Review Article

PORTO BIOMEDICAL JOURNAL
WHERE SCIENCE MEETS KNOWLEDGE

OPEN

Diabetes: a silent player in musculoskeletal interventional radiology response

Sofia Dimitri-Pinheiro, MD[a,b,*], Madalena Pimenta[c], Beatriz Cardoso-Marinho[d], Helena Torrão[a], Raquel Soares[b,e], Apostolos Karantanas[f,g]

Abstract

Diabetes has an important role in the development of several musculoskeletal disorders, such as adhesive capsulitis of the shoulder (ACs) and stenosing flexor tenosynovitis of the finger (SfTf). The etiopathophysiology of ACs and SfTf in diabetic patients is associated with both chronic hyperglycemia, increased amounts of visceral adiposity and chronic inflammation. Chronic hyperglycemia stimulates the creation of cross-links between collagen molecules, impairing degradation and resulting in the build-up of excessive collagen deposits in the cartilage, ligaments, tendon sheaths and tendons. Increased adipocytes in diabetic patients secrete proteins and cytocines such as TNF-α, IL-6 and IL-13 which result in overproduction of pro-inflammatory factors, destruction of normal tissue architecture and fibrosis. Both hyperglycemia and adipocytes inhibit efferocytosis, limiting natural resolution. Recently, multiple image-guided interventional radiology musculoskeletal treatment options have been developed, such as ultrasound-guided glenohumeral capsule hydrodistension for ACs and ultrasound-guided percutaneous pulley release for trigger finger. Diabetes can negatively influence outcomes in patients with ACs and SfTf and may impact the decision of which specific procedure technique should be employed. Further studies are necessary to define how diabetes influences response to interventional radiology treatments of these disorders, as well as the extent to which control of blood sugar levels can contribute towards the personalization and optimization of patient follow up.

Keywords: adhesive capsulitis/shoulder, collagen, diabetes/complications, hydrodistension, trigger finger, ultrasound

Introduction

Diabetes is a metabolic disorder which is caused by resistance to insulin, either as a result of insufficient insulin secretion, insufficient insulin action, or both, and is mainly characterized by the presence of hyperglycemia. Although the latter is the cornerstone of diabetes, the decreased action of insulin on target organs also results in abnormalities in carbohydrate, fat, and protein metabolism, among others.[1] Diabetes affects practically all organs and systems in the human body, with chronic hyperglycemia found in diabetic patients being associated with long-term dysfunction of the heart, blood vessels, eyes, kidneys, central and peripheral nervous system and musculoskeletal system.[1] Although musculoskeletal disorders are not usually considered the most prominent of the alterations originated by diabetes, recent research has suggested that diabetes has an important role in the development of several conditions, such as adhesive capsulitis of the glenohumeral joint (AC), stenosing flexor tenosynovitis of the finger (SfTf), calcific tendinitis, among others, with an increased prevalence of diabetes in these patient populations.[2–5] Diabetes on its own does not cause such disorders, but it can be a predisposing factor, and it also affects the severity, natural history and response to treatment of the disorder, with diabetic patients having more intense symptoms and worst treatment response rates in general.[6,7] Recent studies have begun to define the etiopathophysiology behind the predisposition towards the development of musculoskeletal disorders in diabetic patients, as well as the increased severity of pain and functional impairment and lower treatment efficacy.[6,8,9]

In diabetic patients, the degree of hyperglycemia is influenced by the severity of the underlying metabolic syndrome, as well as other extrinsic and intrinsic factors, such as the level of inflammation caused by osteoarticular disorders.[10,11] There is an intimate relationship between the degree of hyperglycemia and development and gravity of musculoskeletal collagen disorders, such as AC and SfTf. Hyperglycemia affects the predisposition, severity and response to treatment of collagen musculoskeletal disorders, and the higher levels of circulatory inflammation mediators present in patients with these musculoskeletal conditions hindering glycemic control in diabetic patients.[10,11]

Musculoskeletal collagen disorders are common and multiple in diabetic patients.[5,8,12,13] They often result from microvasculature and connective tissue pathologic changes, which are triggered by chronic hyperglycaemia, increased adipose tissue and chronic inflammation.[14] In this review, we will focus on 2 main musculoskeletal disorders which are predisposed to and

[a] Radiology Department, Portuguese Institute of Oncology of Porto – Francisco Gentil EPE, [b] Biomedicine Department, Unit of Biochemistry, Faculty of Medicine, University of Porto, [c] Radiology Department, São João Hospital Centre, [d] Sports Medicine Department, Sports Medicine Centre of Porto, [e] I3S - Institute for Innovation and Health Research, University of Porto, Porto, Portugal, [f] Medical Imaging Department, University Hospital, [g] Radiology Department, University of Crete, Heraklion, Greece

[*] Corresponding author. Rua Gustavo de Sousa, 51, 4100-005 Porto, Portugal. E-mail address: dimitrisofia@hotmail.com (Sofia Dimitri-Pinheiro).

Porto Biomed. J. (2021) 6:1(e112)

Received: 11 October 2020 / Accepted: 14 October 2020

http://dx.doi.org/10.1097/j.pbj.0000000000000112

exacerbated by diabetes—AC and SfTf. Recently, multiple image-guided interventional radiology musculoskeletal treatment options have been developed, such as ultrasound (US)-guided glenohumeral capsule hydrodistension for AC and US-guided percutaneous pulley release for trigger finger. In this review, we aim to summarize recent research providing insight into the etiological mechanisms through which diabetes contributes towards collagen disfunction and accumulation, resulting in the development of adhesive capsulitis and stenosing tenosynovitis of the finger, as well as how diabetes influences response to interventional radiology treatments of these disorders.

Etiopathophysiology of collagen musculoskeletal disorders

The etiopathophysiology of AC and SfTf in diabetic patients is associated with both chronic hyperglycemia, increased amounts of visceral adiposity and chronic inflammation.[13–15] Chronic hyperglycemia stimulates the creation of cross-links between collagen molecules, impairing degradation and resulting in the build-up of excessive collagen deposits in the cartilage, ligaments, tendon sheaths and tendons.[13] Snedeker et al studied collagen crosslinks in diabetic patients and found that the increased connective tissue rigidity found in AC patients may be associated with non-enzymatic oxidative reactions between the increased blood glucose and collagen, with consequent formation of advanced glycation end-products.[13] Oliva et al also found that advanced glycation end-products can cause alterations in the ultrastructure of collagen fibers, making them harder to breakdown, resulting in excess accumulation and biomechanical changes in joint tissue quality.[16] Hyperglycemia also interferes with the inflammatory cascade and inhibits efferocytosis, limiting natural resolution.[17]

Adipocytes secrete proteins and cytokines such as tumour necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), resulting in overproduction of other pro-inflammatory cytokines. This pro-inflammatory environment will, in turn, further exacerbate inflammation and insulin resistance, for instance, via secretion of TNF-α and IL-6 by neutrophils in a continuous positive feedback loop.[13] Adipocytes also secrete IL-13, which has been shown to cause liver fibrosis in mouse models, and most likely also contributes towards synovial and connective tissue fibrosis.[18] Furthermore, adipocytes produce and secrete free fatty acids, which result in the up-regulation of pro-inflammatory mediators and exacerbated production of inflammatory cytokines.[14,19,20] Free fatty acids also promote neutrophil survival and decrease apoptotic cell removal by macrophages and other phagocytic cells, exacerbating collagen deposition in the glenohumeral capsule, among other extracellular matrix components, resulting in a rigid and painful shoulder.[14]

Chronic inflammation in diabetic patients may also lead to the excessive accumulation of collagen and other extracellular matrix components and synovial and connective tissue fibrosis, with resulting destruction of normal tissue architecture in joints, tendons, ligaments and pulleys.[21] The combination of these factors (chronic hyperglycemia, increased adipocytes and inflammation) results in disease perpetuation and fibrosis.

Adhesive capsulitis and diabetes

AC, or "frozen shoulder," is a common condition of the shoulder caused by the contraction of the glenohumeral capsule and adherence to the humeral head. Clinically, AC is characterized by shoulder pain and stiffness, with significant decrease of active and passive range of motion (ROM). The natural history of AC can be described as a progression through 3 phases: freezing (insidious onset of shoulder pain with progressive loss of motion), frozen (gradual subsidence of pain, plateauing of stiffness with equal active and passive ROM), and thawing (gradual improvement of motion and resolution of symptoms).[22] Most cases of AC comprise mild symptoms, are self-limited, lasting 1 to 2 years, and can be managed using analgesics, physiotherapy and intra-articular corticosteroid injections.[23] However, in 20% to 50% of patients, symptoms can persist for longer or watchful waiting may not be practical. In these cases, capsule hydrodistension treatment should be considered as an alternative for short-term management.

Glenohumeral AC may be primary and idiopathic or, more commonly, secondary to diabetes mellitus, hyperthyroidism, rotator cuff tendinopathy, rotator cuff tear, subacromial bursitis, biceps tendinopathy, previous shoulder surgery or trauma or systemic inflammatory diseases.[24,25] Diabetic patients in particular, have a greater prevalence of glenohumeral AC in comparison to nondiabetic patients (11%–30% prevalence in diabetic patients and 2 - 10% in nondiabetics).[2] Mavrikakis et al found that the prevalence of glenohumeral AC in type II diabetic patients was 31.8%, in comparison to 10.3% in nondiabetic controls.[26] According to Tighe et al, prediabetic patients also have an increased prevalence of glenohumeral adhesive capsulitis in comparison with nondiabetic patients.[27–29] The risk of development of diabetic glenohumeral AC increases with age and duration of diabetes.[7] Diabetic patients with glenohumeral AC generally present with more severe symptoms, a longer natural history of disease and are more resistant to treatment.[28]

Zreik et al set out to determine the mean prevalence of diabetes in a population of patients with glenohumeral adhesive capsulitis and found that it was 30%, which is significantly higher than the prevalence of diabetes in the general population.[30] In fact, the link between diabetes and the development of glenohumeral adhesive capsulitis is so strong that several authors have advocated that patients diagnosed with glenohumeral adhesive capsulitis who are not known to be diabetics should undergo diabetic screening.[9,31,32]

Ultrasound-guided glenohumeral capsule hydrodistention

The most effective treatment for glenohumeral AC remains uncertain.[33] Nonsurgical treatments include physiotherapy, oral or intra-articular corticosteroids, acupuncture, and US-guided glenohumeral capsule hydrodistension.[33] Arthroscopic capsular release is a valid treatment option in patients who are refractory to conservative treatment.[34]

There are many different techniques for US-guided glenohumeral capsule hydrodistension employed by different clinicians in different centers, and the ideal technique remains ambiguous. In general, the procedure consists in, under ultrasound guidance, the injection of a combination of corticosteroid and saline solution inside the glenohumeral capsule. The procedure works due to both the physical distention and release of the capsule caused by the fluid injected inside, and the anti-inflammatory effect of the drugs injected in the capsule, which will prevent the inflammatory mechanism resulting in the contraction of the glenohumeral capsule and adherence to the humeral head.

The optimal volume of fluid and components (saline solution and specific corticosteroids) injected into the glenohumeral

capsule remains undetermined, as well as how more or less severe degrees of AC will benefit from different doses of corticosteroid.[23] The procedure may also be carried out with maximum capsular distention without rupture, or with capsular rupture.[23] The role of capsular rupture in the efficacy of the procedure and whether it is essential for a successful outcome also remains to be determined. It is also of interest to determine whether diabetic patients, who in general have more severe disease and lower responses to treatment, will benefit from capsular rupture, which is a more aggressive form of treatment, or not. One could argue that a more severe form of disease will respond better to a more aggressive treatment. However, capsular rupture could also trigger more inflammation and pain in diabetic patients, resulting in longer recuperation times, without significantly better outcomes.

The doses of corticosteroid drug injected into the glenohumeral capsule must also be taken into consideration. It is general practice to inject larger doses of corticosteroid drug in the glenohumeral capsule in patients with more severe impairment of range of motion. Diabetic patients commonly have more severe disease, with more pain and significant limitation of both active and passive range of motion, warranting larger ratios of corticosteroid drug to saline solution, resulting in more significant and quicker symptom relief. However, one must also take into consideration the possible effects of corticosteroid drug in glycemic control in diabetic patients. There are yet to be conducted studies evaluating whether diabetic patients subjected to glenohumeral capsular hydrodistention experience significant blood glucose level fluctuations when injected with different doses of corticosteroid, or whether the impact is minimal due to this being a local treatment. US-guided glenohumeral capsule hydrodistension is a very promising option in the treatment and management of diabetic patients with AC. However, the optimal technique in these patients is yet to be determined.

Stenosing tenosynovitis of the finger and diabetes

SfTf, also known as trigger finger, is a common mechanical disorder, diagnosed clinically and characterized by finger locking and snapping from flexion to extension, pain, oedema and ROM limitation. Although it can affect any digit, it most commonly involves the flexor tendons of the thumb and the metacarpophalangeal joint.[35] It occurs primarily due to a mismatch between the size of the flexor tendons and that of the tendon sheath.[8] Whereas the pathophysiologic mechanism of trigger finger remains controversial, it is most frequently attributed to A1 pulley thickening secondary to repeated microtrauma.[36]

Trigger finger may be primary and idiopathic or, less commonly, secondary to chronic conditions such as diabetes, hyperthyroidism, gout, rheumatoid arthritis or chronic kidney disease, among others.[35] Diabetic patients have a greater prevalence of trigger finger in comparison to nondiabetic patients (5%–20% prevalence vs 1%–2% in the general population), with a 10% lifetime risk of developing trigger finger.[6,37,38] This may be because chronic hyperglycemia stimulates the creation of cross-links between collagen molecules in the tendon sheaths, impairing degradation and resulting in build-up around the flexor tendons of the finger.[13,39] The risk of developing diabetic trigger finger increases with age and duration of diabetes.[8,40] Also, diabetic patients have a bilateral presentation more commonly than nondiabetic individuals, as well as in multiple digits, reflecting the systemic nature of their disease.[8,39]

It remains controversial whether a tight blood glucose control can reduce the risk of developing diabetic trigger finger. For instance, Vance et al found that diabetics with HbA1c levels >7% are more likely to develop trigger finger.[6] However, Chammas et al found that disease duration, more than HbA1c levels, determined the risk of diabetic trigger finger.[3] Grandizio et al also stated that HbA1c levels did not influence trigger finger development in diabetic patients.[41] Furthermore, diabetic patients are more likely to develop trigger finger with severe symptoms[6,37,38] and have lower success rates with local corticosteroid injection treatment in comparison to nondiabetic patients (66% vs 90%).[4,42]

Ultrasound-guided A1-pulley release

Generally, the first-line treatment of trigger finger is conservative, with both oral nonsteroidal anti-inflammatory treatment and a splint or parenteral treatment with corticosteroids.[43] In case of conservative treatment failure, surgical treatment is usually recommended, with surgical release of the A1 pulley.[44–46] However, although positive outcomes are reported in 60% to 97% of cases, the procedure is invasive.[44] Recently, various techniques for US-guided percutaneous aseptic release of the A1 pulley have been developed. While the diagnosis of trigger finger is mainly clinical, ultrasound allows for direct visualization of the finger pulleys and tendons, becoming an increasingly useful tool in both diagnosis and treatment of trigger finger.

Although techniques for US-guided percutaneous pulley release may vary between different clinicians, the procedure generally consists of a few basic steps. Firstly, under ultrasound guidance, local anaesthesia is applied. Then, using a 18-gauge needle which has been bent to 90°, the A1 pulley is cut using the beasel of the needle, which should be positioned perpendicular to the horizontal plane of the flexor tendons. The patient is asked to move the finger, confirming that no locking or snapping is experienced and that the pulley is released. Finally, in order to reduce inflammation, the flexor tendon sheath is infiltrated with a corticosteroid.

US-guided pulley release has the advantage of being a minimally invasive, safe, quick, low-cost alternative to surgery. Real-time visualization of the vascular and nerve structures during the procedure minimizes complications such as interdigital vascular or nerve damage or overly wide pulley release.[45]

However, the current treatment algorithm for diabetic trigger finger remains controversial, as there are still no studies comparing the efficacy of surgical and US-guided percutaneous pulley release in diabetic patients.[12] Diabetic patients generally have longer recovery times in comparison to nondiabetic patients and report higher levels of pain and finger disfunction before treatment.[14,19-21] One could argue that the less invasive US-guided procedure would be beneficial for these patients, as there is less tissue disruption, with quicker healing times. It is currently believed that the local infiltration of low doses of corticosteroid around the flexor tendon sheath does not have a significant impact on glycemic control in diabetic patients, though more studies are still needed on this matter.

Conclusion

The continuous study and definition of the exact extent to which diabetes can influence the development and response to treatment of collagen musculoskeletal disorders, such as glenohumeral adhesive capsulitis and stenosing tenosynovitis of the finger, is of

the upmost importance. The determination of the prevalence of diabetes in populations of patients with musculoskeletal disorders and the comprehension of the etiopathophysiology of such disorders gives us a valuable and rare opportunity to act in the prevention of such diseases through perhaps careful control of blood sugar levels, as well as screening for diabetes in these populations, allowing for earlier detection of diabetes and prevention of permanent damage and dysfunction of other organs and systems. Furthermore, there is also the possibility that tight control of blood glucose levels may aid in response to treatment of glenohumeral adhesive capsulitis and stenosing tenosynovitis of the finger.

There are yet to be studies evaluating and comparing the impact of poorly or tightly controlled blood sugar levels in the response to treatment of these conditions. Further research is necessary to determine, for instance, the impact of diabetes on response to treatment of trigger finger with US-guided percutaneous pulley release or surgical pulley release in nondiabetic and diabetic patients, or the impact of diabetes in response to capsular hydrodistension treatment of glenohumeral adhesive capsulitis. It could also be of interest to determine the relationship between volume of fluid injected into the glenohumeral capsule and treatment efficacy, as well as how different doses of saline solution and corticosteroid affect procedure efficacy and post-procedure blood glucose levels in diabetic patients.[23] Although we have begun to understand the role of diabetes in the development of musculoskeletal disorders, a lot remains to be further determined in terms of pathophysiologic pathways and, in particular, in regards to the best treatment in diabetic patients. Recognizing the importance of diabetes as the silent player in development and response to treatment of collagen musculoskeletal disorders is a key step in the personalization and optimization of follow-up and quality of life of the diabetic patient.

Conflicts of interest

Authors have declared no conflict of interest.

References

[1] Diagnosis and classification of diabetes, mellitus. Diabetes Care. 2010;33 (Suppl 1):S62–S69.

[2] Lequesne M, Dang N, Bensasson M, Mery C. Increased association of diabetes mellitus with capsulitis of the shoulder and shoulder-hand syndrome. Scand J Rheumatol. 1977;6:53–56.

[3] Chammas M, Bousquet P, Renard E, Poirier JL, Jaffiol C, Allieu Y. Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. J Hand Surg. 1995;20:109–114.

[4] Griggs SM, Weiss AP, Lane LB, Schwenker C, Akelman E, Sachar K. Treatment of trigger finger in patients with diabetes mellitus. J Hand Surg. 1995;20:787–789.

[5] Crispin JC, Alcocer-Varela J. Rheumatologic manifestations of diabetes mellitus. Am J Med. 2003;114:753–757.

[6] Vance MC, Tucker JJ, Harness NG. The association of hemoglobin A1c with the prevalence of stenosing flexor tenosynovitis. J Hand Surg. 2012;37:1765–1769.

[7] Balci N, Balci MK, Tüzüner S. Shoulder adhesive capsulitis and shoulder range of motion in type II diabetes mellitus: association with diabetic complications. J Diabetes Complications. 1999;13:135–140.

[8] Fitzgibbons PG, Weiss AP. Hand manifestations of diabetes mellitus. J Hand Surg Am. 2008;33:771–775.

[9] Salek AK, Mamun MA, Haque MA, et al. Serum triglyceride level in type 2 diabetes mellitus patients with or without frozen shoulder. Bangladesh Med Res Coun Bull. 2010;36:64–67.

[10] Schmidt AM. Highlighting diabetes mellitus: the epidemic continues. Arterioscler Thromb Vasc Biol. 2018;38:e1–e8.

[11] Wilson PW, Kannel WB, Silbershatz H, D'Agostino RB. Clustering of metabolic factors and coronary heart disease. Arch Inern Med. 1999;159:1104–1109.

[12] Kuczmarski AS, Harris AP, Gil JA, Weiss AC. Management of diabetic trigger finger. J Hand Surg Am. 2019;44:150–153.

[13] Snedeker JG, Gautieri A. The role of collagen crosslinks in ageing and diabetes—the good, the bad, and the ugly. Muscles Ligaments Tendons J. 2014;4:303–308.

[14] Welty FK, Alfaddagh A, Elajami TK. Targeting inflammation in metabolic syndrome. Transl Res. 2016;167:257–280.

[15] Spite M, Clària J, Serhan CN. Resolvins, specialized proresolving lipid mediators, and their potential roles in metabolic diseases. Cell Metab. 2014;19:21–36.

[16] Oliva F, Piccirilli E, Berardi AC, Frizziero A, Tarantino U, Maffulli N. Hormones and tendinopathies: the current evidence. Br Med Bull. 2016;117:39–58.

[17] Kanter JE, Kramer F, Barnhart S, et al. Diabetes promotes an inflammatory macrophage phenotype and atherosclerosis through acyl-CoA synthetase 1. Proc Natl Acad Sci U S A. 2012;109: E715–E724.

[18] Sinha R, Patel P, Rose N, et al. Analysis of hydrodilatation as part of a combined service for stiff shoulder. Shoulder Elbow. 2017;9: 169–177.

[19] Nguyen MT, Favelyukis S, Nguyen AK, et al. A subpopulation of macrophages infiltrates hypertrophic adipose tissue and is activated by free fatty acids via Toll-like receptors 2 and 4 and JNK-dependent pathways. J Biol Chem. 2007;282:35279–35292.

[20] Glass CK, Olefsky JM. Inflammation and lipid signaling in the etiology of insulin resistance. Cell Metab. 2012;15:635–645.

[21] Kaviratne M, Hesse M, Leusink M, et al. IL-13 activates a mechanism of tissue fibrosis that is completely TGF-beta independent. J Immunol. 2004;173:4020–4029.

[22] Reeves B. The natural history of the frozen shoulder syndrome. Scand J Rheumatol. 1975;4:193–196.

[23] Rymaruk S, Peach C. Indications for hydrodilatation for frozen shoulder. EFORT Open Rev. 2017;2:462–468.

[24] van der Windt DA, Koes BW, de Jong BA, Bouter LM. Shoulder disorders in general practice: incidence, patient characteristics, and management. Ann Rheum Dis. 1995;54:959–964.

[25] Binder AI, Bulgen DY, Hazleman BL, Tudor J, Wraight P. Frozen shoulder: an arthrographic and radionuclear scan assessment. Anna Rheum Dis. 1984;43:365–369.

[26] Mavrikakis ME, Sfikakis PP, Kontoyannis SA, Antoniades LG, Kontoyannis DA, Moulopoulou DS. Clinical and laboratory parameters in adult diabetics with and without calcific shoulder periarthritis. Calcif Tissue Int. 1991;49:288–291.

[27] Tighe CB, Oakley WSJr. The prevalence of a diabetic condition and adhesive capsulitis of the shoulder. Southern Med J. 2008;101: 591–595.

[28] Griggs SM, Ahn A, Green A. Idiopathic adhesive capsulitis. A prospective functional outcome study of nonoperative treatment. J Bone Joint Surg Am. 2000;82:1398–1407.

[29] Del Rosso A, Cerinic MM, De Giorgio F, Minari C, Rotella CM, Seghieri G. Rheumatological manifestations in diabetes mellitus. Curr Diabetes Rev. 2006;2:455–466.

[30] Zreik NH, Malik RA, Charalambous CP. Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. MusclesLigaments Tendons J. 2016;6:26–34.

[31] Rai SK, Kashid M, Chakrabarty B, Upreti V, Shaki O. Is it necessary to screen patient with adhesive capsulitis of shoulder for diabetes mellitus? J Family Med PrimCare. 2019;8:2927–2932.

[32] Sung CM, Jung TS, Park HB. Are serum lipids involved in primary frozen shoulder? A case-control study. J Bone Joint Surg Am. 2014;96: 1828–1833.

[33] D'Orsi GM, Via AG, Frizziero A, Oliva F. Treatment of adhesive capsulitis: a review. Muscles Ligaments Tendons J. 2012;2:70–78.

[34] Warner JJ, Allen A, Marks PH, Wong P. Arthroscopic release for chronic, refractory adhesive capsulitis of the shoulder. J Bone Joint Surg Am. 1996;78:1808–1816.

[35] Saremi H, Hakhamaneshi E, Rabiei MA. Percutaneous release of trigger fingers: comparing multiple digits with single digit involvement. Arch Bone Joint Surg. 2016;4:224–227.

[36] Guerini H, Pessis E, Theumann N, et al. Sonographic appearance of trigger fingers. J Ultrasound Med. 2008;27:1407–1413.

[37] Fitzgerald BT, Setty A, Mudgal CS. Gout affecting the hand and wrist. J Am Orthop Surg. 2007;15:625–635.

[38] Abate M, Schiavone C, Salini V, Andia I. Management of limited joint mobility in diabetic patients. Diabetes Metab Syndr Obes. 2013;6: 197–207.
[39] Brown E, Genoway KA. Impact of diabetes on outcomes in hand surgery. J Hand Surg. 2011;36:2067–2072.
[40] Makkouk AH, Oetgen ME, Swigart CR, Dodds SD. Trigger finger: etiology, evaluation, and treatment. Cur Rev Musculoskelet Med. 2008;1:92–96.
[41] Grandizio LC, Beck JD, Rutter MR, Graham J, Klena JC. The incidence of trigger digit after carpal tunnel release in diabetic and nondiabetic patients. J Hand Surg. 2014;39:280–285.
[42] Sibbitt WLJr, Eaton RP. Corticosteroid responsive tenosynovitis is a common pathway for limited joint mobility in the diabetic hand. J Rheumatol. 1997;24:931–936.
[43] Dala-Ali BM, Nakhdjevani A, Lloyd MA, Schreuder FB. The efficacy of steroid injection in the treatment of trigger finger. Clin Orthop Surg. 2012;4:263–268.
[44] Akhtar S, Bradley MJ, Quinton DN, Burke FD. Management and referral for trigger finger/thumb. BMJ. 2005;331:30–33.
[45] Lapegue F, Andre A, Meyrignac O, et al. US-guided percutaneous release of the trigger finger by using a 21-gauge needle: a prospective study of 60 cases. Radiology. 2016;280:493–499.
[46] Patel MR, Bassini L. Trigger fingers and thumb: when to splint, inject, or operate. J Hand Surg. 1992;17:110–113.
[47] Sbernardori MC, Mazzarello V, Tranquilli-Leali P. Scanning electron microscopic findings of the gliding surface of the A1 pulley in trigger fingers and thumbs. J Hand Surg Eur Vol. 2007;32:384–387.