.21$, df = 4, P = 0.007), with a Cramer's V = 0.258 for k = 3, which indicates a moderate strength of association. These vessels were extremely thin, ran parallel to each other and perpendicular to the tendon insertion, and usually ran in pairs. When visible, the vessels did not reach the tendon insertion, which macroscopically appeared not to be vascularized (see critical area identified in Fig. 2). Some pairs of vessels formed terminal loops. The loops suggest that the vessels were interrupted, and that their inability to be visualized is not simply due to their deep position in the tissue.

The arterial vessels (pulsating) identified as originating from the osteotendinous junction are shown in Fig. 3. A significant difference between the three patient groups was found ($\chi^2 = 19.576$, df = 4,

Table 1. Distribution of patients within the three study groups

Variable	Group I (n = 36)	Group II (n = 45)	Group III (n = 26)
Male:female	19:17	24:21	16:10
Mean age (yr, mean ± SD)	57.8 ± 3.6	60.4 ± 3.5	56.3 ± 5.2

Group I: patients without comorbidities or unhealthy lifestyle factors, Group II: smokers and/or drinkers with 1 of diabetes, hypercholesterolemia, arterial hypertension, or metabolic syndrome, Group III: smokers and/or drinkers with two or more of the above comorbidities.
SD: standard deviation.

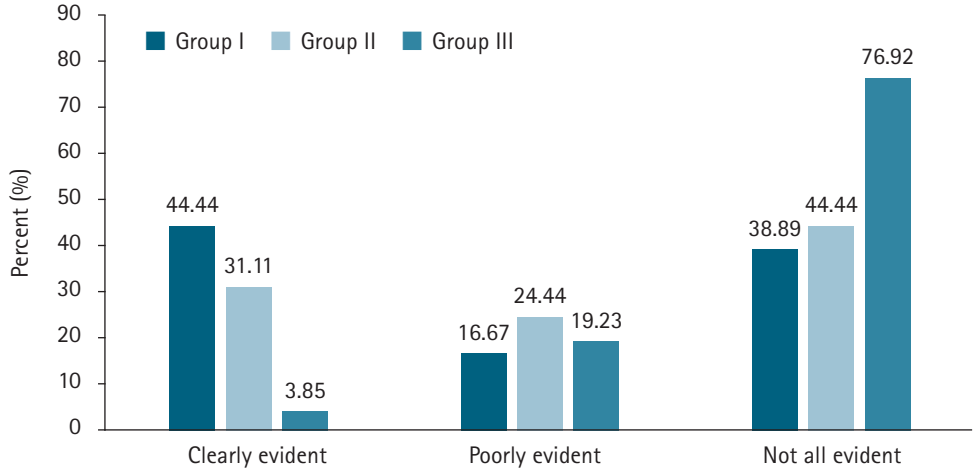

Fig. 1. Comparison of the vessels identified in the myotendinous junction of the shoulder joints of the three patient groups. Group I: patients without comorbidities or unhealthy lifestyle factors, Group II: smokers and/or drinkers with 1 of diabetes, hypercholesterolemia, arterial hypertension, or metabolic syndrome, Group III: smokers and/or drinkers with two or more of the above comorbidities.

P < 0.001) with a Cramer's V = 0.302 for k = 3, which indicates a moderate strength of association. When visible, these vessels were thin, immediately branched into vessels of smaller caliber, and were not observed after 3–7 mm (Fig. 4, Supplementary Video 1).

When viewed from the bursal side of the posterosuperior cuff, a dense anastomotic network (Fig. 5) was identified in 94% (34) of group I patients (clearly evident, 28 patients; poorly evident, 6 patients), 80% (36) of group II patients (clearly evident, 26 patients; poorly evident, 10 patients), and 35% (9) of group III patients (clearly evident, 2 patients; poorly evident, 7 patients) (Fig. 6). These differences were significant ($\chi^2 = 37.15$, df = 4, P < 0.001), with Cramer's V = 0.417 for k = 3, which indicates a strong association. These vessels were very superficial and of small caliber. It was sufficient to place a hooked probe about 8–10 mm from the osteotendinous insertion, without exerting pressure on the underlying tissue, to interrupt the blood flow. By detaching the probe from the cuff, the flow immediately resumed, and the area containing the anastomosis, previously and temporarily avascular, was once again supplied by vessels with flow in the opposite direction (Fig. 7).

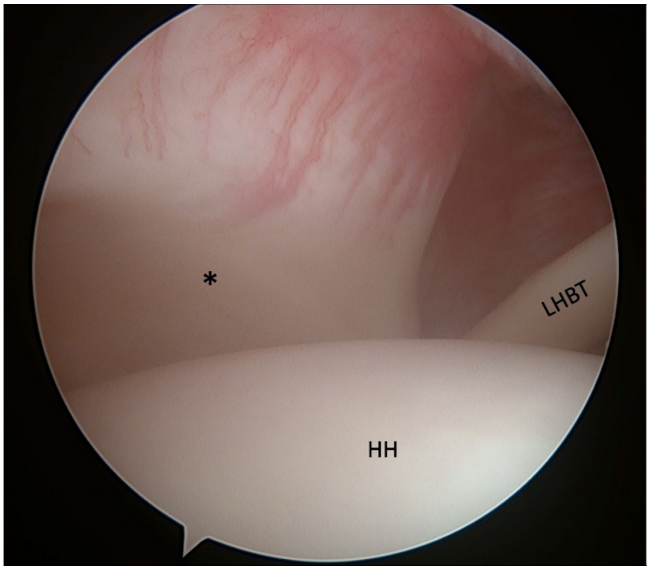

Fig. 2. Intra-articular view of a right shoulder from the posterior portal (patient placed in the beach-chair position). The vessels identified on the articular side were extremely thin, ran parallel to each other and perpendicular to the tendon insertion, and usually ran in pairs. The vessels did not reach the tendon insertion, which appeared macroscopically to not be vascularized (asterisk; critical area). HH: humeral head, LHBT: long head biceps tendon.

DISCUSSION

Our study showed that subjects with comorbidities or lifestyle habits capable of compromising the peripheral microcirculation had a less evident vascular texture of the RC on both the articular and bursal sides. This association was stronger for patients with more comorbidities or unhealthy lifestyle factors. The etiology or mechanism underlying this association remains a matter of debate. Various pathological conditions, including hyperglycemia, hyperlipidemia, hypertension [27-29], aging [27] and exposure to specific drugs [27], may impact endothelial function by disrupting the molecular mechanisms regulating nitric oxide (NO) bioavailability. NO is the major endothelium-derived second messenger responsible for the maintenance of vascular homeostasis. Reduction in NO bioavailability, resulting from reduced NO production and/or increased NO degradation by superoxide anion, marks the onset of endothelial dysfunction [30].

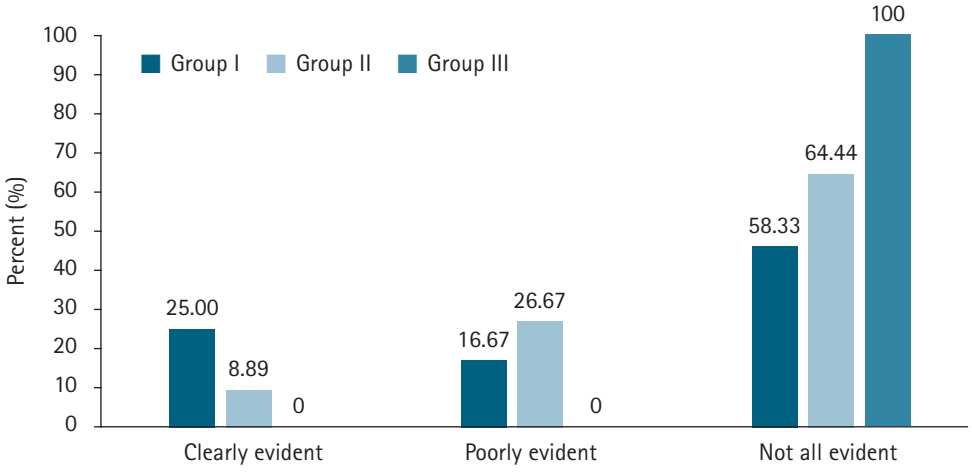

Fig. 3. Comparison of the vessels identified in the osteotendinous junction of the shoulder joints of the three patient groups. Group I: patients without comorbidities or unhealthy lifestyle factors, Group II: smokers and/or drinkers with 1 of diabetes, hypercholesterolemia, arterial hypertension, or metabolic syndrome, Group III: smokers and/or drinkers with two or more of the above comorbidities.

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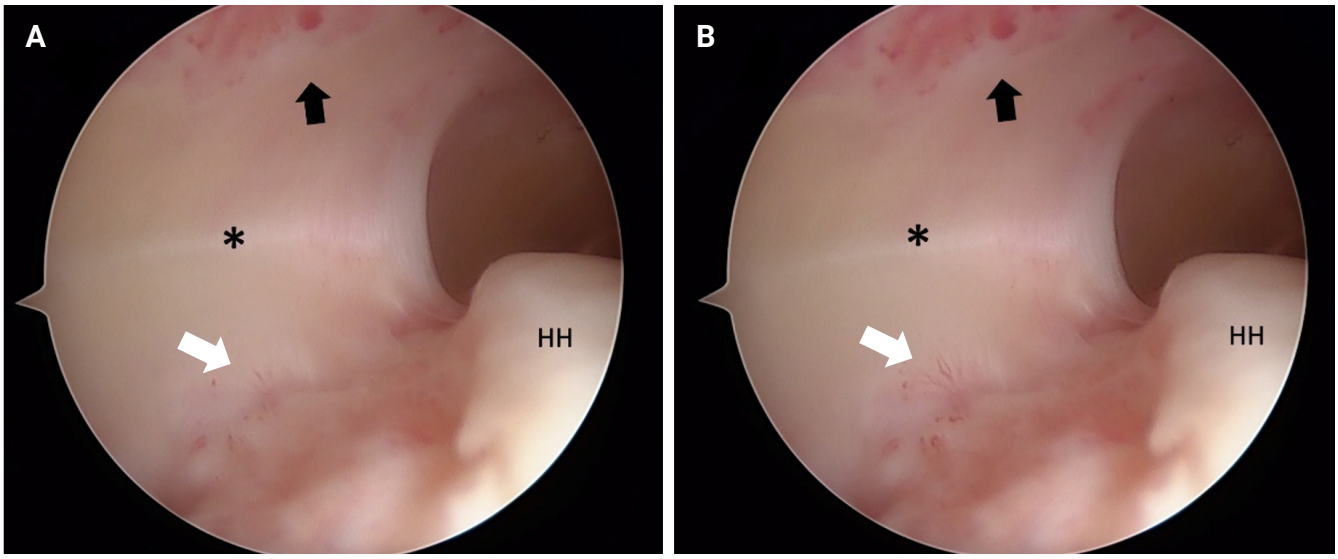

Fig. 4. Intra-articular view of a right shoulder from the posterior portal (patient placed in the beach-chair position). Arterial vessels (pulsating) originating from the osteotendinous junction (white arrows) and arterial vessels originating from the musculotendinous junction (black arrows) were identified. The critical area (asterisks) appeared macroscopically to not be vascularized in both the diastolic (A) and systolic (B) phases. HH: humeral head.

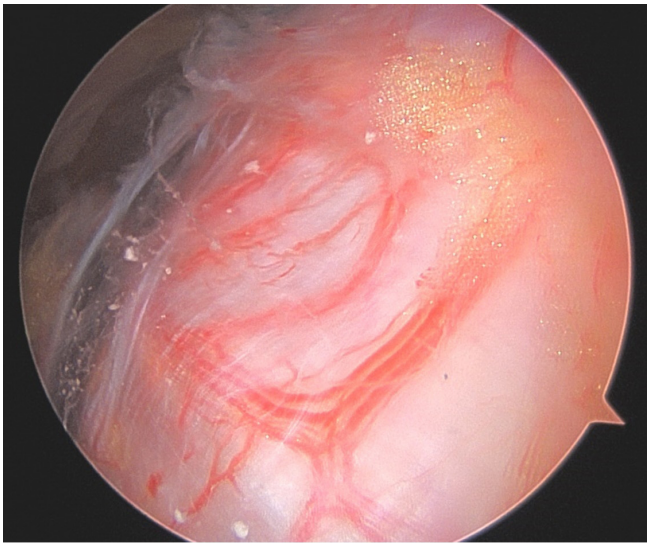

Fig. 5. Subacromial view of a left shoulder from the posterior portal (patient placed in the beach-chair position). A dense anastomotic network was present on the bursal side. The vessels were superficial and of a small caliber.

The aforementioned pathologies are also responsible for an increase in the plasma concentrations of reactive oxygen species (ROS), which are formed as byproducts of physiological metabolism of molecular oxygen [27,30] or during bacterial killing and host defense responses [27,30-32]. At physiological concentrations, ROS act as important second messengers that transduce intracellular signals involved in various biological processes [28,33]. However, when aberrant production of ROS exceeds the buffering capacity of the antioxidant defense systems or when antioxidant enzymes are defective, oxidative stress occurs. Several studies have shown that oxidative stress plays a pivotal role in mediating the production and secretion of cytokines [30-33], linking ROS with inflammation and endothelial activation and dysfunction. Accumulation of ROS may induce apoptosis of tenocytes through the action of mediators such as metalloproteinases, cytokines, and c-Jun N-terminal kinases, which, when phosphorylated, leads to cell death [27,33]. Over time, apoptosis causes matrix degeneration and reductions in collagen synthesis and mechanical tendon resistance, leading to tissue degeneration and disruption/tearing [27,33].

Chansky and Iannotti [19] and Rothman and Parke [6] postulated that hypovascularity plays a significant role in the genesis of RC degeneration and tearing. Rathbun and Macnab [34] undertook a microangiographic study in which injected specimens were sectioned to show a histologic correlation between the area of vascularity and the degenerative pattern observed. Those researchers found that degenerative changes first occurred in the area of hypovascularity and were always more extensive in that area.

As previously noted by Lohr and Uhthoff [22], we observed that the articular side of the tendon is at a disadvantage in terms of blood supply in comparison with the bursal side. It might be hypothesized that this deficient vascularization increases susceptibility of the articular side to degenerative changes and, consequently,

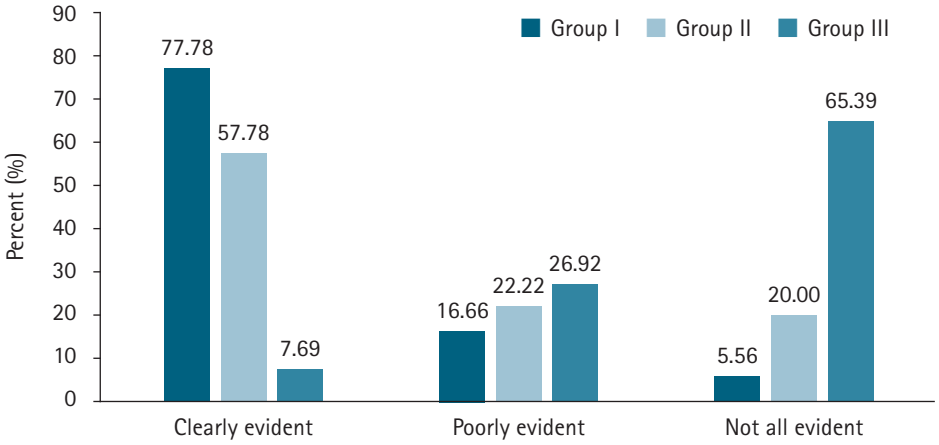

Fig. 6. Comparison of the vessels identified on the bursal side of the shoulder joints of the three patient groups. Group I: patients without comorbidities or unhealthy lifestyle factors; Group II: smokers and/or drinkers with 1 of diabetes, hypercholesterolemia, arterial hypertension, or metabolic syndrome; Group III: smokers and/or drinkers with two or more of the above comorbidities.

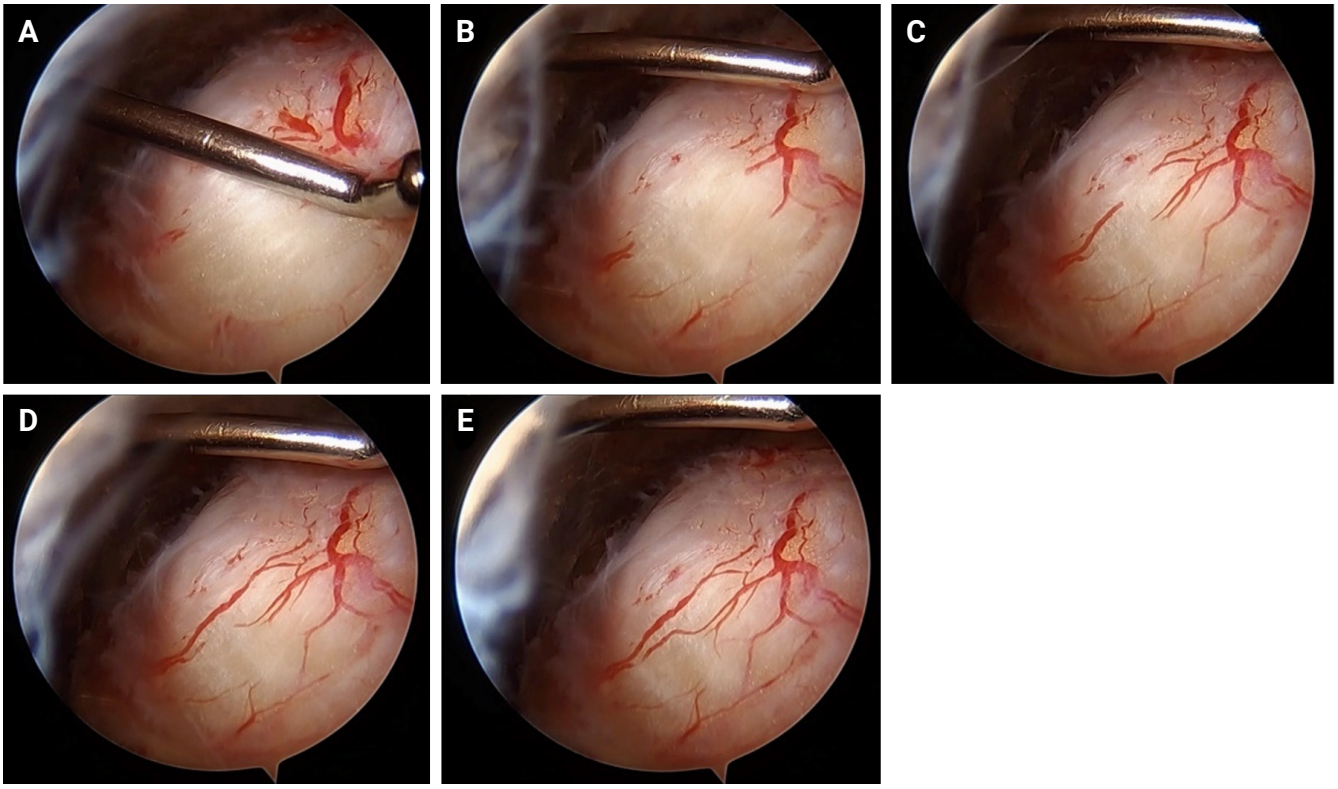

Fig. 7. Subacromial view of a left shoulder from the posterior portal (patient placed in the beach-chair position). The hooked probe was able to interrupt the blood flow even without exerting pressure on the underlying tissue (A). By detaching the probe from the cuff (B-E), the flow immediately resumed, and the area containing the anastomosis, previously and temporarily avascularized, was once again vascularized.

to tears.

Our study also showed that the vessels on the bursal side of the RC form a dense anastomotic network and are thin in caliber. Furthermore, we found that the blood flow of these vessels can be easily interrupted by placing a hooked probe on them without exerting pressure. These findings indicated that the vascular supply of these vessels is precarious. Some authors have suggested that the vascular supply of the cuff can be changed according to the posi-

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tion of the arm [34]. These data led us to ask if double row (DR) complex constructs are strongly ischemic and predisposed to failure. Although the literature provides indications that the DR offers greater likelihood of a mechanical seal than simple constructions (single row [SR]) [35-41], the failure rate of DR is still high.

There is an apparent contradiction with respect to the DR technique, in that it is a biomechanically superior construct but is hypothetically a more ischemic construct compared to the SR technique. However, none of the published studies comparing the two techniques have taken into account the state of degeneration of the tissue to be repaired, which is independent of the macroscopic characteristics of the tissue and the tear size. For example, Sethi et al. [42] found that the degree of tendinopathy was not correlated with the morphological appearance of the tendon, and neither of these parameters were correlated with healing or patient outcome. The authors concluded that the degree of tendinopathy, unlike muscle atrophy, may not be predictive of outcomes, and that a tendon that appears to be of poor quality may have adequate healing capacity. Therefore, abnormal gross tendon appearance should not affect the repair effort or technique. Nor, in order to compare the two techniques, they have been considered parameters, such as osteoporosis [43], comorbidities [14], genetics [44], or direction of the anchors into the greater tuberosity [20] have been found to influence tendon healing. In the last two decades, the focus has shifted from forcing the tendon to remain in contact with the bone through the use of the biomechanically more tenacious construct to optimize healing of the RC by ensuring the tendon had the vascular supply necessary for healing.

Our study demonstrated that the superficial and macroscopically vascularization of the RC is reduced when the shoulder is in extension in patients with comorbidities or lifestyle habits that predispose them to impairment of the peripheral microcirculation. Therefore, it can be postulated that deficient vascularization makes the RC tissue more susceptible to degenerative changes and, consequently, to tears. Furthermore, because the macroscopically visible vessels were thin and their blood flow was easily interrupted upon contact with a probe with little or no pressure exerted, we suggest re-evaluation of the long-term effectiveness of securing a torn RC with strong and taut sutures that may ultimately result in local ischemia and failure of the repair. Unfortunately, the available studies comparing the outcomes of the DR vs. SR techniques for RC repair have not taken into account the initial state of degeneration of the tissue to be repaired, and the findings cannot be used to support our hypothesis.

The limitations of this study included the small sample size and the subjective grading of vascularity of the shoulder joint tissues. Furthermore, no statistical correlation was observed between vascular appearance and age. As acute trauma (acromioclavicular injury) may affect the vasculature of the RC, any patient having a hemarthrosis on preoperative magnetic resonance imaging was excluded.

CONCLUSIONS

The vascularization of the RC is macroscopically influenced by factors than may impair peripheral microcirculation, including comorbidities (diabetes, hypercholesterolemia, uncontrolled arterial hypertension, metabolic syndrome, or obesity) and lifestyle factors.

NOTES

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Conflict of interest

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Data availability

Contact the corresponding author for data availability.

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SUPPLEMENTARY MATERIALS

Supplementary materials can be found via https://doi.org/10.5397/cise.2024.00066.

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