The advances in diagnostic modalities of disorders of the long head of the biceps tendon – review

Postępy w diagnostyce schorzeń ścięgna głowy mięśnia dwugłowego ramienia – praca poglądowa

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Abstract

The broad spectrum of disorders of the long head of the biceps (LHBT) can be divided into 3 main categories: inflammatory reactions, degenerative conditions, and instability. More than 90% of patients with LHB tendinopathy have an additional shoulder pathology. Thus, a detailed radiological and histopathological evaluation is crucial for a complete and accurate diagnosis. The pathology of the LHBT starts from its ultrastructure with the alterations of both tenocytes and extracellular matrix. Furthermore, this is reflected in the light microscopy investigation, with marginal inflammatory response. The ultrasonography is useful in the detection of LHBT instability, hypoechogenic areas, increased tendon diameter, and dynamic examination of the tendon. A standard non-contrast magnetic resonance imaging (MRI) of the shoulder is a useful tool in the diagnostics of advance tendinopathy; however, it is essentially limited in the detection of partial tears. Based on current knowledge, ultrasound and MRI diagnostics modalities should be simultaneously used in the examination of LHBT disorders.

Streszczenie

Schorzenia ścięgna mięśnia dwugłowego ramienia dzielą się na 3 główne kategorie: zapalenie, zmiany degeneracyjne oraz niestabilność ścięgna. Ponad 90% pacjentów z patologią ścięgna bicepsa ma dodatkowe uszkodzenia w obrębie barku, dlatego wnikliwa ocena radiologiczna oraz badanie histopatologiczne są krytyczne dla celnej diagnozy. Patologia ścięgna bicepsa ma podłoże w ultrastrukturze ścięgna, obecne są zmiany zarówno w tenocytach, jak i macierzy zewnątrzkomórkowej. Ponadto zmiany te mają odbicie w mikroskopii świetlnej, w której obserwujemy marginalny naciek zapalny. Ultrasonografia jest użytecznym narzędziem w wykrywaniu niestabilności ścięgna bicepsa, wykrywaniu obszarów hipoechogenicznych, zwiększonego wymiaru ścięgna oraz dynamicznym badaniu ścięgna. Badanie rezonansu magnetycznego barku jest przydatne do oceny zaawansowanej patologii ścięgna bicepsa, jednak ocena uszkodzeń częściowych jest utrudniona. Zgodnie z najnowszymi doniesieniami ultrasonografia oraz badanie rezonansu magnetycznego powinny być jednocześnie stosowane w ocenie patologii ścięgna bicepsa.

Introduction

Disorders of the long head of the biceps tendon (LHBT) are a common source of pain in the anterior shoulder area. They are usually localized at the level of the bicipital groove on the humerus and below towards the elbow [1–3]. LHBT can be divided into intra- and extra-articular portions, which exposes it to various loading patterns [3]. Nevertheless, the intra-articular part is more prone to friction and compress-

ing forces, causing a microtrauma, which initially leads to inflammation with subsequent development of the degeneration process, known clinically as tendinopathy. For this reason, this part of the tendon is more often described in the literature [1]. Moreover, more than 90% of patients with LHB tendinopathy have an additional shoulder pathology. It includes concomitant rotator cuff tears (RCTs), acromioclavicular joint (ACJ) disorders, superior labrum anterior to

posterior injuries (SLAP), omarthrosis, and subacromial impingement [4–6]. Thus, a detailed medical history of a patient, physical examination, and radiological and histopathological evaluation are crucial for a complete and accurate diagnosis. Mechanical forces and extreme loads are the main causes of widespread biceps tendon pathology. Considering the plague of concomitant RCTs, the advances in LHBT diagnostics are receiving more and more attention nowadays.

The broad spectrum of LHBT disorders can be divided into 3 main categories: inflammatory reactions, degenerative conditions, and instability of the biceps tendon. Clinically, all of them manifest with anterior shoulder pain. Although considering the treatment strategy classification of a disorder as belonging to a certain category may be helpful, from the diagnostic perspective, there is significant overlap among these pathologies. We can clearly say that there is a continuum of disease, starting with acute inflammation and ending with tendinous structure degeneration. The pathomechanism of repetitive traction and friction forces during glenohumeral joint rotation results in short-term inflammation, which in the long-term leads to instability and further tears with degeneration [7, 8].

According to our knowledge, there are no reports reviewing the various pathological alterations observed in LHBT disorders, especially from the ultrastructural and microscopic point of view. Moreover, the diagnostic imaging of LHBT disorders, such as ultrasounds (US) and magnetic resonance (MRI) methods, are usually not comprehensively described in the literature. We believe that an update of the actual knowledge on biceps tendon pathology will improve the choice of treatment strategies.

This review aimed to reveal the current state of knowledge in the diagnostics of the broad spectrum of LHBT disorders, with special reference to the following: microscopic examination, ultrasound, and magnetic resonance imaging in biceps pathology.

The current state of knowledge considering the histopathology of LHBT

The current approach to tendon pathology assumes the presence of abundant degeneration in tendinous tissue structure [9]. From the microscopic point of view, a degenerative process of LHBT is similar to changes observed in the Achilles tendon, patellar tendon, extensor carpi radialis tendon, and rotator cuff tendons [10]. Tendinopathy is characterized by impaired healing of the extracellular matrix (ECM) in the absence of inflammatory cells, altered proteoglycan content, chaotic remodelling of collagen architecture, and increased collagen type III level [3]. The exact aetiology of tendinopathy remains elusive. However, some reports point to abnormalities in the mechanobiological interactions between the tenocytes and the ECM as a possible reason for the chaotic production of tissue matrix [11–13]. Moreover, the exact molecular mediators and signals have not yet been discovered. However, increased activity of, e.g., metalloproteinases (MMP-1, -2, and -13) is often observed in tendinopathy [14–16].

The broad spectrum of LHBT disorders includes tendon inflammation, degeneration, and instability, but it is a continuum of disease, as was presented in the introduction. Tendinitis of the LHB is an acute inflammation of the tendon and its synovium. Commonly its symptoms occur at the level where it is constrained within the bicipital groove of the humerus [6, 17]. The disorder is usually transient and results in complete healing of tendinous tissue, similarly as in the phase of inflammation during connective tissue healing. However, in a minority of cases there is a possibility of tissue degeneration (tendinopathy) as an effect of impaired regeneration.

It must be emphasized that the pathology of LHBT starts from its ultrastructure, mainly with the altered morphology of tenocytes and disruption of the ECM architecture [1]. Franchi *et al.* revealed, based on transmission electron microscopy of healthy tendons, the highly organized and hierarchical structure of the ECM with a modest population of tenocytes with the fusiform morphology [18]. The ultrastructure of the pathological biceps tendon is characterized by advanced tenocytes alterations. They become randomly scattered in the ECM, while their shape is deformed. Moreover, they can be enlarged due to the chaotic production of the matrix. The described morphology can be connected with a disturbed mechanotransduction process and further impaired tendon adaptation to the new load, creating a vicious circle. The transformation of the tenocytes into a more round or oval shape could be evidence of cartilage metaplasia. In turn, this could be a form of adaptation to a new load. The intra-articular portion of the LHBT is exposed to friction, compression forces, and traction, similar to those observed in enthesis. Zabrzyński *et al.* revealed some advanced changes in the tenocyte morphology based on an electron microscope study of the LHBT [1]. Furthermore, Maffulli *et al.* showed that the metaplasia of fibroblasts into chondrocytes is characteristic for advanced tendinopathy [19]. Moreover, the apoptotic features in tenocytes were observed in a few studies [1, 20].

Regarding the ECM alterations, a shift in the collagenous/non-collagenous ratio with the accumulation of ground substance between collagen fibrils is observed [1, 21]. Moreover, there is a decrease in the number of crimps – these are elements responsible for the mechanical properties of tendons that act as a shock absorber [1, 22]. The diameter of the collagen fibrils seen with a transmission electron microscope (TEM) is also reduced. Furthermore, the fibrils tend

to create a homogenous mass, which does not show adequate mechanical properties.

In the case of light microscopy, the biceps tendon pathology focuses mainly on tenocyte alterations, ECM disruption, and neovascularization (Figure 1). Moreover, some authors described a marginal inflammatory response. The total tenocyte population is usually increased. The cells are enlarged and more oval in shape. The nuclei swell while the cytoplasm marginalizes. Moreover, ECM destruction results from increased expression of MMP 1, 3, and 9 [23]. MMPs play an important role in the matrix homeostasis, where they degrade collagen and proteoglycans [23].

The ECM alterations mainly involve disrupted collagen architecture, ground substance deposition with myxoid degeneration, and increased collagen III type/I type ratio [3, 24]. The characteristic crimps or wavy configuration of the collagen fibres disappear. Moreover, the fibres are separated by the foci of the non-collagenous matrix. Shishani *et al.* identified a myxoid degeneration in all specimens with deposition of the non-collagenous matrix between collagen fibres [2]. The authors observed increased ground substance accumulation [2, 3]. In turn, Mazzocca *et al.* found a significantly higher number of proteoglycans in the intra-articular portions of the LHBT than in the extra-articular part and control group [25]. The altered ratio of collagen III to collagen I was presented in several studies. Moreover, the intra-articular bicep part, compared to the extra-articular one, was characterized by increased type III collagen [3, 25].

Regarding neovascularization, the groups of capillaries distributed chaotically in the ECM were characteristic for the LHBT pathology [26]. Shishani *et al.* presented newly formed capillaries invasion in the whole examined population. On the other hand, Zabrzyński *et al.* observed neovascularization in 78% of investigated specimens [27]. This data shows the strong association between LHBT pathology and the process of neovascularization.

It is interesting that some tendons, such as the Achilles tendon, rotator cuff tendons, and tibialis posterior tendon, contain hypovascular regions, called the critical zones [28]. Similarly, in the LHBT, a hypovascular region was found about 1–3 cm from its origin [4]. Despite the poorly vascularized regions, in the case of the LHBT disorders, there is an abundant angiofibrous response. However, this phenomenon is reduced in heavy smokers. Moreover, there is a positive correlation between the smoking indexes and the intensity of new vessel formation in the tendinous tissue of the biceps [21]. The inflammatory process with a marginal infiltration of the inflammatory cells in the case of LHB tendinopathy was revealed only by a few authors [2, 26, 29, 30]. Shishani *et al.* observed the evidence of mild chronic inflammation in 2 of 26 specimens [2]. The authors also analysed the te-
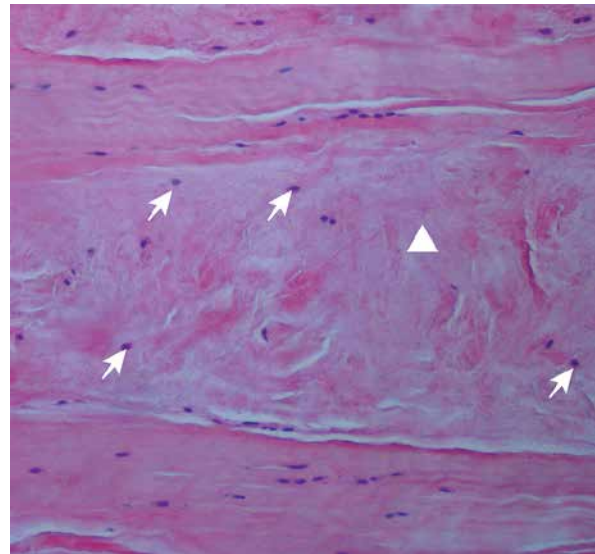

Figure 1. Microscopic slide of pathological LHBT presents the accumulation of non-collagenous ECM (arrowhead) and increased population of altered tenocytes (arrows)

nosynovium and revealed synovial proliferation without any sign of acute or chronic inflammation in the studied material. Thus, they linked the clinically observed pain with the degenerative process, not inflammation. Zabrzyński *et al.* showed a modest infiltration of inflammatory cells around blood vessels in 3 samples [26]. Moreover, Longo *et al.* revealed an absence of inflammatory changes observed microscopically, connecting this fact with impaired healing response of the tendon [30].

Recent advances in sonography of the LHBT

The use of US in the diagnostics of musculoskeletal (MSK) disorders, especially shoulder examination, started in the 1980s, and its use is still increasing. Ultrasound modality in LHBT pathology examination is presented in Table 1 [7, 8, 31–40]. Ultrasonography is a simple, fast, cost-effective, radiation-free, and noninvasive diagnostic method for the evaluation of the shoulder [29]. Moreover, US can potentially be used for dynamic examination and is characterized by high sensitivity and specificity [31, 32]. However, it must be emphasized that it is a highly operator-dependent imaging modality.

Lewis *et al.* revealed that the best diagnostic tool for detecting LHB pathology is US [32]. The sensitivity of US in detecting the LHBT pathology is between 50% and 96%, while its specificity reaches 98–100% compared to MR arthrography and arthroscopy, retrospectively [31, 33, 34]. However, some authors proved the relatively low sensitivity of the US in biceps pathology detection. Chen *et al.* showed sensitivity of US in 39.1% of patients with isolated LHBT pathology

Table 1. Summarization of ultrasound (US) modality in LHBT pathology examination

Authors	US
Chen *et al.*, Lewis *et al.*	Dynamic examination of the LHBT
Chen *et al.*, Armstrong *et al.*, Le Corroller *et al.*	Sensitivity in detecting the LHBT pathology is between 50–96%, specificity is 98–100%
Chen *et al.*	Sensitivity in detecting the LHBT pathology is 55.3% with coexisting rotator cuff injury
Skendzel *et al.*	Sensitivity in the healthy biceps tendon detection is 90%
Zabrzyński *et al.*, Ohberg *et al.*	Power Doppler function in the assessment of hyperaemia, inflammation, and the neovascularization process
Chen *et al.*	Instead of MRI in THE presence of: metal implants, pacemakers, claustrophobia
Zabrzyński *et al.*	Increased transverse LHBT diameter (> 5 mm) in pathology
Zabrzyński *et al.*, Huang *et al.*	Hypoechogenic areas in the tendon structure are a manifestation of pathology
Ptasznik *et al.*, Huang *et al.*	Increased transverse LHBT diameter ≥ 4.6 mm for women and ≥ 5.5 mm for men as pathology
Zabrzyński *et al.*, Chen *et al.*, Armstrong *et al.*	Instability of the biceps tendon: specificity 100% and sensitivity 96%
Zabrzyński *et al.*, Zappia *et al.*, Kao *et al.*, Ricci *et al.*	Ultrasound in the detection of microinstability of LHBT

and in 55.3% with coexisting rotator cuff injury [31]. On the other hand, Skendzel *et al.* revealed that the sensitivity of US in healthy biceps tendon detection is very high (90%) [35]. In their study of 66 patients examined sonographically, none of those identified as healthy biceps tendons showed signs of pathology during the shoulder arthroscopy. Another advantage of US is its possible use to provide guided injections of steroids, hyaluronic acid, or PRP into the synovial sheath. Moreover, with power Doppler signal processing, it is a useful tool in the assessment of hyperaemia, inflammation, and the neovascularization process [8, 36, 41]. Additionally, in some circumstances, such as the presence of metal implants, pacemakers, and claustrophobia, MRI is prohibited and US can be introduced instead [31].

The most common pathological changes observed in US of the biceps tendon are hypoechogenicity of the tendinous structure, the presence of fluid in the area of the bicipital groove, thickening of the tendon with increased transverse diameter, calcification, pathological vascularization, and dislocation of the tendon or subluxation during dynamic examination [8, 31, 38] (Figure 2). Zabrzyński *et al.* in their study of the biceps pathology revealed an increased transverse LHBT diameter (> 5 mm) measured in the bicipital groove in all included subjects. Moreover, there were hypoechogenic areas in the tendon structure in 95% of the population. All of the cases were assessed arthroscopically and confirmed microscopically [8]. The authors concluded that the distortion in the highly organized tendon structure can result in decreased

reflection of ultrasound waves within the tendon during sonographic examination, which can be visible as hypoechoic areas [8]. Moreover, they showed an important limitation of the sonographic examination because it does not allow for the visualization of the entire intracapsular part of the LHBT.

Huang and Wang examined a total of 336 shoulder pain patients with suspected biceps tendinitis.

The authors evaluated the tissue echogenicity using greyscale pixel data of the range of interest (ROI) in both the transverse and longitudinal views of the biceps [38]. Moreover, they determined a certain threshold when the echogenicity counted as ROI values in different planes of examination corresponded to LHB tendinopathy. Various authors set a limit of the maximal transverse diameter of the biceps tendon examined sonographically as ≥ 4.6 mm for women and ≥ 5.5 mm for men [8, 31, 37, 38]. Increased diameter of the tendon is a common pathology, presented by numerous authors. Increased thickness is classified as a pathological abnormality and is often linked with local oedema, inflammation, and degeneration of tendinous structure [8, 42]. On the other hand, the increased size and diameter of the tendon may result from age and sports activities [37, 38].

The sonographic examination was presented as a very precise modality to identify subluxation, dislocation, and instability of the biceps tendon, with high specificity (100%) and sensitivity (96%) [8, 31, 33]. Overall the presence of tendinopathy is often linked with overuse or instability of the tendon [6]. The LHBT instability with dislocation or subluxation can
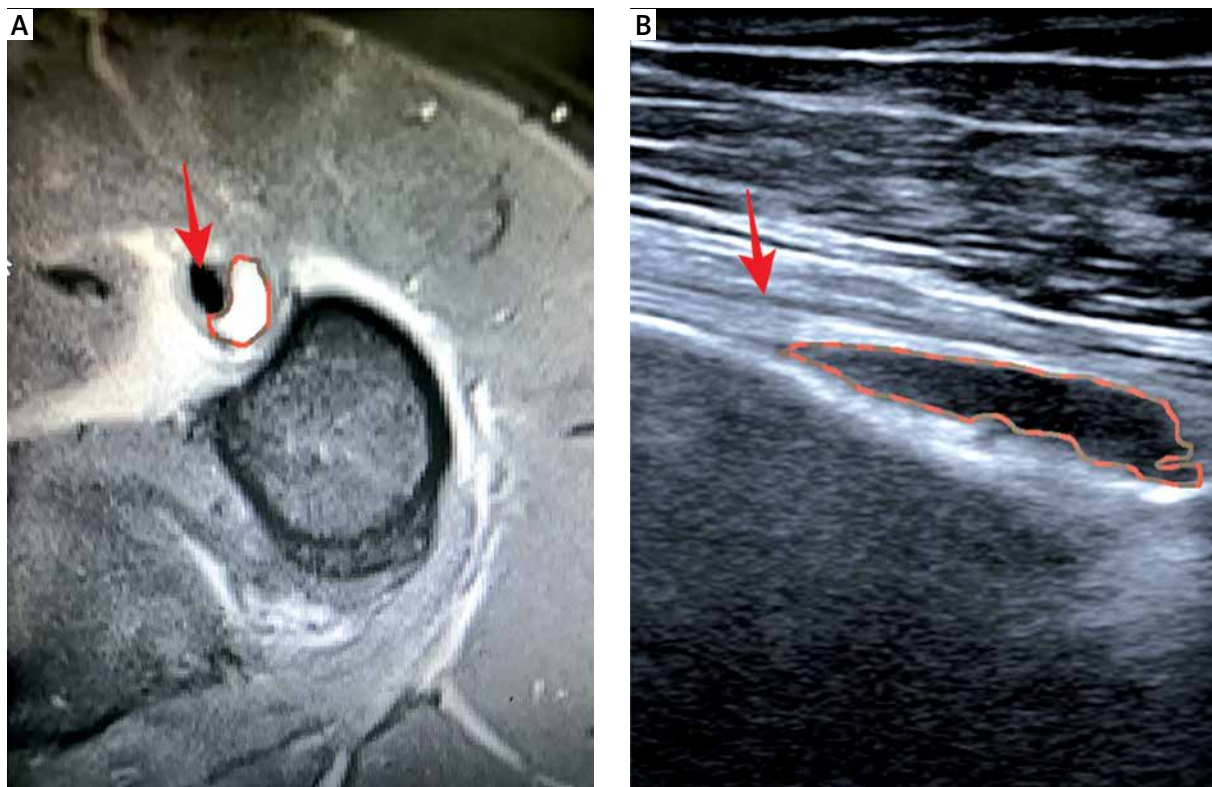
Figure 2. A – MRI transverse scan of the LHBT (arrow) with accumulation of fluid inside the bicipital groove (orange line). **B** – US longitudinal scans of the LHBT (arrow) with hypoechoic fluid below the tendon (orange line)

occur dynamically during both external and internal rotation of the arm. However, the advanced instability may cause a deformation of the bicipital groove, with irregularities on the bony walls, leading to a full-thickness tear [8]. It was suggested that instability can be hidden and occur without dislocation beside the bicipital groove [7, 8, 39]. Microinstability, leading unavoidably to gradual development of tendinopathy, may not be initially detected in a sonographic examination. Furthermore, it can gradually progress, with concomitant supraspinatus or subscapularis tendon tears, to dynamic subluxation or dislocation [40].

MRI evaluation of the LHBT

MRI is considered a gold standard for the examination of soft tissue injuries. However, it was presented that in the case of the biceps tendon it has a relatively low sensitivity [43, 44]. Tendon degeneration diagnosis in MRI is based on changes in tendon diameter and alterations in the signal of its structure. However, the changes can be subtle and recognized as an artifact or simply missed [45]. The accuracy of MRI in the diagnose of partial LHBT tears and inflammation has been questioned by numerous authors [46]. Mohtadi *et al.* observed a poor correlation between MRI findings and arthroscopic examination, especially for

LHBT tears [47]. Moreover, low sensitivity for detecting partial-thickness tears and LHBT instability was shown by Carr *et al.* They noted a 27% sensitivity and 86% specificity for detecting partial LHB tears by MRI scan, while complete tears were diagnosed in 54% [44]. Dubrow *et al.* revealed similar results, in which the MRI modality identified 43.9% of patients as having a pathologic lesion of the biceps despite the fact that the arthroscopy confirmed tears in 89.4% [48].

Mucoid degeneration found in abnormal tendinous tissue is characterized by hyperintense signal compared with fatty infiltration in T2-weighted fat-saturated images, and hyperintense compared with magic-angle artifacts in proton density-weighted fat-saturated images. Buck *et al.* compared the histological findings with MRI alterations showing the agreement between these two methods [45]. Pathological changes in tendinous tissue, such as accumulation of myxoid substances and collagen fibre disruption, unavoidably lead to microtears, partial-tears, and subsequently to complete tears of the bicep tendon. On the other hand, the exact connection between intra-tendinous alterations and MRI findings is not entirely understood.

Obese patients can be misdiagnosed with ultrasound. In such cases, MRI is advised [31]. MRI diagnostics also prevails in patients with other concurrent injuries of the shoulder, to plan surgery, or in those

Table 2. Summary of MRI modality in LHBT pathology examination

Authors	MRI
Khazzam *et al.*, Carr *et al.*, Dubrow *et al.*	LHBT pathology: Sensitivity 27.7–56.3% Specificity 84.2–98%
Mohtadi *et al.*	Poor correlation between MRI findings and arthroscopic examination for LHBT tears
Buck *et al.*	Correlation between histopathology (mucoid degeneration) and MRI
Chen *et al.*	Useful in obese patients
Ahrens *et al.*, Lewis *et al.*, Zanetti *et al.*	MRI arthrogram useful in LHBT + SLAP lesions (sensitivity 89–92%, specificity 56–81%)

with SLAP lesions. Alternatively, the MR arthrogram may be used if the LHBT pathology is connected with SLAP lesions, with sensitivity of 89–92% and specificity of 56–81% [6, 46, 49].

The MRI modality in the LHBT pathology examination is summarized in Table 2 [6, 31, 44–50].

Conclusions

The pathology of the LHBT starts from its ultrastructure with alterations of both tenocytes as well as ECM. Furthermore, this is reflected in light microscopy investigations, where the pathology is focused on the increased tenocyte population, increased neovascularization process, disorganization of the collagenous ECM, and accumulation of the non-collagenous elements. It should be accented that there is a marginal inflammatory response. The ultrasound modality is useful in the detection of LHBT instability, dynamic examination the tendon, the examination of hypoechogenic areas, and increased tendon diameter. Moreover, the power Doppler function allows the detection of extensive vascularization and early inflammation. A standard non-contrast MRI of the shoulder is essentially limited in LHBT pathology detection, specifically in the case of partial tears. However, the MRI modality with contrast agents is useful in LHBT and SLAP injury detection. Based on the current knowledge, ultrasound and MRI diagnostics modalities should be simultaneously used in the examination of LHBT disorders.

Conflict of interest

The authors declare no conflict of interest.

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