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ACTA ORTHOPAEDICA ET TRAUMATOLOGICA HELLENICA

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"Acta Orthopaedic Et Traumatologica Hellenica" is the official journal of the Hellenic Association of Orthopaedic Surgery and Traumatology, first published in 1948. This revived edition of Acta Orthopaedic Et Traumatologica Hellenica, published in English, aspires to promote scientific knowledge in Orthopaedics and Traumatology worldwide. It is a peer-reviewed Journal, aiming at raising the profile of current evidence-based Orthopaedic practice and at improving the scientific multidisciplinary dialogue. Acta Orthopaedic Et Traumatologica Hellenica presents clinically pertinent, original research and timely review articles. It is open to International authors and readers and offers a compact forum of communication to Orthopaedic Surgeons and related science specialists.

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- **Letters to the editor:** Communication to the editor is welcome and will be published if they offer pertinent and/ or constructive comment on articles published in the Hellenic Journal of Radiology. Letters are published at the discretion of the Editorial team and should be received within three months after on-line publication of an article. Following acceptance, letters will be sent to authors for response. Letter communications should include text of no more than 500 words, up to 2 figures and 10 references, without any abstract or keywords and a maximum of 3 authors.

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Patient anonymity should be ensured. All identifying data (name, identification numbers, initials) must be removed from text, images and tables. If it is mandatory for a patient's face to be included in the manuscript, the eyes should be sufficiently masked. If there is a possibility that a patient may be identified from a photograph or relevant legend and text, the patient's written consent should be submitted.

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■ Journal article:

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or

Papaioannou NA, Triantafyllopoulos IK, Khaldi L, et al. Effect of calcitonin in early and late stages of experimentally induced osteoarthritis. A histomorphometric study. *Osteoarthritis Cartilage* 2007; 15(4): 386-95.

■ Book chapters:

Triantafyllopoulos IK, Papaioannou NA. The Effect of Pharmacological Agents on the Bone-Implant Interface. In: Karachalios Th. (ed). Bone-Implant Interface in Orthopaedic Surgery. Springer – Verlag, London 2014, pp 221-237.

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12. Review of manuscripts

Acceptance of manuscripts for publication is decided by the Editor, based on the results of peer review. Authors need to make proof corrections within 72 hours upon pdf supplied, check the integrity of the text, accept any grammar or spelling changes and check if all the Tables and Figures are included and properly numbered. Once the publication is online, no further changes can be made. Further changes can only be published in form of Erratum.

ΓΡΑΜΜΑ ΤΟΥ ΕΚΔΟΤΗ

Επανεκκίνηση...

Ν. Α. Παπαϊωάννου
Πρόεδρος Εκδοτικής Επιτροπής

Το 1948, έναν χρόνο μετά την ίδρυση της Ελληνικής Εταιρείας Χειρουργικής και Τραυματολογίας (ΕΕΧΟΤ), εκδίδεται το 1ο Τεύχος του περιοδικού της Εταιρείας με την επωνυμία «Δελτίον της ΕΕΧΟΤ». Το Δελτίο αυτό που είχε περισσότερο ενημερωτικό χαρακτήρα εξελίχθηκε στο επιστημονικό περιοδικό της Εταιρείας «***Acta Orthopaedica et Traumatologica Hellenica***» με περιεχόμενο δημοσιεύσεις από το κλινικό και ερευνητικό έργο των Ελλήνων Ορθοπαιδικών. Η αρίθμηση των τευχών διατηρήθηκε μέχρι και σήμερα η ίδια με έναρξή της εκείνο το ιστορικό πρώτο τεύχος του Δελτίου του 1948. Έτσι αυτή την χρονιά θα εκδοθούν τα Τεύχη του 68ου ετήσιου Τόμου.

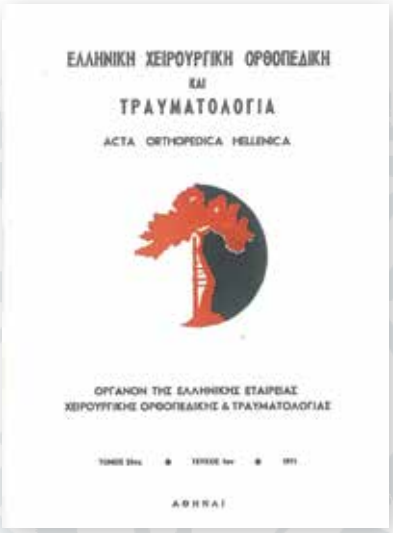
Ως πρώτος υπεύθυνος έκδοσης του περιοδικού αναφέρεται ο Αθ. Κονταργύρης (1952) και ακολούθησαν οι Β. Σουρμελής (1960), Γ. Βαρούχας (1961), Κ. Ηλιόπουλος (1973), Σ. Θεοδώρου (1981), Β. Πετρόπουλος (1986), Γ. Παπαχρήστου (1993), Ι. Γερμάνης (1999), Β. Τσεμάνης (1999), Θ. Ξενάκης (2001), Δ. Κορρές (2012), ενώ από το 2014 έχει αναλάβει η παρούσα εκδοτική επιτροπή.

Διαχρονικά, οι εκάστοτε εκδοτικές επιτροπές, παρακολουθώντας τις τάσεις της εποχής τους, προχώρησαν σε βελτιώσεις της εμφάνισης και του περιεχομένου του περιοδικού, ενώ τα τελευταία χρόνια καθιερώθηκε η δημοσίευση των εργασιών και στα ελληνικά και στα αγγλικά.

Μόνιμη επιθυμία όλων ήταν, και παραμένει, η διασύνδεση και ένταξη του περιοδικού μας στις διεθνείς βάσεις δεδομένων της βιβλιογραφίας (PUBMED, SCOPUS κ.λπ.). Έγιναν πολλές προσπάθειες προς αυτήν την κατεύθυνση και παλαιότερα και πρόσφατα, οι οποίες όμως για διάφορους λόγους δεν τελεσφόρησαν.

Από το παρόν τεύχος, επιχειρείται άλλη μια φορά, η αναβάθμιση και ο εκσυγχρονισμός του περιοδικού, με μεταβολές στην εμφάνιση, στη σελιδοποίηση και στην έγχρωμη εκτύπωση. Η πιο σημαντική όμως μεταβολή αφορά την απόφαση να εκδίδεται πλέον μόνο στα αγγλικά και παράλληλα να υπάρχει, εκτός από την έντυπη μορφή, η ηλεκτρονική έκδοσή του μέσα από την δημιουργία μιας ανεξάρτητης ιστοσελίδας του περιοδικού. Ο σχεδιασμός αυτός προσβλέπει στην αξιοποίηση του διαδικτύου ώστε να αποκτήσουν οι συγγραφείς του περιοδικού μας αναγνωσιμότητα στην διεθνή επιστημονική κοινότητα. Η επιτυχία αυτού του εγχειρήματος, εκτός από τις δικές μας προσπάθειες, χρειάζεται χρόνο και πρωτίστως την δική σας υποστήριξη και εμπιστοσύνη.

Covers



Η εξέλιξη των εξωφύλλων στον χρόνο
Covers evolution throughout the years

EDITORIAL

Restarting

N. A. Papaioannou, *Chief Editor*

Back in 1948, the Hellenic Association of Orthopaedic Surgery and Traumatology (HAOST) was founded. One year later, the official journal of the HAOST was officially published under the name "HAOST bulletin". This bulletin had initially an informative character for the Greek Orthopaedic community. Gradually, it took its final form as the scientific journal of HAOST publishing the clinical and research work of the Greek Orthopaedic Surgeons. The name of the journal was and still is "***Acta Orthopaedica et Traumatologica Hellenica***". The numbering of the volumes and issues of the journal is kept until today so this year the issues of the 68th annual volume will be published.

The first editor of the journal was Ath. Kontargyris (1952) and V. Sourmelis (1960), G. Varouxas (1961), K. Heliopoulos (1973), S. Theodorou (1981), V. Petropoulos (1986), G. Papachristou (1993) I. Germanis (1999) V. Tsemanis (1999), Th. Xenakis (2001), D. Korres (2012) were followed. Since 2014, I have the honour to be the editor of the journal.

During the past decades, the editorial boards of the journal followed the current publication models of their era and proceeded to innovative changes of the form of the journal. Furthermore, the last decade the journal publishes scientific papers written in bilingual texts in Greek and English language.

The will of the editorial boards was always the introduction of our journal to the international data bases and search engines such as Pubmed, Scopus, etc. Despite the great efforts and for several reasons this aim was never accomplished.

From this current issue, the new editorial board decided to make new amendments regarding the illustration and publication of the journal. The most important change is the decision to accept manuscripts written only in the English language. Furthermore, the journal will be published both in hard copies and in electronic form through its own new Internet site and platform. This innovation will allow the published articles to be shown on the Internet and their authors to expose their work and their names into the World Wide Web.

The achievement of our venture depends not only on our efforts but most of all on your contribution with clinical, research, review and case report works that you would constantly submit for publication. The time will tell if our dream for a strong and internationally recognized journal will finally come true…

ACTA
ORTHOPAEDICA ET TRAUMATOLOGICA HELLENICA

CONTENTS

VOLUME 68 | ISSUE 1 | JANUARY - MARCH 2017

MONOGRAPHY

Scapulothoracic disorders

Ioannis K. Triantafyllopoulos[1], Chrysa Argyrou[2]

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[2]*MD, Resident in Orthopaedics*

ABSTRACT

Scapulothoracic articulation disorders may cause significant malfunction of the shoulder girdle. Scapular winging, dyskinesia, crepitus and bursitis are different pathological entities associated with the scapulothoracic joint. Their pathogenesis is a combination of anatomic, posture, traumatic and neuromuscular alterations. In this monography, the causes, diagnosis and treatment of the scapulothoracic disorders will be discussed.

KEY WORDS: scapula; winging; dyskinesia; crepitus; bursitis

Introduction

The scapulothoracic articulation is essential in the kinesiology of the shoulder girdle. Few papers are referred to scapulothoracic disorders compared to glenohumeral and acromiclavicular joints of the shoulder girdle. Disorders associated with scapulothoracic joint are often poorly understood or difficult to diagnose. They are rather common in heavy manual workers as well as in water sport and overhead sport athletes, as the continuous and intense movement of upper limbs and trunk makes the area of the scapula the most functionally active site. The pathogenesis of scapulothoracic disorders involves anatomic and neuromuscular alterations that affect the biomechanics of the shoulder.

A. Surgical anatomy

Scapula is a thin bone which is the site of attachment of 17 muscles of trunk and upper limb and plays a key role in the coordinated movement of the upper limb (**Fig. 1**).

The muscles attached to scapula can be divided into three groups (**Table I**): (a) The scapulothoracic muscles adjust the scapulothoracic movement and include the major and minor rhomboids, the levator scapula, the anterior serratus, the trapezius, the omohyoid and the pectoralis minor. Conditions of these muscles present with winged scapula or scapulothoracic dyskinesia. (b) The scapulohumeral muscles provide functional strength to the humerus and include the deltoid, the long and short heads of biceps

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TABLE 1. *Muscles attached to scapula*

SCAPULOTHORACIC	SCAPULOHUMERAL	ROTATOR CUFF
1. Levator scapula	1. Long head of biceps brachii	1. Supraspinatus
2. Omohyoid	2. Short head of biceps brachii	2. Infraspinatus
3. Rhomboid major	3. Deltoid	3. Subscapularis
4. Rhomboid minor	4. Coracobrachialis	4. Teres minor
5. Serratus anterior	5. Teres major	
6. Trapezius	6. Long head of triceps brachii	
7. Pectoralis minor		

brachii, the coracobrachialis, the long head of the triceps brachii and the teres major. (c) The rotator cuff muscles control the movement of glenohumeral articulation and consist of the supraspinatus, infraspinatus, subscapularis and teres minor. Disorders of these muscles are common and compose a special subject of study not included in this issue.

B. Biomechanics

The interplay of 4 articulations (Sternoclavicular Joint, Acromioclavicular Joint, Scapulothoracic Joint and Glenohumeral Joint) of the shoulder complex, results in an coordinated movement pattern of the arm elevation. The involved movements at each joint are continuous, although occurring at various rates and at different phases of arm elevation. The movement of the scapula can be described by rotations in relation to the thorax. The scapula moves around a dorso-ventral axis, resulting in a rotation in the frontal plane. In this movement the glenoid cavity is turned cranially (upward rotation) or caudally (downward rotation). In the sagittal plane, around a latero-lateral axis the scapula rotates posteriorly (posterior tilting) or anteriorly (anterior tilting). External and internal rotation occurs around a cephalo-caudal (longitudinal) axis. The external rotation brings the glenoid cavity more into the frontal plane, whereas the internal rotation turns the glenoid cavity more to the sagittal plane.

At rest, the scapula is rotated 30° anteriorly in frontal plane. The inferior angle of the medial border of the scapula is also deviated 3° away from

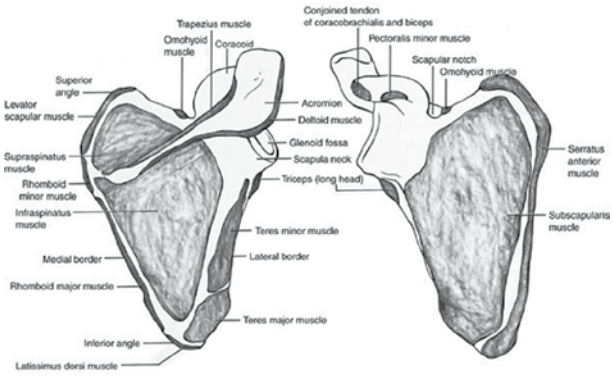

Fig. 1. *Muscular attachments to scapula*

the midline. When observing the lateral side of the scapula, there is an anterior inclination of 20° in the sagittal plane (**Fig. 2**).

Almost every upper limb movement includes elements of scapulothoracic and glenohumeral motion. In the first 30° of humeral abduction, most of the movement is provided by the glenohumeral joint. The rest 60° are equally distributed to glenohumeral and scapulothoracic joints. Overall, the glenohumeral to scapulothoracic ratio during abduction of the shoulder up to 90° is 2:1.

During abduction, in the scapular level, the point of rotation of the scapula moves so that for the first 0° - 30° the scapula rotates around its actual center. From 30° to 60° rotates around the glenoid fossa, leading to medial and superior displacement of the inferior angle of the scapula (**Fig. 3**). Moreover, during arm abduction, the coracoid process and the ac-

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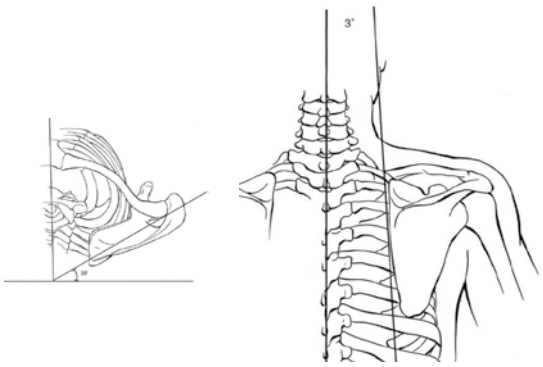

Fig. 2: *Stereoscopic position of scapula*

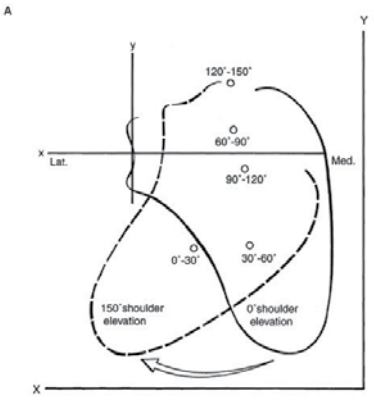

Fig. 3: *Movements of scapula*

romion move superiorly, reducing the sub-acromial impingement (**Fig. 4**).

C. Muscle function around the scapula

Activation of the upper fibers of trapezius, which insert at the lateral part of the scapular spine, the acromion and the lateral end of the clavicle, moves the scapula superiorly. This action is opposed by the gravity and the latissimus dorsi muscle, which acts as the main stabilizer of the scapula. Its action is reinforced by the inferior fibers of the anterior serratus, the pectoralis minor and the lower trapezius.

The upward rotation of the scapula begins with the activation of middle trapezius, which stabilizes the scapula by its attachment to the medial part of the scapular spine. At the 45° of scapular protraction the serratus anterior draws the inferior angle of the scapula outwards. The upper trapezius draws the lateral angle of the scapula superiorly, while the lower trapezius which inserts to the medial part of the scapular spine, draws the scapula downwards, leading to upward rotation (**Fig. 5**).

The downward rotators of the scapula are mainly the rhomboids and the levator scapula, which draw superiorly the medial border of the scapula, while the pectoralis minor, the lower part of pectoralis major and the latissimus dorsi stabilize the lateral border of the scapula (**Fig. 6**).

The anterior movement of the scapula is performed by the serratus anterior, pectoralis major and minor, as they move the scapula anteriorly and

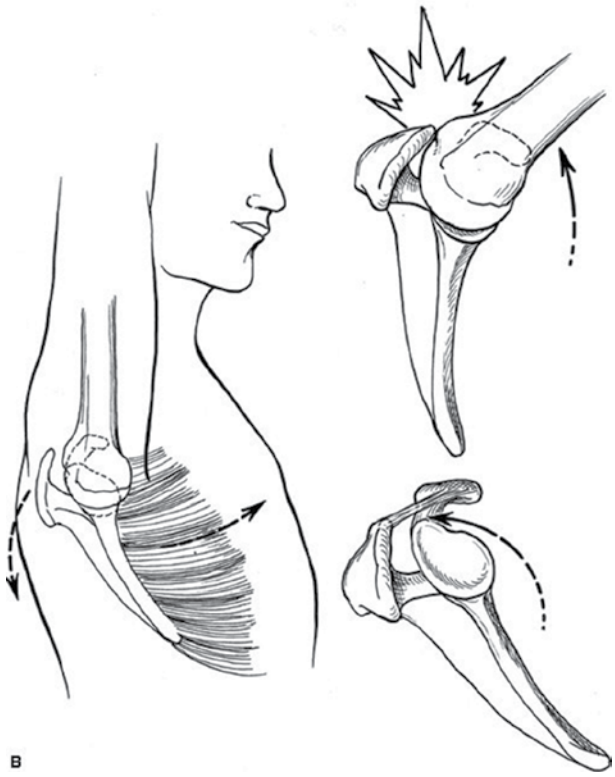

Fig. 4: *Scapular-humeral synergy*

outward. The posterior movement of the scapula is performed by the middle trapezius and rhomboids (**Fig. 7**).

D. Bursae of the scapula

The bursae of the scapula ensure the smooth scapulothoracic movement. There have been described

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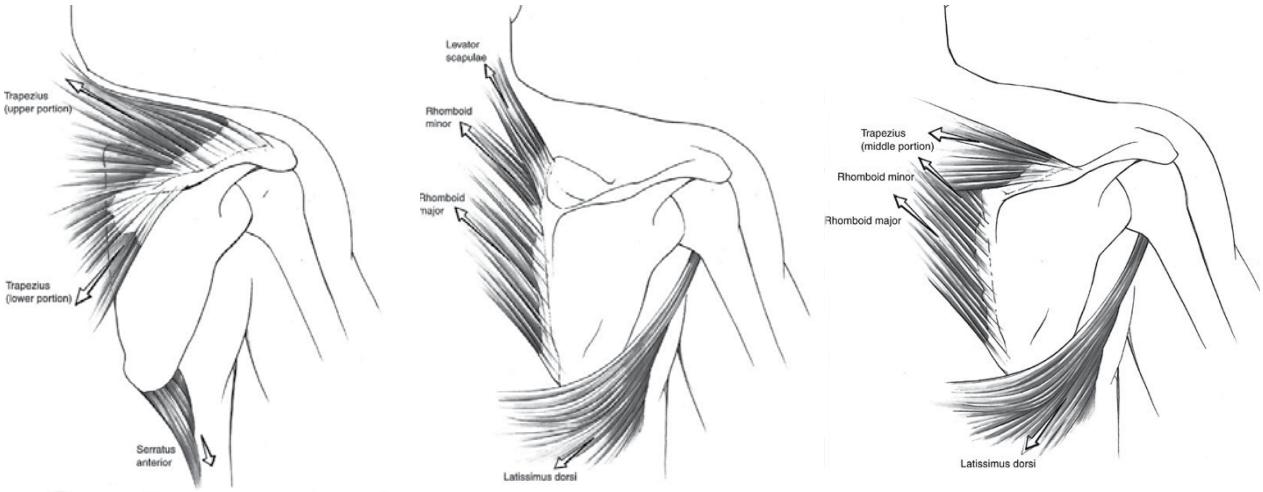

Fig. 5-7: *Muscular forces applied to the scapula*

2 main and 4 accessory bursae in this area (**Table 2**, **Fig. 8**). The first main bursa is located between the serratus anterior muscle and the thoracic wall. The second is located between the subscapularis and the serratus anterior muscles.

Bursitis of the scapulothoracic joint is clinically manifested in two locations: At the superior medial angle and the inferior angle of the scapula. At the inferior angle of the scapula, the inflamed bursa is located between the serratus anterior and the thoracic wall. Many names have been attributed to this bursa so far, like *infraserratus bursa, bursa mucosa serrata* etc. Initially, Codman believed that the bursa of the superior medial angle is located between the three first ribs and the scapula[1]. Finally, Von Grueber was the one who identified the bursa between the serratus anterior and the subscapularis muscle and named it *bursa mucosa angulae superioris scapulae*[2].

An accessory bursa is found in the triangular surface of the inner part of the scapular spine and below the trapezius. Codman believed that this bursa was responsible for the scapulothoracic crepitus and named it *trapezoid bursa*. Many authors believe that the accessory bursae developed in order to counteract the abnormal biomechanics of the scapulothoracic joint and this is the reason that are not steadily found at the same region or at the same tissue plane.

D. Scapulothoracic disorders

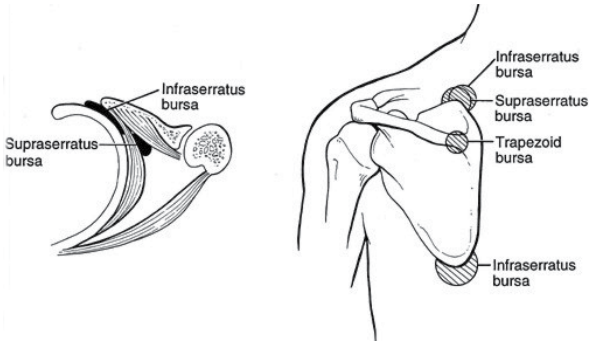

Fig. 8: *Bursae of scapula*

1. WINGED SCAPULA

Winged scapula is the most common disorder of the scapulothoracic articulation and various factors are implicated in its pathogenesis. It is classified to primary, secondary and voluntary (**Table 3**). **Primary** winging refers mainly to anatomic disorders that directly affect the joint. The **secondary** form is usually related to pathology of the glenohumeral joint. Finally, the **voluntary** form is rather rare and is attributed to psychological causes.

I. PRIMARY WINGED SCAPULA

A. Neurological disorders

1) Major and minor rhomboids palsy

This is a rare cause of winged scapula. These muscles are innervated by the dorsal scapular nerve (C5 root). This nerve passes deeply or through the le-

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TABLE 2. *Bursae of the scapula*

CLASSIFICATION	ANATOMIC SITE		INTERSPACE
Major/Anatomic		Below SA	SA-Thoracic wall
		Above SA	SA-Subscapularis
Minor/Ectopic	Superior Medial angle	Below SA	SA- Thoracic wall
		Above SA	SA-Subscapularis
	Inferior angle	Below SA	SA-Thoracic wall
	Scapular spine	Trapezius	Inner part of spine-T

SA: Serratus Anterior, T: Trapezius

vator scapula muscle and then reaches the rhomboids. Winging of the scapula can be the result of C5 pathology or damage to the dorsal scapular nerve.

Patients with weakening of the rhomboids are presented with pain along the medial border of the scapula. The winging of the scapula is not very obvious at rest. It is possible, though, to resemble with the winged scapula caused by the trapezius weakening with depression of the scapula, outward displacement and outward rotation of the inferior angle. Furthermore, atrophy along the medial border of the scapula may exist. During arm abduction, the inferior angle of the scapula is drawn downwards and outwards due to the action of anterior serratus. The winging becomes even more evident when the arm is slowly adducted starting from the position of anterior flexion, so that the inferior angle of the scapula is drawn outwards and dorsally while hands are lying on the hips. Electromyography and other tests of neurological conduction can be very helpful in the differential diagnosis and the distinction from other neurological disorders.

Treatment is based on the strengthening of the trapezius. If symptoms persist and conservative measures fail, patients can benefit by the ***Dickson procedure*** [3]. In this procedure, two cylindrical grafts of the fascia lata connect the lower part of the medial border of the scapula with the paraspinal muscles and the inferior angle of the scapula with fibers of the latissimus dorsi. This technique stabilizes the scapula and partially inhibits the high thoracic scoliosis that can emerge due to the paralysis of the rhomboids and the levator of scapula. However, there is a possibility that the grafts will eventually become loose and elongated.

TABLE 3. *Classification of winged scapula*

I. PRIMARY
A. Neurological causes
1. Long thoracic nerve: Anterior serratus palsy
2. Accessory nerve: Trapezius palsy
3. Dorsal scapular nerve: Rhomboids palsy
B. Osseous causes
1. Osteochondroma
2. Poor fracture healing
C. Soft tissues
1. Flexion deformities
2. Muscular detachments
3. Muscular agenesis
4. Scapulothoracic bursitis
II. SECONDARY
1. Disorders of glenohumeral joint
2. Disorders of the subacromial space
III. VOLUNTARY
1. Psychological causes

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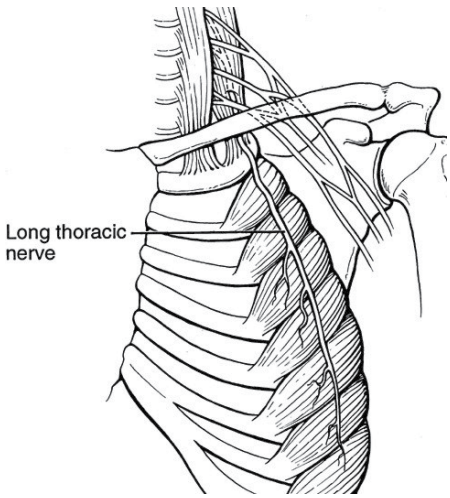

Fig. 9: *Course of long thoracic nerve.*

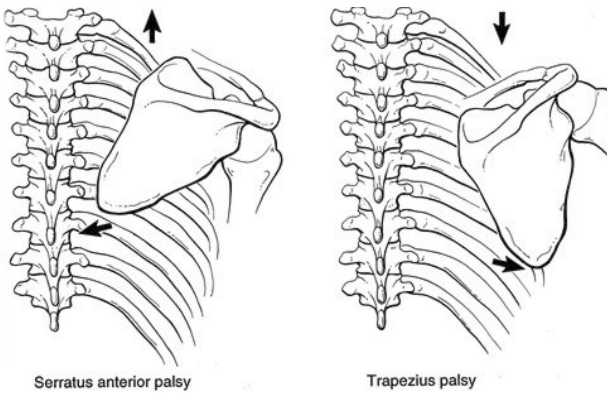

Fig. 10: *Serratus anterior (left) and trapezius (right) palsy*

2) Serratus anterior palsy

Palsy of the serratus anterior can cause a painful winged scapula. The long thoracic nerve that innervates the serratus anterior is formed by the C5,6,7 roots, passes below the brachial plexus and the clavicle and above the first rib. It then runs superficially along the lateral thoracic wall, where it is more susceptible to trauma (**Fig. 9**). Blunt trauma or protraction of the nerve is rather common in athletes, especially those involved with tennis, golf, hockey on ice, soccer, basketball, bowling, javelin throw, wrestlers etc. Repeated minor injuries in workers that use their shoulder extensively have been reported as causes of nerve paralysis. Penetrating trauma can rarely cause nerve damage. Nevertheless, surgical procedures like radical mastectomy, first rib resection and transaxillary sympathectomy have been associated with long thoracic nerve damage. The nerve can also get paralyzed due to non-traumatic causes, like the faulty positioning of the surgical patient under general anesthesia, viral causes, inoculation, brachial plexus or long thoracic neuritis. Moreover, sleeping with the arm placed under the head so that supports it in order to facilitate reading of a book has been shown to cause nerve dysfunction. Lastly, C7 radiculitis can also be a cause of serratus anterior weakening and winged scapula.

Patients with serratus anterior palsy usually complain about pain, as the rest of the scapular muscles are trying to compensate for the winging of the scapula. Extreme pain should lead us to consider the possibility of brachial plexus neuritis or mononeuritis of the long thoracic nerve (*Parsonage-Turner syndrome*) [4]. The scapula is located in a more superior position, displaced inwards with the inferior angle facing inwards (**Fig. 10**). The patient presents with difficulty abducting the arm over 120°, where the winged scapula is more obvious. Pain is even worse in arm abduction when the head of the humerus tilts to the ipsilateral shoulder.

Electromyography is considered gold standard for the confirmation of the diagnosis. It should be repeated every 1-3 months to monitor the healing of the long thoracic nerve, since most cases with paresis are resolved spontaneously within the first 1 to 2 years.

Conservative treatment starts immediately after the diagnosis and includes exercises for the maintenance of the range of movement of the glenohumeral joint. There are many types of braces that keep the scapula attached to the thoracic wall, but their role is controversial.

Penetrating trauma should be treated with early nerve repair. On the other hand, late surgical repair with neurolysis and neuronal grafts does not always give satisfactory results. In patients with symptomatic winged scapula for over a year, surgical repair can alleviate the pain and repair the functional-

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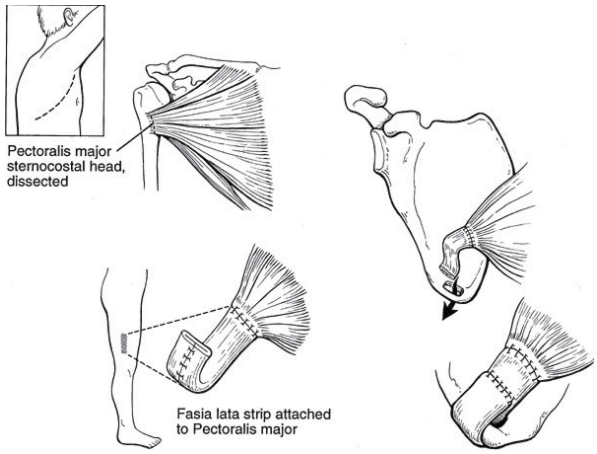

Fig. 11: *Dissection of the sternocostal head of the pectoralis major and enhancement with fascia lata autograft for the treatment of the serratus anterior palsy*

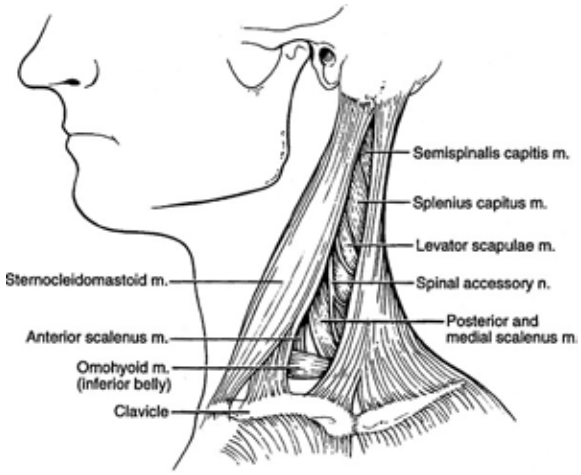

Fig. 12: *Course of the accessory nerve*

ity of the scapula. Historically, these procedures are classified into three categories: (a) arthrodesis of the scapulothoracic joint, (b) use of fascial support and (c) muscle-tendon transfers. For the muscle-tendon transfer, graft sources are usually the pectoralis major, the sternocostal and clavicular heads of pectoralis major, the rhomboids and combinations of the above muscles.

The arthrodesis procedures for the scapulothoracic joint can relieve the pain, but can also lead to loss of scapular mobility. Other complications of arthrodesis are pseudoarthrosis and pneumothorax. The indications of this method are limited to cases where other techniques have failed, simultaneous paralysis of many muscles of the shoulder and in workers that perform heavy tasks and apply too much pressure on the shoulder. Fascial grafts have the drawback of loosening and failure of their supportive ability. Consequently, muscle-tendon transfers have earned interest in the treatment of the winged scapula due to anterior serratus paralysis. Transfer of various muscles has been used, with that of sternocostal head of pectoralis major to be the most popular (**Fig. 11**). The patient is placed at the lateral decubitus position and the incision runs through the axilla, from the pectoralis major up to the inferior angle of the scapula. Alternatively, two different incisions can be made and the pectoralis major is reversed subcutaneously. The sternocostal head of the pectoralis major is released from its insertion biceps groove at the humerus. Then, an autologous graft from the ipsilateral fascia lata (18x15 cm) is prepared. The 18 cm tube-shaped autograft is then side-to-side sutured at the free end of the tendon of the pectoralis major. An aperture is made at the inferior end of the medial border of the scapula and the graft is passed through it sutured under mild tension. Postoperatively, the arm is suspended with a triangular bandage and passive mobilization begins. Active mobilization starts after the first 6 weeks and strengthening exercises after 12 weeks. Early complications of this technique include pneumothorax, while fracture of the inferior angle of the scapula and graft failure has been reported as late complications. Arthrodesis of the scapulothoracic joint can be performed in case of severe complications.

3) Trapezius muscle palsy

The accessory nerve innervates the majority of the trapezius muscle. Its course is superficial, found in the subcutaneous tissue of the posterior cervical triangle, making it susceptible to trauma (**Fig. 12**). The causes of the accessory nerve trauma include blunt trauma, traction, sharp trauma (during lymph node biopsy) and the radical cervical lymph node excision.

Patients suffering from trapezius palsy usually

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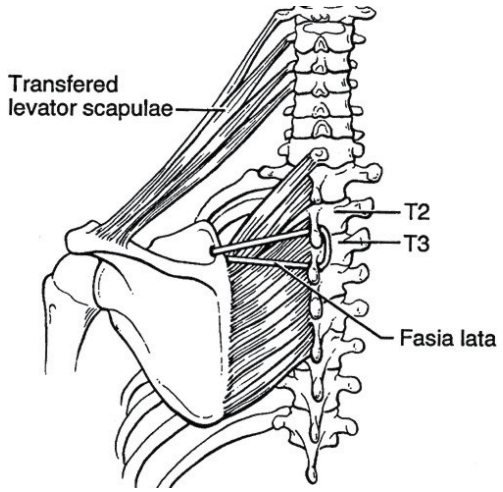

Fig. 13: *Dewar-Harris procedure*

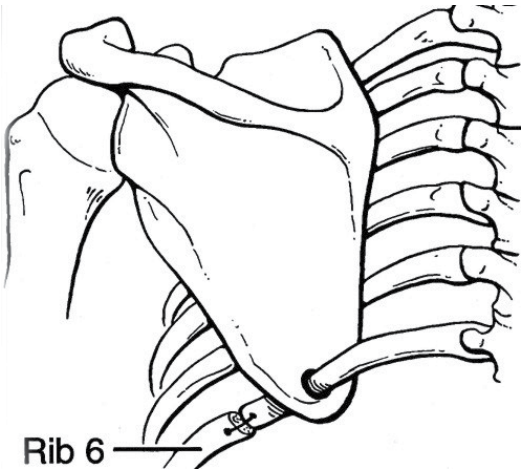

Fig. 14: *Spira procedure*

present with pain due to the counterbalancing action of the levator of the scapula and the rhomboids. Other causes of pain in these patients are the progression to frozen shoulder, the subacromial friction and cervical radiculitis due to protraction of the brachial plexus as a result of the shoulder drop.

Clinical evaluation reveals inability to shrunk the shoulder, as well as inability of abduction and forward flexion of the ipsilateral arm. The scapula is found in a lower level than normal, displaced outwards with the inferior angle headed outwards (**Fig. 10**). Electromyography is used to confirm the diagnosis.

Treatment of patients with winged scapula due to accessory nerve palsy depends on the duration and the intensity of the symptoms. Initial treatment is conservative, with the suspension of the arm in a triangular bandage in order to provide relaxation for the rest of the muscles. Physical therapy aims at the preservation of range of motion of the glenohumeral joint and the avoidance of the progression to frozen shoulder. In cases of blunt trauma-related paralysis, regular follow-up with electromyography every 4-6 weeks is necessary to monitor nerve function. In cases of palsy due to sharp trauma or when no nerve function can be detected, surgical investigation with neurolysis, placement of nerve grafts or combination of the above techniques is the treatment of choice. The results of those methods are rather variable, while they seem to be better when neurolysis is performed within 6 months.

Patients that present with symptoms lasting up to a year are not likely to benefit from conservative treatment. Historically, a great variety of surgical techniques has been reported for the treatment of winged scapula due to trapezius muscle palsy. These techniques can be divided in (a) static stabilization, including arthrodesis of the scapulothoracic joint and (b) dynamic stabilization, including muscle-tendon transfers.

Dewar-Harris technique used to be a very popular method.[5] The medial border of the scapula is stabilized on the spinous processes of T1 and T2 vertebrae with bundles of fascia lata that substitute the middle and lower trapezius. The levator scapula is then transferred to the peripheral end of the scapular spine to substitute the upper trapezius (**Fig. 13**). Postoperatively the arm is placed in spica and at 45°-50° abduction for 6-8 weeks.

Partial arthrodesis of Spira is another historically interesting procedure [6]. During Spira procedure, an aperture is created at the inferior angle of the scapula. The sixth rib is dissected, penetrates the scapula through the hole and its ends are reattached (**Fig. 14**). Full arthrodesis procedures of scapulothoracic joint are indicated in cases of generalized inability of the muscles of the shoulder. Since arthrodesis limits the mobility of the scapula and fascial

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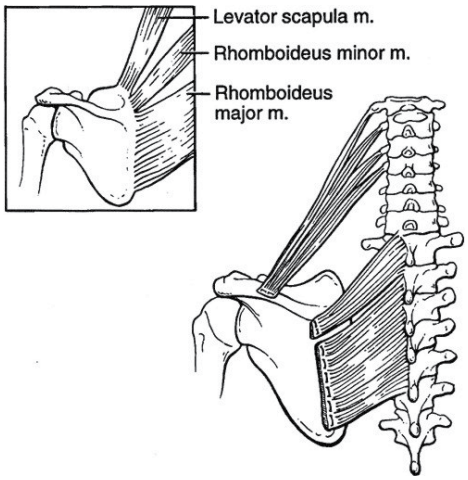

Fig. 15: *Eden-Lange procedure*

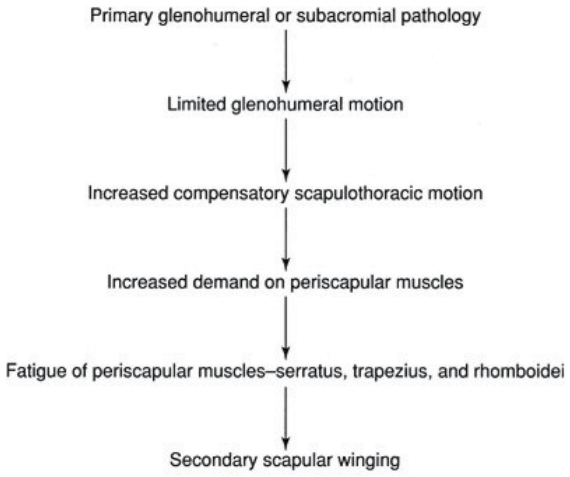

Fig. 16: *Secondary winging cataract*

grafts usually fail due to loosening after 2-3 years, dynamic stabilization with muscle-tendon transfers is currently the treatment of choice.

In the Eden-Lange technique, the levator scapula and the rhomboids are transferred outwards (**Fig. 15**) [7-9]. The levator of the scapula substitutes the upper trapezius, the rhomboid minor substitutes the middle and the rhomboid major the lower trapezius. The lateral relocation of the insertions of these three muscles optimizes the biomechanical outcome and deceases the winging. This method includes two incisions. The first one is performed along the medial border of the scapula and the second one above the scapular spine. The insertions of the three muscles are detached along with an osseous segment. The rhomboids are then directed peripherally under the infraspinatus and fixated with intraosseous sutures (*via* osseous holes made 5 cm peripherally of the medial border of the scapula). The levator of the scapula is directed subcutaneously towards the second incision and gets fixated to the spine through osseous apertures. Postoperatively, the patient uses an arm abduction brace for 4-6 weeks, followed by physical therapy program with passive and active mobilization. The outcomes of this method are considered excellent, with 91% reporting pain alleviation and 87% significant improvement in the functionality of the shoulder.

B. Osseous malformations

Osteochondroma of the scapula or the ribs is the main osseous causes that can lead to winged scapula. This type of winging is due to structural rather than functional causes and can be accompanied by scapular crepitus. Patients usually present with winging that does not change in respect to upper limb movement. Electromyography is normal and the osteochondroma is revealed when either a tangential X-ray of the scapula or a CT scan are performed. Surgical excision of the osteochondroma is the treatment of choice.

Primary causes of the winged scapula include poor positioning during scapula and clavicular fracture healing. These patients can remain asymptomatic due to intact muscle functioning.

C. Soft tissues disorders

Muscular disorders causing winged scapula include traumatic muscle ruptures and congenital muscle agenesis. Electromyography is normal and MRI reveals the cause of winging. Muscle detachments are treated with direct repair. In cases of congenital muscle agenesis of the trapezius, anterior serratus and rhomboids, patients usually compensate for the functional deficiency without the need of surgery.

Winged scapula can also present in 50% of patients suffering from scapular bursitis. Bursitis is usually accompanied with pain and crepitus. Con-

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servative or surgical management of bursitis leads to the effective treatment of the winging and the associated symptoms.

II. SECONDARY WINGED SCAPULA

Secondary winging of the scapula is due to disorders of the glenohumeral articulation that cause abnormal scapulothoracic dynamics (**Fig. 16**). Detailed evaluation of the patient is imperative. The patient should be able to perform passive, active and resisted flexion of the ipsilateral arm and scapula motion should be monitored. Electromyography is normal.

Flexion deformities and muscle contractions around the glenohumeral joint cause secondary winged scapula. Patients with damage in the upper brachial plexus develop this kind of contractions due to the disordered equilibrium of the muscles of the shoulder, with the humerus in abduction and internal rotation. When the humerus is forcibly adducted and externally rotated, the lateral angle of the scapula projects away from the thoracic wall, producing the "*scapular Putti sign*"[10]. Furthermore, the fibrosis of the deltoid causes pronounced winging during adduction while disappears during abduction. Fibrosis of the deltoid can be congenital or due to intramuscular injections.

Winged scapula can be manifested as a reflex muscle spasm after painful conditions of the glenohumeral joint and the subacromial space (**Table 4**). Patients with painful shoulder tend to limit the movement of the scapulothoracic joint. This forces the muscles around the scapula to work harder, as the scapulothoracic motion has to increase in order to compensate for the decreased motion of the glenohumeral joint. When fatigue of these muscles occurs, winging ensues (**Fig. 16**). Management of the primary causes will improve scapular winging. Physical therapy is also needed.

III. VOLUNTARY WINGED SCAPULA

These are rare cases and the physician should seek for a phycological background.

2. SCAPULAR DYSKINESIA

Certain shoulder disorders like glenohumeral instability and subacromial impingement can cause secondary alterations in the dynamics of the scapulothoracic joints causing scapulothoracic dyskinesia. The opposite can also occur, when scapulothoracic dyskinesia leads to disorders of the glenohumeral joint (**Table 5**).

The use of various electromyographic methods revealed a particular and repetitive type of scapulothoracic asymmetry. The synchronized movement of the athletes engaging in throwing sports, depends on the coordination of the scapulothoracic movement. Any pathology in the stabilizing muscles leads to inadequate attempt and increased risk of trauma. The position of the scapula at the time of throwing determines the length-traction relationship of the muscles and enables the creation of the maximal muscle forces and the best possible performance of the athlete. Scapula movement determines the position of the glenohumeral joint in space and optimizes the relevant position of the glenoid fossa towards the head of the humerus to maintainstability of the glenohumeral joint. It also raises the acromion to avoid subacromial impingement and last-

TABLE 4. *Causes of secondary winged scapula related to painful disorders of the shoulder*

Rupture of the rotator cuff muscles
Pseudarthrosis of acromial fractures
Poor healing of clavicular and glenoid fractures
Avascular necrosis of the humeral head
Acromegalic arthropathy of the shoulder
Disorders of the acromioclavicular joint
Shoulder instability

TABLE 5. *Relation of scapulothoracic asymmetry with shoulder pathology*

Shoulder pathology	Static asymmetry	Dynamic asymmetry
Glenohumeral instability	32 %	64 %
Subacromial impingement	57 %	100 %

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TABLE 6. *Causes of scapulothoracic crepitus*

Connective tissue	
Muscle	Atrophy Fibrosis Anatomic abnormalities
Bone	Rib osteochondroma Scapular osteochondroma Rib fracture Hooked superomedial angle of the scapula Lushka's tubercle Reactive bone spurs from muscle avulsion
Other soft tissues	Bursitis Tuberculosis Syphilis
Disorders of the scapulothoracic congruence	
Spine scoliosis Thoracic spine kyphosis	

ly, the muscles around the scapula are important in order to ensure strength against the eccentric load at the various stages of throwing.

Kibler was the first to describe the lateral slide test for the assessment of the scapulothoracic dyskinesia.[11-13] This test is designed to evaluate the patient's ability to stabilize the medial border of the scapula during different positioning and loading. Arms are placed in three different positions: (a) at resting position, (b) hands on hips with fingers anterior and thumbs posterior and (c) arms at 90° with internal rotation. In asymptomatic patients, asymmetry is less than 1 cm. In symptomatic patients with pain and limited range of motion, there is significant asymmetry more than 1 cm in positions (a) and (b).

Clinically, the outward position of the scapula leads to greater anteversion of the glenoid fossa, leading to an increase in the anterior interspace of the glenohumeral joint, and therefore to instability and cartilage lesions. Moreover, disorders of shoulder motion lead to elevation of the acromion and subsequent subacromial crepitus.

Management of the scapulothoracic dyskinesia consists of strengthening exercises of the scapular muscles, while strengthening of the rotator cuff muscles should be avoided until the functionality of the scapula has been restored.

3. SCAPULOTHORACIC CREPITUS

Over time, many names have been attributed to the symptomatic scapulothoracic crepitus, like *snapping scapula, washboard syndrome, scapulothoracic syndrome, rolling scapula, grating scapula, scapulocostal syndrome*, while many causes have been identified (**Table 6**). Although Codman [1] was the one who said that he was able to make his scapula produce a noise loud enough to be heard in the whole room without feeling any pain, Boinet was the first to describe this disorder at 1867. It wasn't until 37 years later, when Mauclaire classified the scapulothoracic crepitus in three groups: (a) *froissement*, is described as a normal mild friction sound, (b) *frottement*, is a more intense sound of coarse friction which is usually pathologic and (c) *craquement* is the intense and loud noise of scapular popping, which is always abnormal [14]. These scapular sounds arise from two sources: either from the tissue between the scapula and the thoracic wall, or from congruence disorders of the scapulothoracic joint. Milch states that the *frottement* is associated with soft tissue pathology or bursitis, while the *craquement* indicates osseous causes [14].

Muscular causes consist of atrophy, fibrosis and abnormal muscle insertions, while tuberculous and syphilitic lesions represent other soft tissue causes.

Osteochondroma of the ribs or scapula is the most common cause of scapulothoracic crepitus. Other causes include poorly healed rib fractures, abnormalities of superomedial angle of the scapula (hooked angle), Lushka's tubercle and reactive osseous spurs from repetitive chronic muscle avulsion. Any osseous cause that produces scapulothoracic crepitus can lead to bursitis. On the other hand, an inflamed bursa can lead to painful crepitus. Finally, disorders of the scapulothoracic congruence, like scoliosis and thoracic kyphosis, can also be a cause of crepitus.

Diagnosis is made through meticulous history

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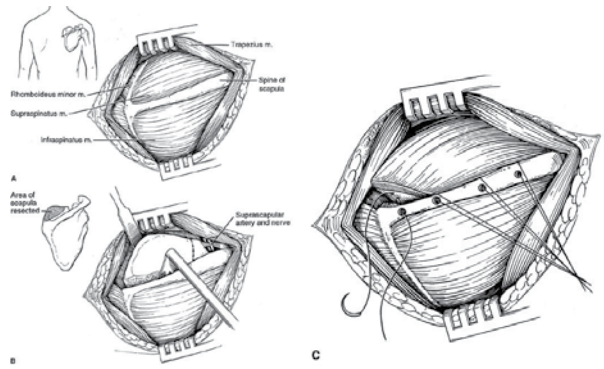

Fig. 17: *Superomedial scapular angle resection*

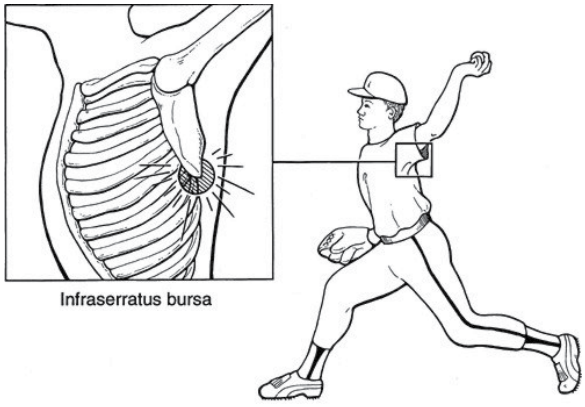

Fig. 18: *Scapulothoracic bursitis*

and clinical evaluation. Patients are often athletes engaging in throwing sports and workers involved in overhead activities. At inspection, winging implies a space-occupying lesion. Palpation and auscultation during shoulder motion will locate the crepitus. Palpation of a mass, crepitus and eminence at resting position combined with normal scapulothoracic motion are key features in differential diagnosis from winged scapula due to neurological causes. The tangent X-ray and CT scan are also helpful tools in diagnosis.

It is important to note that scapulothoracic crepitus is found in 35% of normal individuals. Furthermore, it is possible that patients with psychiatric background will not respond to treatment. Finally, crepitus is considered an abnormal finding, when associated with pain, winging or other scapulothoracic disorders.

When osseous causes are involved, like osteochondroma, surgical resection is the treatment of choice. In cases of soft tissue pathology, initial management is conservative, with exercises that aim to avoid downward inclination of the scapula (use of figure-of-eight bandage), strengthen exercises of the adjacent muscles and corticosteroid infusion in painful sites.

Surgical techniques include muscle transfers for muscle palsy cases, such as the transfer of rhomboids, trapezius and their re-attachment under the scapula. These procedures are associated with muscle atrophy and failure. Other procedures are the partial resections of the scapula, mainly the medial border or the superior medial angle.

During the superior medial angle resection, the patient is placed at prone position (**Fig. 17**). The incision is made over the medial part of the scapular spine and the overlying soft tissues are dissected. The periosteum of the spine is then elevated to create space between trapezius and scapula. The supraspinatus, rhomboids and levator scapula are subperiostically dissected starting from the spine. The superomedial angle of the scapula is resected with the use of a scapular saw. Care is taken not to injure the suprascapular nerve and the dorsal scapular artery. The muscles and periosteum are then reattached in place and sutured through osseous tunnels. Postoperatively, the arm is suspended in triangular bandage and patients start passive mobilization immediately. Active mobilization starts at 8 weeks following the operation and strengthening exercises at 12 weeks.

Complications of the partial scapular excision are pneumothorax and postoperative hematoma. Recurrence is more common in younger patients, but are rarely symptomatic.

4. SCAPULOTHORACIC BURSITIS

Scapulothoracic bursitis can either accompany scapular crepitus or be a separate entity. Patients often complain of pain related to activities or present with audible or palpable crepitus. They usually de-

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scribe a repetitive activity that makes scapula move against the posterior thoracic wall. A chronic inflammation is then developed that leads to fibrosis and scarring followed by crepitus and pain (**Fig. 18**).

Initial management is conservative with rest, analgesics and NSAIDs. Physical therapy, with strengthening exercises and stretching of the dorsal musculature, improves posture. Heat patches and cortisone injections offer pain. If symptoms persist, surgical treatment is indicated.

Sisti & Jobe performed bursectomy of the inferior scapular angle in athletes. An oblique incision distal to the inferior angle is made and trapezius and latissimus dorsi are dissected along their muscle fibers. Physical therapy started 1 week postoperatively and within 6 weeks the athletes were able to perform mild throwing exercises.

McCluskey & Bigliani performed open bursectomy of the superomedial angle with vertical incision on the inside of the medial border of the scapula. After dividing the trapezius, the levator scapula and the rhomboids were subperiostically released from the scapular medial border. The space created between the latissimus dorsi and the thoracic wall enabled the resection of the thickened bursa. Muscles are then reattached. Triangular bandage is used to suspend the arm and after 3 weeks active range of motion exercises begin, while 12 weeks postoperatively strengthening begins.

Ciullo & Jones conducted endoscopic bursae resection with debridement and reconstruction of the superomedial or inferior scapular angle. Arthroscopic bursae resection was described by Matthews. Patient is placed at either lateral or prone positioning, which enables the arthroscopic evaluation of the glenohumeral articulation and the subacromial space. Furthermore, the scapula moves away from the thoracic wall with abduction and internal rotation, facilitating access to the bursa. Three trocar insertion sites are created and the trocars are placed at least 2 cm on the inside of the medial scapular border and between the spine of the scapula and the inferior angle. For the middle insertion site, a needle is placed inside the bursa between the latissimus dorsi and the thoracic wall. The needle should enter between the spine and the inferior angle and at least 3 fingers on the inside of the medial border in order to avoid injury of the suprascapular artery and nerve. The bursa first gets larger with fluid infusion, before the insertion of the instruments. The upper insertion site is created 3 fingers on the inside of the medial scapular border, right under the spine and penetrates the space between the two rhomboids. This site enables access at the superomedial angle. A more medial positioning of the insertion site would jeopardize the suprascapular artery and nerve, the accessory nerve and the circumflex scapular artery. The lower insertion site is placed in a similar way on the inferior angle. Shaver is used for bursa resection. Postoperatively, active mobilization begins immediately.

Conflict of interest:

The authors declared no conflicts of interest.

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READY - MADE CITATION

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ΠΕΡΙΛΗΨΗ

Η ωμική ζώνη κινείται μέσω τριών αρθρώσεων, της γληνο-βραχιονίου, της ακρωμιο-κλειδικής και της θώρακο-ωμοπλατιαίας. Η τελευταία, αν και αποτελεί σημαντικό στοιχείο της λειτουργίας του ώμου, δεν έχει λάβει την απαραίτητη προσοχή σε σχέση με τις δύο πρώτες, τόσο στην ιατρική βιβλιογραφία όσο και στην καθημερινή ιατρική πρακτική Οι παθήσεις της θωρακο-ωμοπλατιαίας άρθρωσης είναι δύσκολα κατανοητές ή και διαγιγνώσκονται ακόμα πιο δύσκολα. Συμβαίνουν συχνά σε χειρώνακτες αλλά και σε αθλητές υγρού στίβου και ρίπτες όπου η διαρκής και έντονη κίνηση των άνω άκρων και του κορμού καθιστά την ωμοπλατιαία χώρα την πλέον ενεργή λειτουργικά περιοχή. Η αιτιοπαθογένεια της θωρακο-ωμοπλατιαίας δυσλειτουργίας είναι συνήθως συνδυασμός ανατομικών, μυϊκών και νευρολογικών διαταραχών που επηρεάζουν την εμβιομηχανική της ἀρθρωσης αυτής.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: ωμοπλάτη, πτερυγοειδής, δυσκινησία, κριγμός, ορογονοθυλακίτιδα

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ORIGINAL PAPER

Femoral distal-end fractures treatment using the Ilizarov circular frame

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ABSTRACT

AIM: The distal femur fractures are usually results of high energy injuries, pathological or periprosthetic fractures. The aim of this report is to describe the indications of Ilizarov external fixator (IEF) device as a suitable surgical treatment for these severe injuries, to describe the construct design and to evaluate the results in 16 patients treated using this method.

MATERIAL AND METHODS: 16 patients were assessed, (8 women - 8 men), with a range of 21 to 85 years of age. The fractures were of AO type 33-A1, 2, 3 & 33-C1, 2, 3. In two patients the fractures were periprosthetic. Two patients presented with nonunion and failure of the previously applied osteosynthesis respectively. In these patients knee bridging was deemed necessary. The IEF construct design featured a twin ring module for the supracondylar fracture fragment in the majority of the cases.

RESULTS: The mean hospitalization time was 7 days and the postoperative follow up was 6 - 52 months. Complete union was achieved in all cases without the need of reoperation in any case. The tibial part of the construct was removed after 4-8 weeks postoperatively and the femoral part of the construct was removed after 18 weeks respectively. The average time to union was 18 weeks. There were neither deformities, nor osteoarthritic lesions in the longest follow up cases. The range of motion of the knee was satisfactory in all cases.

CONCLUSION: The treatment of distal femur fractures of AO types 33-A1, 2, 3 & 33-C1,2,3 using the IEF is highly effective and it is our belief that this is the preferable method for the management of the above described injuries. This method shows numerous advantages, such as adjustment of the joint alignment, respect of soft tissues due to less invasive technique, early mobilization and no need for a second anesthesia and operation for IEF removal. The method shows no major complications apart from the common problem of pin site infection which in the majority of cases is easily managed with wound dressing, antibiotics administration or relocation of the pins.

KEY WORDS: distal end femoral fractures; surgical treatment; Ilizarov circular frame

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1. Introduction

Fractures of the distal femur represent 3-4% of femoral fractures and approximately 0.4% of all fractures. Their epidemiological curve in relation to age and gender shows a typical bimodal distribution, with a first peak for young males in their third and a second for elderly women in their seventh decade respectively. The mean age of all patients with such fractures is 60 years, while over 50% of fractures occur in patients over 65 years. The fractures are twice more frequent to women than men, while 55% of the fractures are intra-articular [1, 2]. The most commonly used classification is this of AO / OTA, depending on the location and the comminution of the fracture namely: 33-A1,2,3 extra-articular, 33-B1,2,3 partial articular, 33-C1,2,3 complete articular (**Fig. 1)** [3]. A separate category of distal femoral fractures are the periprosthetic fractures, over a previously applied total knee arthroplasty, which are usually classified according to the displacement of the fracture and the stability of the femoral component by Lewis & Rorabeck (**Fig. 2**) [4,5]. Pathological fractures of this type related to neoplasmatic lesions of the distal femur can also occur. Fractures in elderly people are usually low-energy injuries such as falls on a flexed knee and occur in an osteoporotic bone, while young patient's fractures are high-energy injuries as car or industrial accidents and falls from a great height. Fractures in young people are usually intra-articular and highly comminuted, accompanied approximately in 20% with ligamentous injuries. As a result of violent injuries, these fractures can be part of complex lesions around the knee, that is with a coexisting fracture of the proximal tibia ("floating knee"), in association with dislocation of the knee or as second or third degree open fractures, with associated vascular injury. The treatment of these serious injuries is quite challenging and an increased rate of complications such as infection, septic nonunion in 7-13% (25-30% in open fractures), non-septic nonunion (in 10-14%), a significant reduction of the range of motion in 35% and posttraumatic arthritis of the knee up to 50% [1].

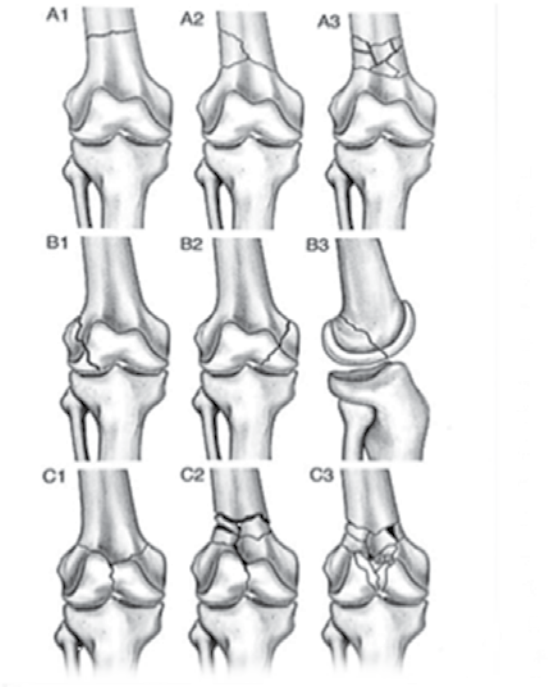

Fig. 1. *AO classification of distal femoral fractures*

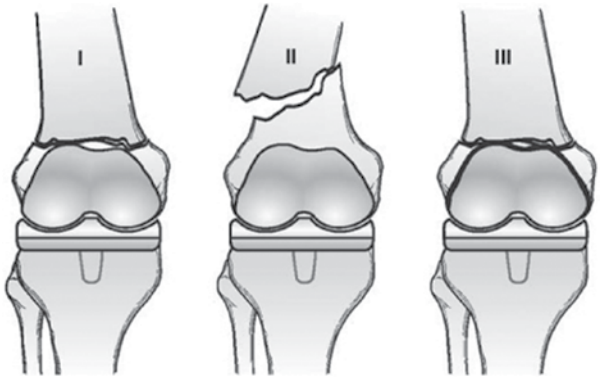

Fig. 2. *Lewis & Rorabeck classification of distal femoral fractures following a TKR*

2. Material and Methods

The patients: This study includes 17 fractures in 16 patients, 8 men and 8 women, aged 21 to 85 years. All fractures were either extra-articular AO type 33-A1,2,3 or intra-articular AO type 33-C1,2,3. Two patients presented with periprosthetic fractures following total knee arthroplasty and two patients presented with nonunions and failure of the applied fixation. One fracture was accompanied with a fracture in the ipsilateral proximal tibia ("floating knee"). In one patient a 33-A3 type fracture occurred below a DHS plate.

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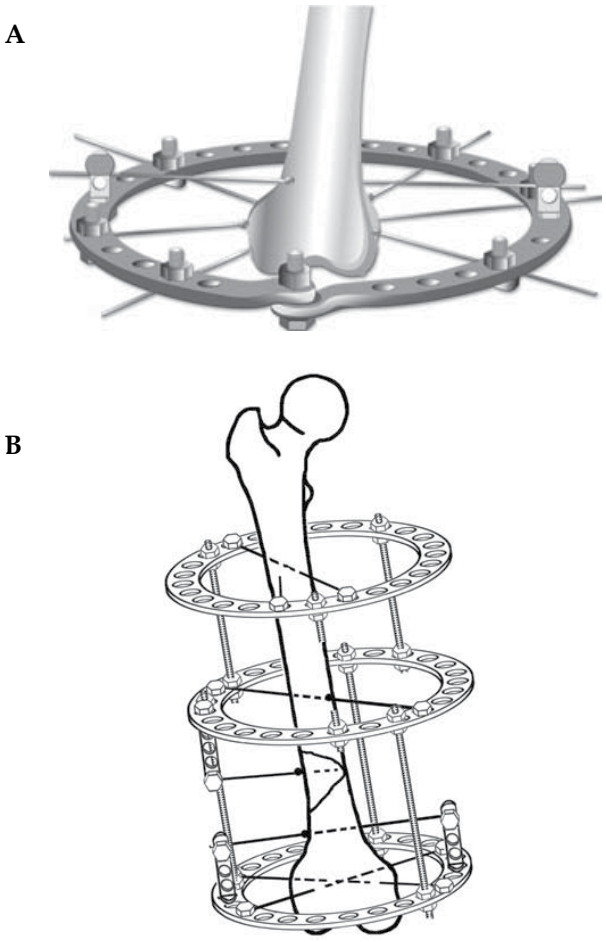

Fig. 3. *femoral frame, (a,b)*

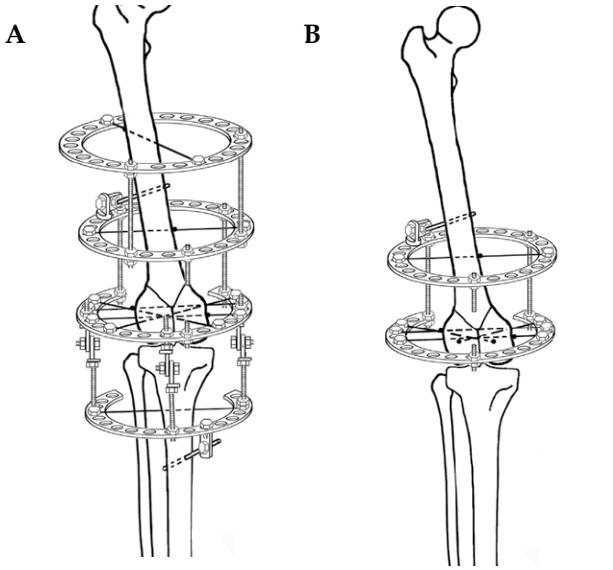

Fig. 4. *bridging of the knee (a) and removal of the tibial part of the frame 4-8 weeks later (b)*

The construct and the surgical technique: The application of the frame started in all cases with placement of the distal femoral part, using a twin-ring in most cases and the connecting rods parallel to the anatomic axis of the femur (**Fig. 3**) [6,7]. In all fractures bridging of the knee for 4-8 weeks was considered necessary (**Fig. 4)** [6].The rings stabilization was achieved using fine wires in combination with half - pins. The placement of fine wires and half - pins and the fracture reduction assessment was done using a C-arm image intensifier. The mean operative time was 70 minutes.

Postoperatively a second generation cephalosporin was administered for 24 to 48 hours and all patients were given low molecular weight heparin for 5 weeks. All patients were prescribed the same postoperative physical therapy protocol (mobilization of hip - ankle and early low-weight bearing).

As healing time was defined the time from the operation to the removal of the device and full weight bearing. Hospitalization time, healing time and the major and minor complications were recorded. Bone healing and functional outcome were evaluated using the ASAMI scale (**Table 1)**.

3. Results

The mean hospitalization time was 7 days. Follow-up (clinical and radiological) ranged from 6-52 months. The tibial part of the frame was removed after an average time of 5 weeks (4-8 weeks), whereas the femoral part after 18 weeks. The mean time to union was 18 weeks. Complete union of fractures was achieved in all patients with no need of reoperation in them.

Major complications (neurological, deep vein thrombosis, pulmonary embolism, compartment syndrome) were not developed in any patient. Among minor complications (pin tract infection, knee stiffness, depressive illness, delayed union, nonunion, septic nonunion, axis disorder, shortening more than 1.5 cm) only pin tract infections presented in 3 patients, which were treated conservatively

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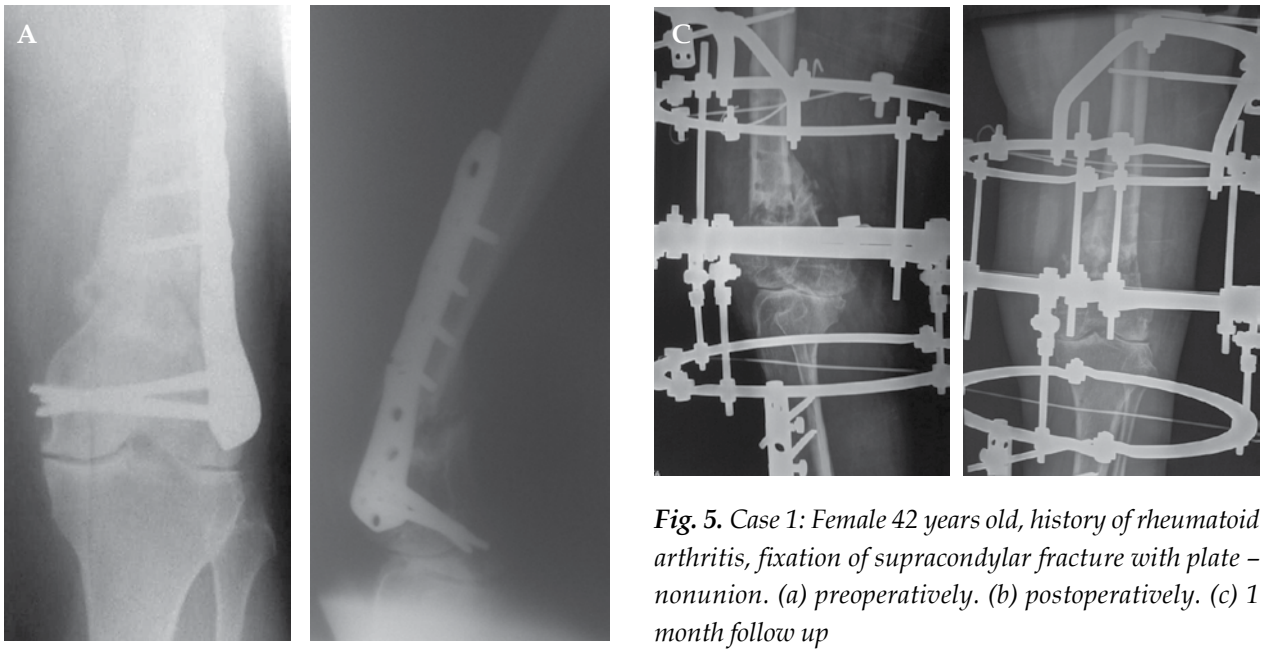

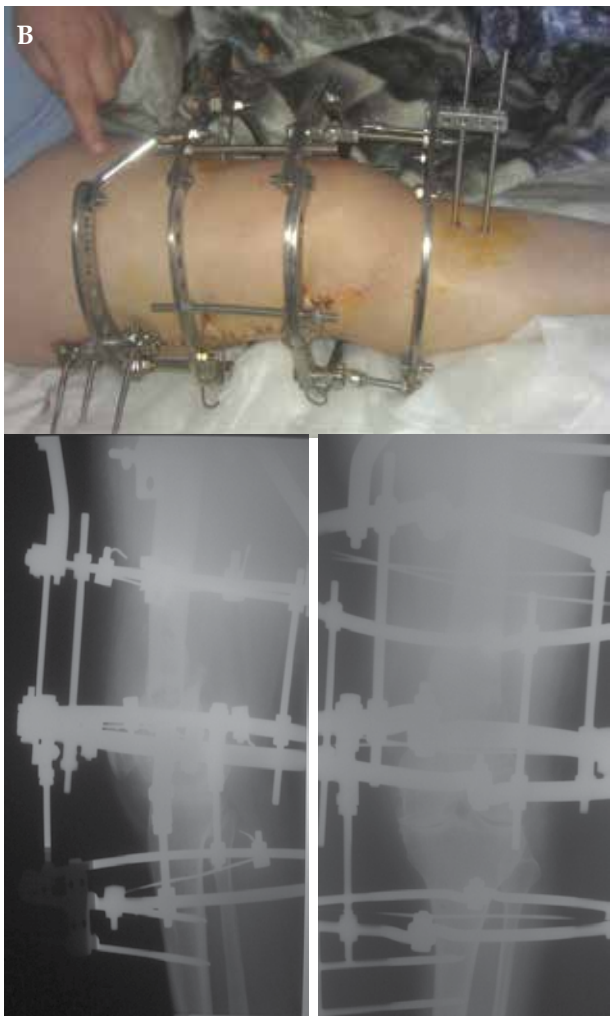

Fig. 5. *Case 1: Female 42 years old, history of rheumatoid arthritis, fixation of supracondylar fracture with plate – nonunion. (a) preoperatively. (b) postoperatively. (c) 1 month follow up*

with antibiotics with no need to remove or replace them. Concerning the two most common complications, stiffness and development of postoperative osteoarthritis of the knee, the results were good, with a satisfactory range of motion of the knee in all patients and no osteoarthritic lesions development even in those with the longest follow up (**Fig. 5-8)**.

4. Discussion

Fractures of the distal end of the femur are a challenge for the surgeon, presenting both a high degree of technical difficulty during the operation and high rates of failure of the fixation and nonunion formation. Fractures in young patients are high-energy injuries and usually coexist with injuries of other systems or can be accompanied by severe ligamentous lesions of the knee, other fractures of the ipsilateral limb or neurovascular lesions relating to open fractures. Fractures in elderly patients usually occur in osteoporotic bone or in the femur after a preexisting fixation (below nails, stems, DHS plates etc.) or in the bone of the femoral component of a total knee replacement, in the periprosthetic fractures. For all these challenges, the Ilizarov method is a powerful and effective tool in surgeon's hands presenting numerous advantages compared with other fixation

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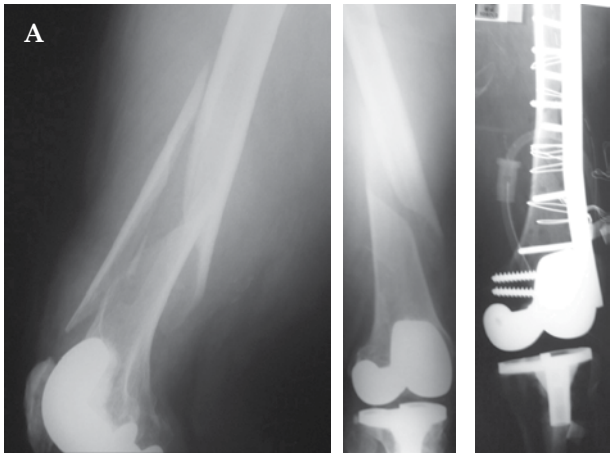
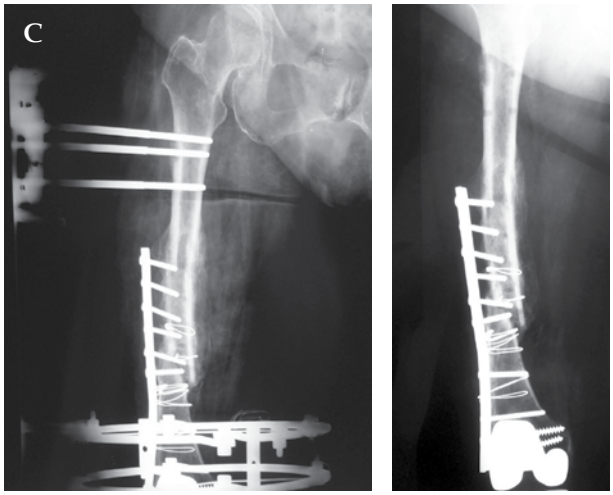
TABLE 1. *Evaluation of the outcomes using the ASAMI scale*

	Bone results	
excellent	union, no infection, deformity <7°, limb-length discrepancy <2.5 cm	8
good	union + any two of the following: absence of infection, <7° deformity and limb-length inequality of <2.5 cm	8
fair	union + only one of the following: absence of infection, deformity <7° and limb-length inequality <2.5 cm	1
poor	nonunion / re-fracture / union + infection + deformity >7° + limb-length inequality >2.5 cm	_
	Functional results	
excellent	active, no limp, minimum stiffness (loss of <15° knee extension / <15° dorsiflexion of ankle), no reflex sympathetic dystrophy (RSD), insignificant pain	6
good	active, with one or two of the following: limp, stiffness, RSD, significant pain	10
fair	active, with three or all of the following: limp, stiffness, RSD, significant pain	1
poor	inactive (unemployment or inability to return to daily activities because of injury)	_
failures	amputation	_





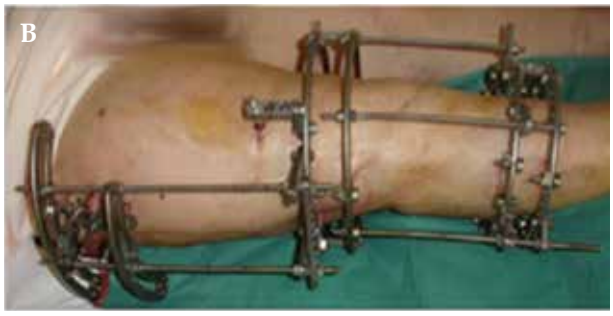


Fig. 6. *Case 2: Female 68 years old, fixation of supracondylar fracture of the right femur, failure of the fixation, (a) preoperatively, (b) postoperatively, (c) 4 months follow up*

methods, such as plates (anatomical, LISS) and retrograde nails for supracondylar fractures. The effective intraoperative adjustment of the femoral axis yet the possibility of a postoperative additional adjustment, the relatively short operative time, the minimal operative soft tissue damage, the reduction of even small bone fragments using olive-wires and the device removal at the outpatient department without the need for a second general anesthesia and reoperation are only some of the method's advantages. Above all, the bridging of the knee for as long as needed, depending upon the fracture character is a major ad-

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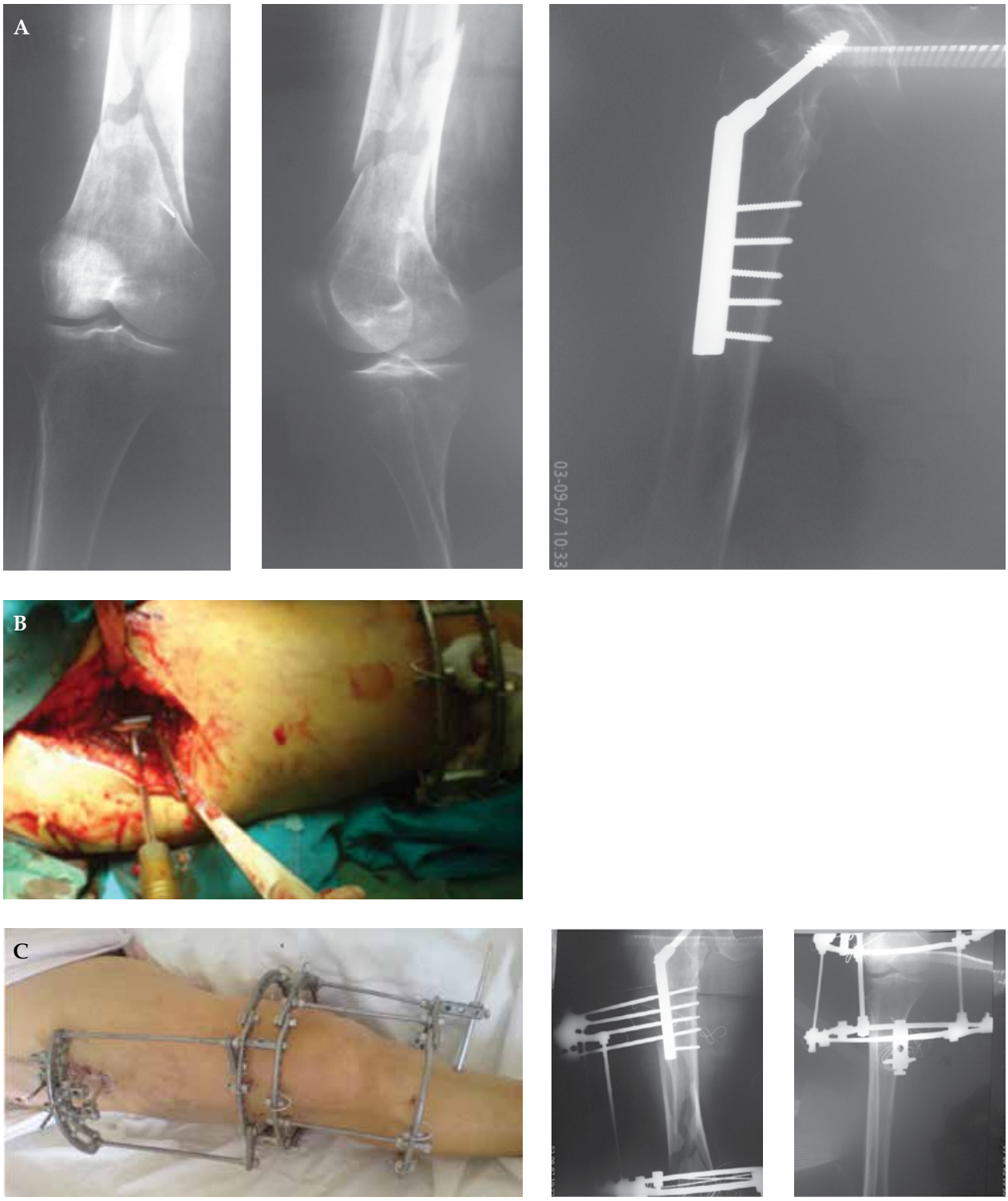

Fig. 7. *Case 3: Female 72 years old, right supracondylar periprosthetic femoral fracture (below DHS). (a) preoperatively. (b) intraoperatively. (c) postoperatively*

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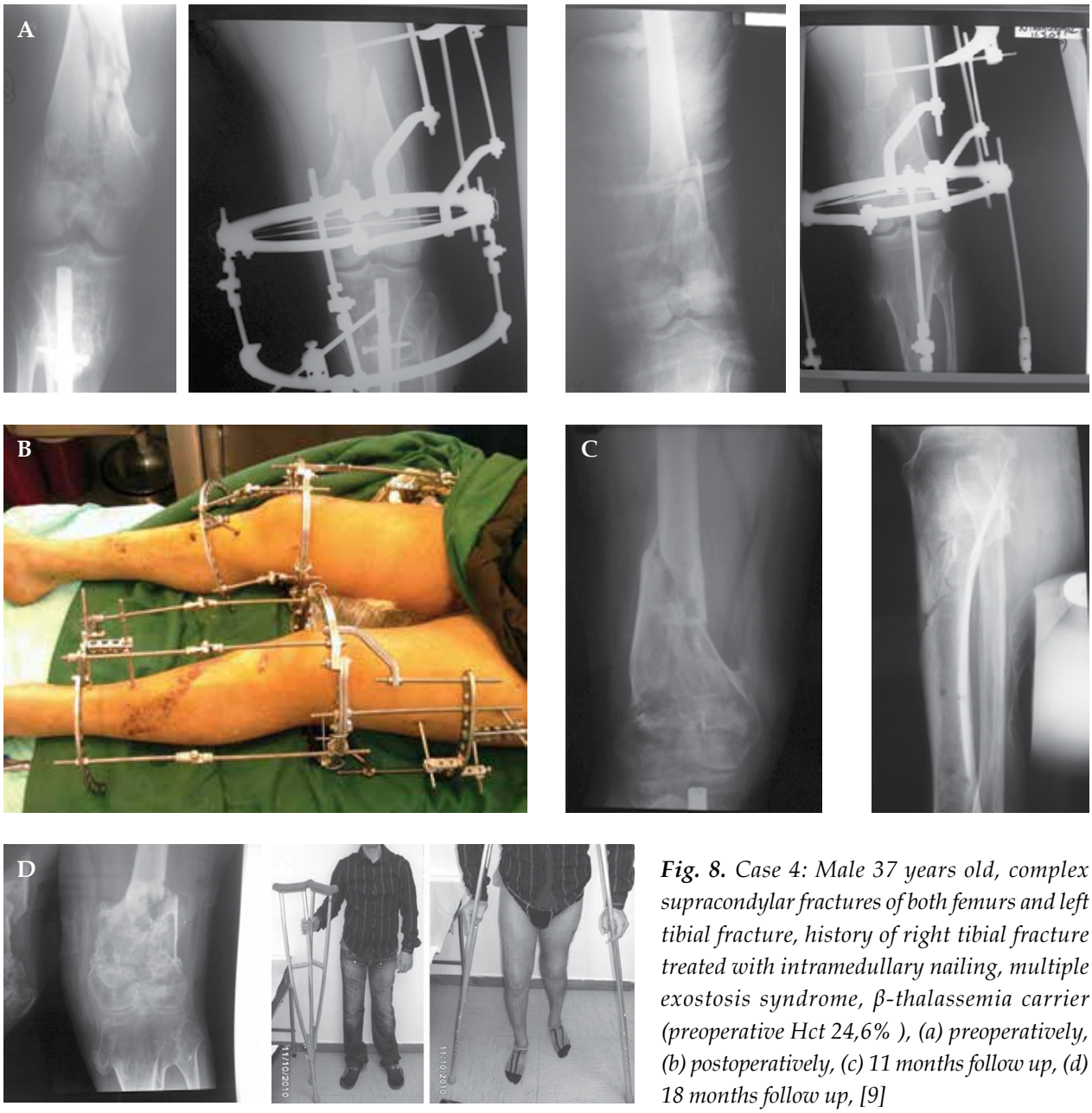

Fig. 8. *Case 4: Male 37 years old, complex supracondylar fractures of both femurs and left tibial fracture, history of right tibial fracture treated with intramedullary nailing, multiple exostosis syndrome, β-thalassemia carrier (preoperative Hct 24,6%), (a) preoperatively, (b) postoperatively, (c) 11 months follow up, (d) 18 months follow up, [9]*

vantage of the method, often necessary to preserve both the reduction and the stability of the joint, especially in highly comminuted fractures or those which are accompanied by severe ligamentous injuries. The frame construction by the surgeon intraoperatively, its versatility and the numerous possible combinations of the frame components enables the physician to exploit any characteristic of the fracture. In addition, this method minimizes or even eliminates the patient's need for intra- or postoperative blood transfusion, which is very important for both multi-trauma and elderly patients. Finally this method presents low incidence of complications, commonly the minor and easily treatable complication of pin tract infection [7, 8].

Conflict of interest:

The authors declared no conflicts of interest.

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ΠΕΡΙΛΗΨΗ

ΣΚΟΠΟΣ ΤΗΣ ΜΕΛΕΤΗΣ: Τα κατάγματα του άπω πέρατος του μηριαίου αφορούν κακώσεις υψηλής ενέργειας σε νέους κυρίως ασθενείς, χαμηλότερης ενέργειας σε ηλικιωμένους οστεοπορωτικούς ασθενείς, παθολογικά κατάγματα, είτε περιπροθετικά κατάγματα μετά από ολική αρθροπλαστική γόνατος. Σκοπός της εργασίας είναι ο καθορισμός των ενδείξεων και η εφαρμογή της μεθόδου Ilizarov ως θεραπεία των καταγμάτων αυτών, καθώς και η αξιολόγηση των αποτελεσμάτων σε 16 τέτοιους ασθενείς.

ΥΛΙΚΟ ΚΑΙ ΜΕΘΟΔΟΣ: Μελετήθηκαν 16 ασθενείς (8 γυναίκες - 8 άνδρες) ηλικίας από 21 έως 85 ετών. Τα κατάγματα ήταν τύπου 33_A1,2,3 και 33-C1,2,3 κατά ΑΟ. Σε 2 περιπτώσεις τα κατάγματα ήταν περιπροθετικά και σε 2 περιπτώσεις αφορούσαν ψευδάρθρωση με αστοχία προηγούμενου υλικού. Σε όλους τους ασθενείς εφαρμόστηκε γεφύρωση του γόνατος και χρήση δίδυμου δακτυλίου στο μηριαίο μέρος του πλαισίου στις περισσότερες περιπτώσεις.

ΑΠΟΤΕΛΕΣΜΑΤΑ: Ο μέσος χρόνος νοσηλείας ήταν 7 ημέρες και ο χρόνος μετεγχειρητικής παρακολούθησης των ασθενών ήταν από 6 έως 52 μήνες. Το πλαίσιο της κνήμης αφαιρέθηκε 4-8 εβδομάδες μετεγχειρητικά, ενώ το αντίστοιχο του μηρού κατά μέσο όρο μετά από 18 εβδομάδες. Ο μέσος χρόνος πώρωσης των καταγμά-

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των ήταν 18 εβδομάδες. Δεν παρατηρήθηκαν παραμορφώσεις, ούτε οστεοαρθριτικές αλλοιώσεις στους ασθενείς με το μακρύτερο χρονικό διάστημα παρακολούθησης. Η κινητικότητα του γόνατος μετεγχειρητικά ήταν ικανοποιητική και δεν παρατηρήθηκε κανένα ασθενής ψευδάρθρωσης.
ΣΥΜΠΕΡΑΣΜΑΤΑ: Η αντιμετώπιση των καταγμάτων του άπω πέρατος του μηριαίου τύπου 33-A1,2,3 και 33-C1,2,3 με συσκευή Ilizarov είναι επιτυχής και θεωρούμε ότι αποτελεί μέθοδο εκλογής. Τα πλεονεκτήματα αυτής είναι ο έλεγχος του άξονα του σκέλους, ο ελάχιστος χειρουργικός τραυματισμός, η πρώιμη κινητοποίηση καθώς και η αφαίρεση της συσκευής στα εξωτερικά ιατρεία χωρίς να υποβληθούν οι ασθενείς σε επανεπέμβαση. Σημαντικές επιπλοκές δεν παρουσιάστηκαν, παρά μόνο λοίμωξη στο σημείο εισόδου των βελονών, επιπλοκή όμως σχετικά εύκολα θεραπεύσιμη.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: κατάγματα άπω πέρατος μηριαίου, χειρουργική θεραπεία, κυκλικό πλαίσιο Ilizarov

BASIC SCIENCE
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The significance of peroxisome proliferator activated receptors PPAR-γ pathway in Arthritis

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ABSTRACT

PPAR – γ are members of a nuclear receptors superfamily located in various parts of the human body. They are implicated in many biochemical pathways and are shown to hold a key role in adipogenesis, adipose tissue differentiation and regulation of glucose levels, while also exhibiting anti-inflammatory properties. Furthermore, numerous studies have verified their implication in the process of cell differentiation and apoptosis. Over the last decade there has been ongoing interest and continuous investigation concerning their potential role on bone metabolism and the treatment of arthritis.

KEY WORDS: arthritis; cartilage; peroxisome proliferator activated receptors; pathways

1. Introduction

Peroxisome proliferator activated receptors (PPARs) are part of a nuclear receptor superfamily and can be separated into 3 subgroups: (a) PPARs-α, are usually detected at the liver, pancreas, lungs and kidneys, whilst there is also an abundance of PPAR-α receptors in muscle tissue and vascular wall cells. (b) PPARs-β/δ, are located at the human embryonic kidneys, small intestines, muscle and adipose tissue as well as the developing brain and heart. (c) PPARs-γ, are mostly found in white and brown adipose tissue, osteoclasts, synoviocytes, chondrocytes, macrophages, T-lymphocytes, the mammary and adrenal gland, skeletal muscle and prostate as well as the heart and type 2 alveolar pneumonocytes [1]. When triggered, PPARs-γ promote adipogenesis and adipocyte differentiation, while regulating glucose homeostasis. Furthermore,

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by suppressing NFκB, they exhibit anti-inflammatory action and promote cellular differentiation and apoptosis [2]. According to Ricotte et al. it is also possible that PPARs-γ trans-repress the expression of pro-inflammatory mediators by inhibiting STAT-1 (signal transducers and activators of transcription-1) and AP-1 (activation protein-1) signaling [3, 4]. Moreover, after studying human chondrocytes, Yamamoto et al. reported that it plays a critical role in the control of B cell response and diseases in which B cell hyper reactivity is involved, such as arthritis and autoimmunity [5].

Various pharmaceutical compounds such as Thiazolidinediones (TZDS), NSAIDS, prostanoids (15dPGJ2), leukotriene B4 (LTB4), 15-hydroxy-eicositetranoic acid (15-HETE), Non-Steroidal-Anti-Inflammatory Drugs (NSAIDs), eicosanoids, glitazars and fatty acids are among the identified PPAR-γ ligands. Despite their differences, all ligands have carboxylate moiety and lipophylic backbone [6]. However, Jee et al. reported that 15dPGJ2 had the greater PPAR-γ affinity being 5- to 30-fold more potent than TZDS [7], with troglitazone, indomethacin, fenoprofen and ibuprofen following [8].

2. The role of PPAR-γ in arthritis

Osteoarthritis (OA) and rheumatoid arthritis (RA) are associated with inflammation mediated by IL1β and TNFα. They consequently trigger the production of MMPs by chondrocytes and synovial fibroblasts [9] causing destruction of extracellular matrix components in both bone cartilage and tendons.

PPAR-γ ligands prevent joint destruction by either reducing the expression of inflammatory cytokines [10] or limiting the expression of MMPs [11]. In addition they hold an important role in the *in vitro* transformation of fibroblast like synovial cells (FLS) into adipocyte like cells [13]. In RA, FLS are the most common cell type at the pannus–cartilage junction and contribute to joint destruction through their production of cytokines, chemokines, and matrix-degrading molecules and by migrating and invading joint cartilage. They share some characteristics with malignant cells [14,10] and it has been suggested that after their differentiation into adipocyte like cells their proinflammatory character is diminished [13].

3. Degenerative arthritis

Osteoarthritis (OA) is a degenerative disease, highly associated with motor disability, resulting in degradation of the articular cartilage and declined life quality. Factors such as aging, excessive mechanical stress, traumatic injury and genetic susceptibility are major risk factors for the occurrence of osteoarthritis. The pathophysiology of this condition is related with a shift of balance between the production rate and the degradation of the human cartilage, possibly due to the induction of proteolysis. Excessive release and production of cytokines, matrix metalloproteinases (MMP's) and nitric oxide (NO) are playing an immense role in the occurrence of OA [12].

Cytokines IL-1b and TNFα induce the production of MMP1, 8 and 13.The latter are considered primary mediators of the joint destruction process by degrading type II collagen. Furthermore, IL-1b and TNF-α are responsible for the increased levels of NO. According to Clansy et al., NO can lead to cartilage degradation by 1) reducing the production of cartilage matrix, 2) enhancing the activity of MMP's, 3) inhibiting the IL1 receptor antagonist and 4) inducing chondrocyte apoptosis [15].

After studying guinea pigs suffering from experimental osteoarthritis Kobayashi et al. found that PPAR-γ ligand Pioglitazone caused dose-dependent decrease of MMP13 and IL-1b amounts situated in the osteoarthritic cartilage. This resulted in remission of osteoarthritic symptoms and decrease of the degenerative lesion's depth and size [16].

It has been asserted that COX-2 inhibitors can demonstrate biologic activities other than the simple inhibition of COX action and release of prostaglandins [17]. Therefore the effect of NSAIDS as PPAR-γ ligands was investigated and confirmed [5]. However, despite that confirmation, other studies showed that nimesulide and other PPAR-γ ligands such as 15fPGJ2 and ciglitazone, increase COX2 mRNA in dose dependent manner [18].

Curcumin is another PPAR gamma ligand that de-

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celerates osteoarthritis progression and offers adequate pain relieve. However, it remains unclear whether its action is related with the PPAR gamma pathway [19, 20].

Irrespective of the equivocal results of older studies it has been confirmed that PPARγ deficiency results in severe, accelerated osteoarthritis [21].

4. Acute gouty arthritis

Acute gouty arthritis is a monoarticular disease with excruciating symptoms and sudden onset. It is caused after the deposition of monosodium urate monohydrate (MSU) in the articular and periarticular tissue. MSU crystals infiltrate leucocytes and stimulate synovial cells through leucocyte secreted chemokines (IL-8, monocyte chemoatractant protein 1), oxygen radicals, cytokines (TNFα, IL-6, IL-1), arachidonic acid, metabolites and proteinases [22]. The activation of neutrophils after the MSU crystals sedimentation initiates the tissue damage, which is perceived as pain, edema and periarticular erythema.

MSU crystals induce monocyte PPAR-γ expression *in vitro* and in vivo, while PPAR-γ ligands decrease the production of crystal-induced cytokines. When tested, both 15dPGJ2 and indomethacin reduced significantly the production of cytokines. However, troglitazone failed to exert significant results. Those results imply that PPAR-γ may hold a significant biologic role in the self-limiting episodes of gouty arthritis [23].

5. Psoriatic arthritis

Psoriatic arthritis is a chronic disorder affecting both the joints and skin. It affects 5-7% of the population suffering from psoriasis and appears approximately 10 years after it is diagnosed. Dactylitis, tendinitis as well as joint inflammation and neovascularization are among its primary clinical manifestations. Its treatment does not differ from that of rheumatoid arthritis. Non-steroidal anti-inflammatory drugs (NSAIDs) have been used as treatment as well as Disease Modifying Antirheumatic Drugs (DMARDs) with the latter demonstrating inconsistent and unsatisfactory efficacy. Moreover, anti-TNF agents have limited use due to their high costs.

Activation of the PPAR-γ pathway had positive results in several *in vitro* and *in vivo* models (e.g. collagen-induced arthritis). Pioglitazone was used in most studies and exerted remarkable anti-inflammatory properties while suppressing neovascularization [24]. It could therefore be a promising treatment for psoriatic arthritis, even though more clinical studies are needed in order to define its response in patients with psoriasis and psoriatic arthritis [25].

6. Rheumatoid arthritis

Rheumatoid arthritis (RA) is a chronic polyarticular autoimmune relapsing and destructive disease affecting mainly diarthroidal joints. It is mainly associated with massive synovial cell proliferation, inflammation and angiogenesis [26, 27].

An antigen-dependent T cell activation stimulates mesenchymal and fibroblast-like synovial cell proliferation, causing irreversible damage in both bone and cartilage [28]. Therefore the rationale for the treatment of RA would be suppressing or blocking osteoblast and fibroblast mitogens such as prostaglandins, nitric oxide and cytokines (TNFα and IL-1b) that promote the production of other cytokines and the formation of hyperplasic synovium.

The concomitant use of the PPAR-γ agonist pioglitazone and methotrexate appears to be promising therapeutic strategy for rheumatoid arthritis patients [32]. Thiazolidinediones inhibit macrophage activation and contribute to the decrease of inflammatory cytokine expression and release in macrophages and monocytes. They also induced synoviocyte apoptosis and reduced secretion of TNF-α, IL-6 and IL-8 in synoviocytes of rheumatoid arthritis patients [29]. Tsubouchi Y et al., came to the same conclusion after investigating the anti-inflammatory effects of 15dPGJ2 and troglitazone as well as their impact on RA cell growth [30].

Apart from the above mentioned promising use of thiazolinediones, other studies confirmed that the activation of PPAR-γ caused by some NSAIDs may help prevent the degradation of articular cartilage in rheumatoid arthritis. It appears that NSAIDs in-

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duce the apoptosis of synovial cells, by preventing synovial hyperplasia and pannus formation [31].

7. Conclusion

PPAR-γ has been under thorough investigation for over a decade. There has been a marked increase in available data on their involvement in mammalian development, their applications in cardiology, endocrinology and bone metabolism. Even though there have been numerous studies using human and animal specimens the clinical significance of PPAR-γ remains unclear. Not all of its pathways are identified and the PPAR-γ independent actions of its ligands haven't been completely dissociated. Despite these problems, it is a fact that the use of PPAR-γ ligands could be promising for the treatment of arthritis and joint inflammation in general on the premise that its physiology is integrally understood.

Conflict of interest:

The authors declared no conflicts of interest.

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ΠΕΡΙΛΗΨΗ

Τα PPAR-γ αποτελούν μέλη μιας οικογένειας υποδοχέων που βρίσκονται σε διάφορα όργανα του σώματος. Η συσχέτισή τους με διάφορα βιοχημικά μονοπάτια να καθιστά για την εύρυθμη λειτουργεία πολλών συστημάτων μεταξύ των οποίων και του μυοσκελετικού. Εκτός από την ήδη καλά μελετημένη δράση τους στην λιπογένεση, τη διαφοροποίηση λιποκυττάρων και την ομοιοστασία της γλυκόζης, φαίνεται να συσχετίζεται με αντιφλεγμονώδη δράση, κυτταρική διαφοροποίηση και απόπτωση. Την τελευταία δεκαετία πραγματοποιείται σημαντική έρευνα για το ρόλο τους στον οστικό μεταβολισμό και την αντιμετώπιση διαφόρων μορφών αρθρίτιδας.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: αρθρίτιδα, αρθρικός χόνδρος, PPARs, παθογενετικοί μηχανισμοί

CASE REPORT

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Popliteal artery thrombosis resulting from a fracture of the proximal tibial epiphysis in a 12-year-old boy

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ABSTRACT

Proximal tibial epiphyseal fractures in children are very rare but the association to vascular complications in these types of injuries is notoriously known. However, the insidious presentation of popliteal vascular trauma especially in children can often be overlooked leading to limb-threatening ischemia. We describe a rare case of a 12-year-old boy, involved in a car accident, with popliteal artery thrombosis caused by intimal disruption, complicating a Shalter-Harris type I fracture of the proximal tibial epiphysis. Although capillary refill was less than 2sec in both feet, ipsilateral distal leg pulses were absent. The patient was treated with closed reduction and stabilization of the fracture. A reverse great saphenous vein graft was successfully used to replace the injured popliteal artery segment. In such injuries, a high index of suspicion for vascular trauma should be maintained even in a warm foot. Asymmetry of distal pulses necessitates further investigation of the arterial network. Prompt diagnosis and treatment of the fracture and vascular injury will allow limb salvage.

KEY WORDS: proximal tibial epiphysis fracture; popliteal artery thrombosis; compartment syndrome

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1. Introduction

Usually what we call epiphyseal fractures are fractures through the epiphyseal growth plate, also known as physis. The fracture line usually passes through the hyperthrophic zone of the physis which is the weakest from a bio-mechanical point of view. Physeal injuries are commonly classified according to Shalter-Harris classification [1]. Physeal injuries represent 15-30% of all fractures in children [2]. However, the incidence of proximal tibial epiphyseal fractures accounts for only 0.5-3% of all epiphyseal fractures [2, 3]. A possible explanation is that the proximal tibia epiphysis does not receive any ligamentous or tendinous attachments and any stresses are transmitted directly to the metaphysis [4]. Thus, these types of fracture should be considered as the result of high energy trauma potentially associated with neurovascular complications [5]. Shelton et al. in 1979 reported on 39 fractures of the tibia involving the proximal tibial epiphyseal cartilage, treated over a twenty-five-year period6. Two patients had disruption of the popliteal artery. In 1991 Wozasek et al., reported on 30 injuries involving the proximal tibial epiphysis, treated during a period of 28 years [5]. In their series, 3 patients presented with peripheral ischemia on admission. In sharp contrast, proximal tibia metaphyseal fractures in children are considered as low energy injuries [7]. Although blunt traumatic vascular injuries in children are relatively rare they carry a high risk of surgical intervention and contribute to significant morbidity if not diagnosed and managed early [8,9,10]. We present the case of a twelve year old boy who suffered a popliteal artery thrombosis resulting from a proximal tibial epiphyseal fracture. The patient's parents were informed that data concerning the case would be submitted for publication and they provided consent.

2. Case Report

A 12-year old boy was involved in a car accident and sustained an anterior shear injury to his left knee. Plain x-rays revealed a trans-epiphyseal fracture of the proximal tibia with anterior and medial displacement of the distal segment. Closed reduction was performed under general anesthesia and the knee was immobilized in a plaster. Twenty hours after injury, the patient complained for paresthesias and pain of his right leg and foot and transferred to our institution for further evaluation and treatment. At presentation (24 hours after accident) the patient complained for increasing pain of his right leg. On clinical examination he had hypoesthesia of the foot in the distribution of the peroneal and the tibial nerve, and absence of foot pronation and active dorsiflexion of the ankle and toes. The foot was warm and pink colored, however popliteal artery, posterior tibial and dorsalis pedis artery pulses were not palpable. Monophasic Doppler signal could be detected in the posterior tibial artery. Capillary refill of both feet was less than 2 seconds. The angiography revealed above knee popliteal artery occlusion (**Fig.1A**). Delayed images showed reconstitution of the anterior, posterior tibial and the peroneal artery distal to the trifurcation, *via* poor genicular collateral branches (**Fig.1B**). The extension of the arterial lesion could not be identified because of an apparent prograde and retrograde thrombosis. X-ray evaluation revealed a Salter Harris Type I proximal tibial epiphysis fracture. On the lateral and anterior-posterior knee X-rays, a 5 mm posterior-lateral displacement of tibial metaphysis at the fracture site was noticed.

Under general anesthesia and supine position of the patient, the fracture was anatomically re-reduced, under fluoroscopy control, with the knee placed in full flexion. The fracture was stabilized with two 2.4mm crossing Kirschner wires (KW) (**Fig. 2**). No significant ligamentous instability was detected under manipulation. An external fixator was applied at the anterior-lateral side spanning the knee in order to temporarily stabilize the knee joint and fracture and provide a stable joint for vascular dissection and repair. With the patient in prone position and through a posterior S-shaped incision the popliteal space was explored. The gastrocnemius heads for 2-3 cm proximal and distal to the level of the fracture were severely traumatized, with muscle mass loss. The posterior tibial nerve was contused with epineural hematoma infiltration. The popliteal artery was thrombosed at the level of the knee joint line for a 7 cm length distally. An intimal disruption was extended for 3 cm. Thrombectomy

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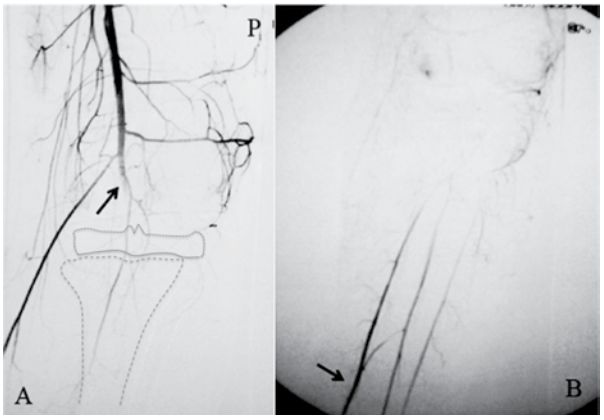

Fig 1. *Occlusion of the popliteal artery above the knee joint line (arrow). The collateral circulation is sustained by genicular branches. P: proximal B: Peripheral refilling of the tibial arteries by collateral circulation. Posterior tibialis artery (arrow)*

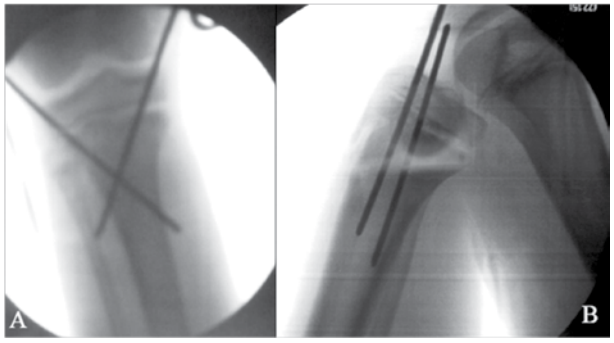

Fig 2. *A: Anteroposterior intraoperative x-ray. A: Two crossing KWs stabilize the fracture. B: lateral intraoperative x-ray. The fracture is anatomically reduced with full knee flexion*

of the posterior tibial artery was performed and slow blood back flow was noticed. Vascular repair was performed with interposition of a 5 cm reversed great saphenous vein graft, bypassing the injured part of the artery. Both proximally and distally, an end-to-end anastomosis was performed, with 6-0 polypropylene interrupted suture. Fasciotomy of the four compartments of the tibia was also performed. However the muscles of the anterior compartment were not responding to electrical stimulation. Immediately post-operatively a dorsal pedal and posterior tibial artery biphasic flow was detected by Doppler and in the second post-operative day there were palpable pulses. The 3rd post-operative day the patient developed necrosis of the muscles of the anterior and lateral compartment complicated by infection and high fever. Multiple surgical debridement procedures were undertaken resulting in complete resection of soft tissues of the anterior and lateral compartment including peroneal nerve and tibialis anterior artery. Thirty days post op the patient had well healed incisions and the external fixator was removed. Ten days later the epiphyseal fracture was considered healed and KWs were removed too.

The ultrasound color Doppler performed 3 and 12 months later, showed a patent graft without stenosis. At 12 months follow up there was a 30 mm leg length discrepancy, resulting from premature closure of the femoral and tibial physis around the knee. The patient had 110° knee flexion and full extension. Equinus – varus deformity of the foot resulted from the un-opposed action of the tibialis posterior tendon, despite the use of foot-ankle orthoses. The patient underwent Achilles tendon lengthening and anterior transfer of the posterior tibialis tendon at the age of 14 years and is currently walking with a shoe lift to compensate for the 3.5 cm leg length discrepancy.

3. Discussion

The Salter-Harris type I fracture epiphysiolysis of this case represents an equivalent to antero-lateral knee dislocation injury, stretching the popliteal artery, the tibial and the peroneal nerves and can severely threaten the leg's viability. The popliteal vessels are tethered by fibrous attachments around the knee joint at the exit of the Hunter's canal and at the upper tibial metaphysis by the soleus tendinous arch [2]. Depending on the mechanism of injury, an epiphyseal fracture even with small displacement of the distal fragment can compress the popliteal artery and jeopardize arterial flow. The severe antero-lateral translation, as in this case, stretched the artery to a degree where the intima was ruptured, the peroneal nerve sustained severe intraneural damage and the gastrocnemious heads severely contused with muscles mass loss. The collateral genicular vessels around the knee joint, if not damaged, can provide adequate blood supply for the leg and foot skin, but inadequate for the deep tissues.

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Pediatric vascular injuries differ from adult injuries in several aspects. Pediatric injuries are more difficult to detect due to insidious presentation of blunt vascular trauma [12]. In the setting of acute popliteal artery occlusion, the important clinical question is if this collateral circulation can provide adequate blood supply and oxygene for all bone and soft tissue, thus permitting limb salvage. However children's nerves and muscles are considered more susceptible to ischemia than adult's tissues [13]. In the presence of other significant bone or nerve injuries a thorough vascular evaluation, in a seemingly perfused leg, might be neglected [12]. Aitkin et al. underlined the critical importance of earliest possible reduction [11].

Minimal injuries, as endothelial disruption could be asymptomatic and heal spontaneously, but larger intimal flaps could later result in thrombus formation and ischemia. Any delay in diagnosis of arterial insufficiency and treatment after 6 hours could irreversibly affect limb viability, therefore close monitoring with repeated examination of the patient and its leg is mandatory [12,14,15,16]. Arterial spasm of the small size vessels is common feature in childhood, and this can further complicate the diagnosis of a vascular injury [12,14,17].

The described patient had a seemingly well perfused leg with pink skin and less than 2 sec capillary refill at the pulp of the toes. The posterior tibial artery at the ankle level and dorsalis pedis was not palpable but monophasic blood flow was detected. Clinically, the collateral circulation provided enough blood supply to the skin of the leg, the foot and the toes but not to the deeper musculature. The hallmark of an ischemic anterior and lateral compartment syndrome is pain that becomes intolerable with plantar flexion, not relieved by analgesic medication [18]. Additionally, revascularization after prolonged ischemia triggers the development of reperfusion injury to the ischemic tissues of the compartment [16]. Nerve dysfunction is also an early symptom of compartment syndrome [18]. In our case, the sensory deficit expressed with paresthesias and hypesthesia in the distribution of peroneal and tibial nerve, could be the result of direct nerve trauma during the initial injury. However the progressive nature of neural symptoms in association to increasing leg pain supported the clinical diagnosis of compartment syndrome. The anterior and lateral compartments were more severely affected and developed complete muscle necrosis.

In conclusion, proximal tibia epiphyseal fractures should be clearly distinguished from the innocuous proximal metaphyseal fractures in children. Epiphyseal fractures at this location are high energy injuries and can be accompanied by severe neurovascular damage. In clinical practice, these injuries should be regarded as equivalent to a knee dislocation and managed as a true emergency. A pink pulseless foot, even if Doppler examination demonstrates blood flow, should immediately raise the suspicion of vascular occlusion necessitating angiography and exploration to maximize the opportunity for limb salvage and to decrease subsequent complications.

Conflict of interest:

The authors declared no conflicts of interest.

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ΠΕΡΙΛΗΨΗ

Σε αντίθεση με τα κατάγματα της εγγύς μεταφύσεως της κνήμης, τα κατάγματα δια της εγγύς επιφύσεως της κνήμης στα παιδιά είναι αρκετά σπάνια και είναι γνωστή η συσχέτισή τους με αγγειακές κακώσεις. Η προοδευτική εξέλιξη των σημείων της θρομβώσεως της ιγνυακής αρτηρίας μπορεί εύκολα να παραβλεφθεί στα παιδιά και να οδηγήσει σε απειλητική για το άκρο ισχιαμία. Περιγράφουμε την περίπτωση ενός αγοριού 12 ετών, που τραυματίστηκε σε τροχαίο ατύχημα και υπέστη κάταγμα δια της εγγύς επίφυσης της κνήμης τύπου Shalter-Harris I και συνοδό θρόμβωση της ιγνυακής αρτηρίας λόγω κάκωσης του ενδοθηλίου. Παρότι η τριχοειδική επαναφορά ήταν μικρότερη των 2 δευτερολέπτων και στα δύο πόδια, στο τραυματισμένο άκρο δεν υπήρχαν ψηλαφητές περιφερικές σφύξεις. Ο ασθενής αντιμετωπίστηκε με κλειστή ανάταξη της παρεκτόπισης του κατάγματος και σταθεροποίηση με 2 χιαζόμενα KW. Η αποκατάσταση της ιγνυακής αρτηρίας έγινε με τη χρήση ανεστραμμένου μοσχεύματος σαφηνούς φλέβας. Σε κακώσεις εγγύς επιφύσεως της κνήμης στα παιδιά πρέπει να υπάρχει υψηλός δείκτης υποψίας αγγειακής κάκωσης ακόμα και όταν το άκρο εμφανίζεται ζεστό με τριχοειδική επαναφορά. Σε ασυμμετρία ψηλαφητικού περιφερικού σφυγμού στα κάτω άκρα πρέπει να γίνεται περαιτέρω διερεύνηση του αγγειακού δικτύου. Η έγκαιρη διάγνωση και θεραπεία της οστικής και αγγειακής κάκωσης επιτρέπει την διάσωση του σκέλους.

ΛΕΞΕΙΣ ΚΛΕΙΔΙΑ: κατάγματα κνημιαίων κονδύλων, θρόμβωση ιγνυακής αρτηρίας, σύνδρομο διαμερισματος