Medial humeral epicondylar lesions in the canine elbow
A review of the literature

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Summary
A calcified fragment near the medial epicondyle of the humerus was originally described as an ununited medial epicondyle in 1966. Since then several papers reported similar lesions as a cause of elbow lameness. The aetiology and clinical significance of those lesions is poorly known. This paper gives an overview of the veterinary and human literature in an attempt to explain the aetiology and to suggest a diagnostic protocol and treatment plan.

Introduction
Forelimb lameness in dogs is often localised in the elbow. Several developmental disorders are recognised as a cause of lameness including an ununited anconeal process, a fragmented coronoid process, osteochondritis dissecans of the humeral condyle, and incongruity. These disorders are grouped under the term ‘elbow dysplasia’ and have been well documented in the literature (1–3).

An ununited medial humeral epicondyle is a lesser-known condition and has been historically classified as elbow dysplasia (2). It was considered as a failed fusion of the medial epicondyle ossification centre to the humerus, characterised by the presence of loose ossified bodies either on the medial side of the elbow joint or distal to the medial humeral epicondyle (4).

Calcified bodies similar to those described as an ununited medial epicondyle have been reported over the past years. The most frequently described appearance is a calcified structure near the medial epicondyle (2, 5–12). One report describes spur formation at the caudal part of the medial epicondyle (8). Several terms have been suggested to describe these lesions: dystrophic calcification of the flexor tendon origins, traumatic avulsion of the humeral medial epicondyle, medial humeral condylar osteochondritis dissecans, and development of a preformed ossification centre (5–7, 9–12).

Up to now, the precise cause of medial humeral epicondylar lesions in dogs has been poorly understood and therefore it is difficult to define a correct term. It can also be questioned if all lesions can be classified under one term. Certainly the most frequently used term ‘ununited medial epicondyle’ seems incorrect since several reports indicate that there is no radiographic evidence of a failed fusion between the medial epicondyle and the humerus (9, 10, 12).

Medial humeral epicondylar lesions can cause lameness, but may also be asymptomatic since they have been described as an incidental finding (4). Therefore interpretation of the radiographic changes may be difficult, which may then lead to inadequate treatment.

The pathological changes diagnosed in the canine elbow seem to have similarities to certain disorders in human medicine. Of note are little leaguer’s elbow and golfer’s elbow, both of which are characterised by comparable images to those seen with medial epicondylar lesions in dogs (13, 14). Since these conditions have been well documented in human literature, this information can be used to further investigate medial epicondylar lesions in dogs.

The purpose of this paper is to describe the different forms of medial epicondylar lesions reported in veterinary literature. By reviewing the reported cases and comparing them to similar disorders in human medicine, an attempt is made to explain the aetiology and to propose a diagnostic protocol and treatment plan.

Development of the elbow joint and anatomy of the flexor muscles
The elbow joint is a complex synovial hinge joint formed by the distal part of the humerus and the proximal part of the radius and the ulna. It is supported with strong collateral ligaments, and several muscles originate from it and insert on it (15).

The distal end of the humerus develops from three centres of ossification: one within the capitulum, one in the trochlea, and another in the caudal portion of the medial epicondyle. The medial epicondylar epiphysis forms the medial epicondyle, from which many carpal and digital flexor muscle groups originate. Radiographically, the centre of ossification for each part of the condyle appears during the second to third

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week after birth. The ossification centre of the medial epicondyle appears four to eight weeks after birth (depending on the breed) and fuses to the distal humeral physis at approximately 10 weeks (2, 15, 16) (Fig. 1).

The muscles of the flexor group originate from the medial epicondyle and attach to it through a short tendinous part (17). The flexor carpi radialis muscle arises from the medial epicondyle and inserts on the proximal palmar surface of metacarpals II and III. It runs caudal to the pronator teres, which arises from the medial epicondyle, and inserts on the medial side of the radius. The superficial digital flexor muscle is located caudal to the flexor carpi radialis, arises from the medial epicondyle, and inserts with four tendons on the proximal palmar part of the middle phalanx of digits II, III, IV and V. The origin of the flexor carpi ulnaris has two separate muscles; an ulnar and a humeral head. The short balled 

ulnar head arises from the caudal part of the olecranon and inserts distally as a tendon on the accessory carpal bone. The deep digital flexor muscle has three points of origin: the medial epicondyle, the proximal part of the ulna, and the medial part of the radius. All three heads of the deep digital flexor muscle have one common distal tendon which divides into five tendons and inserts on the proximal palmar side of digits I, II, III, IV and V (17) (Fig. 2 and 3).

Fig. 1  Lateral elbow radiographs, illustrating the development of the medial epicondyle, of a) a mixed breed at six weeks, b) a Border Collie at three months and c) a mixed breed at four months of age. 1: Two separate centres of ossification of the humeral condyle are superimposed. 2: The large ossification centre of the medial epicondyle is partially ossified in b) and completely in c). 3: The proximal radial epiphysis. 4: Separate centre for the olecranon process.

Fig. 2  The superficial muscles at the medial side of a right forelimb. Image based on drawing made by B. Van Ryssen.

Fig. 3  The deep muscles at the medial side of a right forelimb. Image based on drawing made by B. Van Ryssen.
Medial humeral epicondylar lesions

Different types of lesions have been described and different terms have been used, suggesting a different aetiology. Nevertheless, most reports describe similar lesions. Based on the terminology the authors used to describe the pathology, an overview of the reported lesions is given.

Ununited medial epicondyle

The first report on calcified bodies near the medial humeral epicondyle mentioned an eight-month old German Shepherd dog in 1966 (2). Islands of bone (1.5 cm long and 0.5 cm wide) caudal and distal to the medial epicondyle were diagnosed on lateral and craniocaudal radiographs (2). On histopathology, the calcifications consisted of normal cancellous bone which had been attached to the humerus by fibrocartilage (2). This lesion was suggested to be an ununited medial epicondyle and was considered as a form of elbow dysplasia (2). However, in later reports, several authors demonstrated the absence of radiographic evidence of failed fusion of the medial epicondyle to the humerus, thus suggesting that ‘ununited medial epicondyle’ is an incorrect term to describe those calcified bodies near the medial epicondyle (9, 10).

In later reports, the existence of an ununited medial epicondyle is sustained; however it is explained as a form of osteochondrosis in which fragments of the cartilage avulse with the attachment of the muscles. The rationale in believing that osteochondrosis is the underlying problem is that an ununited medial epicondyle is often bilateral and is often seen without trauma (4) (Fig. 4 [4]).

A recent study reported on the appearance of an ununited medial epicondyle in a group of related Labrador Retrievers that were diagnosed at six and eight years of age. The authors pointed out that this lesion does have a hereditary character, however it has an unknown clinical relevance (18). No other reports of the inheritance of this condition were found. According to the latest guidelines of the International Elbow Working Group, an ununited medial epicondyle is no longer included in the elbow dysplasia scoring system (19).

Preformed ossification centre

Calcified bodies similar to those described in the reports on an ununited medial epicondyle, which were located medially at the origin of the humeral head of the deep digital flexor muscle, were diagnosed in the elbow joint capsule (5) (Fig. 5 [5]). Histopathological examination had revealed

**Fig. 4** Different examples of an ‘ununited medial epicondyle’. A) Lateral view of an ununited medial epicondyle. B) Lateral view of a large ununited medial epicondyle in another dog. C) Craniocaudal view of the same dog shown in B. The densities seen in site B on the craniocaudal view are not obvious on the lateral projection (white arrowheads). On the craniocaudal view, the densities in site a are obscured by the humerus, but are evident on the lateral view (black arrows) (Printed with kind permission from WB Saunders Elsevier: Piermattei DL, Flo GL, DeCamp CE. Brinker, Piermattei and Flo’s Handbook of Small Orthopedics and Fracture Repair. 4th ed. Philadelp-hia: WB Saunders Elsevier; 2006 [4]).

**Fig. 5** Example of a ‘preformed ossification centre’: calcified body, bilaterally in the elbow joint capsule, located medially at the origin of the humeral head of the deep digital flexor muscle (white arrows) (Printed with kind permission from John Wiley and Sons: Grondalen J, Braut T. Lameness in two young dogs caused by a calcified body in the joint capsule of the elbow. J Small Anim Pract 1976; 17: 681-684. [5]).
bone trabeculae centrally, degenerated cartilage at the end of the calcified body and columns of irregular cartilage infiltrated by fibrous tissue towards the tendon of the humeral head of the deep digital flexor muscle (5). Instead of describing this lesion as an ununited medial epicondyle, it was suggested that this calcified mass had developed from a preformed ossification centre, comparable to a sesamoid bone. The calcified body was considered as a possible cause for the development of osteoarthritis (5).

**Traumatic avulsion of the medial humeral epicondyle**

Several papers reported on calcified bodies near the medial epicondyle as a traumatic avulsion fracture of a part of the epicondyle. A few case reports describe traumatic avulsions in immature dogs that were presented with acute lameness (6, 7, 9, 10). On radiographs, an irregularly shaped calcified mass below the medial humeral epicondyle and a misshapen medial epicondyle can be diagnosed (6) (Fig. 6 [6, 7]). Histologically, the bony fragment is covered by cartilage at the level of the tendino-osseous junction (7). It is suggested that when a fragment of the immature medial epicondyle is separated by trauma, it continues to grow with nourishment from the attaching flexor muscles (9). Traumatic avulsion in the immature dog would presumably happen before the fusion of the growth centre at 10 weeks (9, 16).

In human medicine a similar condition has been described as little leaguer’s elbow. It involves young children (baseball players) in which valgus stress across the elbow results in injury to the week apophyseal plate of the medial epicondyle. This results in complete avulsion of the epicondyle, whereas in dogs only a small part of the medial humeral epicondyle is separated (13, 20) (Fig. 7 [21]).

Traumatic avulsion of the medial epicondyle can also appear in mature dogs. Two different presentations are described: acute cases associated with recent trauma and chronic cases without evident trauma (7). On radiographs a bony fragment is separated from the medial epicondyle. A unilateral appearance of the lesion suggests a traumatic cause while a developmental cause is more likely in bilateral lesions. To differentiate between both disorders, radiographs of both elbows are recommended (7).

**Dystrophic calcification of the flexor muscle origins**

Another frequently described theory for the existence of calcified bodies is dystrophic calcification of the origin of the flexor muscles. According to several case reports, the calcified bodies were located mainly in the tendinous endpiece of the flexor muscles (5, 9–12) (Fig. 8 [12]). This led to the presumption that the calcified bodies were metaplastic changes of tendon tissue, which connects the flexor muscles to the medial epicondyle. This presumption was supported by the histological examination, which revealed bone trabeculae changing into tendon tissue or surrounded by fibrocartilaginous tissue. The histopathological diagnosis was tendinitis ossificans with reactive new bone formation.
proliferation (5, 9, 10, 12). Because the joint capsule has extensions under the flexor carpi radialis muscle and deep digital flexor muscle, it may also be involved in the pathology (9, 15). All flexor muscles can be involved, but the deep digital flexor muscle seems to be predisposed (12).

The real cause for the development of dystrophic calcifications is still unknown and most papers report it as a solitary elbow problem (2, 5, 7, 8). However, in some studies elbow dysplasia, such as incongruity and fragmented coronoid process with chronic inflammation, was associated with the calcifications (9, 10, 12). In two studies describing a total of five dogs, incongruity was found in three dogs, and in one study of 26 joints, a fragmented coronoid process was diagnosed in six joints (9, 10, 12). Whether chronic arthrosis can lead to dystrophic calcifications remains uncertain (9, 12). In a study of 26 elbow joints with calcifications, only 13 had moderate to severe arthrosis. The remaining 13 elbow joints did not have any arthrosis, or just a mild form (12).

Another possible cause of dystrophic calcification within the flexor muscles is increased stress (9, 10, 12, 14, 22). In human medicine, overuse injuries are well known and are described as enthesopathy or insertional tendinopathy. Tendinopathy refers to an overuse injury of the tendon close to the insertion on the bone, while enthesopathy is defined as a pathological change affecting the enthesis, at which the tendon attaches to the bone (14). ‘Tennis elbow’ is known as lateral epicondylitis or enthesopathy of the tendons attaching to the lateral epicondyly. It occurs more frequently than ‘golfer’s elbow’ or medial epicondylitis, which is located around the medial epicondyly. Both these conditions are caused by repeated microtrauma and stress and are considered as overuse injuries with a multifactorial origin: besides intrinsic factors such as anatomical variations, malalignment problems and muscle weakness, there are extrinsic factors of which excessive loading is the main pathological stimulus for degeneration (23, 24). The enthesis is vulnerable to overuse injuries due to the stress concentration at the hard-soft tissue interface (14, 22).

**Spur formation at the caudal part of the medial humeral epicondyle**

Bony spur formation or enthesophytes are bony outgrowths, extending from the skeleton into a tendon at its enthesis (14). Bony spur formation at the caudal aspect of the medial epicondyle has not received attention as a clinical problem since it has only been reported once in veterinary literature. In this paper, four cases of bony spur formation are described (8) (Fig. 9 [8]). The authors suggested that trauma to the superficial digital flexor muscle insertion led to a local bony proliferation, and therefore the disorder was described as a traumatic enthesopathy. Histology of the resected humeral epicondylar spur revealed...
woven and compact bone with clusters of chondrocytes, surrounded by dense and loose fibrous connective tissue (8). It was believed that in the early stage of this disorder pain and lameness may occur without the obvious presence of enthesophytes (8). In two of the four described cases, simultaneous osteoarthritic changes were present. It was therefore unclear whether the epicondylar spur was a manifestation of osteoarthritis or the primary problem causing osteoarthritis (8). In human literature, it is suggested that osteophyte and enthesophyte formation are linked and that they both represent a skeletal response to stress. While osteophytes develop to adapt to a changed loading on synovial joints caused by injury or disease, enthesophytes represent a comparable adaptation at entheses (14). Because an enthesophyte consists of fibrocartilage at the tip, it is believed that bony spur formation is an extension of normal enthesis growth in the direction of the tendon reflecting the orientation of the fibrocartilage cells (14, 25).

Clinical data

According to the current literature, there does not seem to be a breed predisposition for medial humeral epicondylar lesions. The reported breeds are German Shepherd dog, Bernese Mountain dog, Rottweiler, Newfoundland, Labrador Retriever, Collie, Basenji, Bassett Hound, Sheltie, Dalmatian, and Airedale Terrier (2, 7–10, 12). Medial humeral epicondylar lesions seem to occur in dogs of all ages (7, 9, 10, 12). Traumatic fragmentation was seen in a number of dogs before 10 weeks of age, after which the medial epicondylar epiphyseal fuses with the humerus (2, 9).

Medial epicondylar lesions may cause lameness or may be asymptomatic (4). The presence of a calcification or spur at the medial epicondyle is often considered as a coincidental finding without any clinical significance (4, 8). However, several reports attributed lameness to the presence of these lesions (2, 5, 7–12). Other clinical findings are non-specific and comparable to those seen with any other elbow pathology: a painful and swollen elbow joint, painful and limited flexion and extension sometimes associated with crepitation, and in chronic cases atrophy of the shoulder muscles (2, 7–10, 12).

Diagnostics

Radiographic examination is the first step in visualising the bony changes of the elbow joint. The lateral flexed and craniocaudal view should be examined carefully to determine the presence, shape and location of the calcified bodies and the presence of spur formation (4, 8). According to the guidelines of the International Elbow Working Group, a spur is described as a sign of osteoarthrosis (26). A calcification is most frequently diagnosed on the craniocaudal view, and is located caudoventral, medial or distal to the medial epicondyle at the level of the joint space (4, 12) (Fig. 8 [9, 12]). The calcified body may be missed on the lateral view because of superimposition of the humerus and radius (4). Care should be taken not to confuse the calcification with a displaced osteochondritis dissecans flap (27, 28). However when the fragment is visible on the lateral view, the flexed and extended projection can be used to determine whether the fragment hinges dorsally on the flexed view, demonstrating the localisation within the flexor muscles (9). Since the presence of concurrent elbow problems has been described, the joint should be inspected for other lesions to exclude them as a possible cause of lameness and to make sure that lameness is indeed related to the radiographic lesion of the humeral epicondyle (9, 10, 12).

Ultrasoundography of both elbows is recommended to evaluate whether the flexor muscles are involved in the process. In man, it is a commonly used technique. The main ultrasonographic findings of flexor enthesisopathy are pre-insertional hypoechoic swelling, outward bowing, and thickening of the common tendon of the flexor muscles. The tendon appears to be heterogeneous with decreased echogenicity and focal or diffuse areas of irregular fibrillar appearance and ill-defined margins with partial or complete tears. Additionally cortical irregularities at the medial epicondyle (spur formation) and intratendinous calcifications can be detected (29, 30). Comparison between both elbows is necessary in order to notice subtle differences (30). Advantages of ultrasoundography are the ability to examine soft tissue which is poorly visible on radiographs, real-time assessment of joint and tendon movement under manipulation, and lack of ionizing radiation (31). Limitations are operator dependence, a long learning curve, and the need for high resolution ultrasound equipment (30).

Fig. 10 An axial T1 weighted spin echo magnetic resonance image (MRI) (A) and an axial fat suppressed T2 weighted spin echo MRI (B) of a human elbow joint with medial epicondylitis. A) Abnormal intermediate signal intensity within a thickened common flexor tendon origin (black arrow). B) Abnormal intermediate to high signal intensity within a thickened common flexor tendon origin (white arrow). A high signal intensity is also visible in the superficial subcutaneous tissue at the lateral side of the elbow (white arrowhead), which most likely represented in homogeneous fat suppression (Printed with kind permission from Springer Science+Business Media: Skeletal Radiol, Magnetic resonance imaging findings in patients with medial epicondylitis. Volume 5, 2005, pages 196-202. Kijowski R, De Smet AA. [33]).
Magnetic resonance imaging (MRI) is another diagnostic tool to confirm the involvement of flexor muscles in medial epicondylar lesions, commonly used in human medicine (32–34). The MRI findings of medial epicondylitis are increased signal intensity within the common flexor tendon on both T1-weighted and T2-weighted images, tendon thickening, small joint effusions, and periostitis (33, 35) (Fig. 10 [33]). Advantages include visualisation of the anatomy in multiple planes, superior soft-tissue detail, and avoidance of ionizing radiation exposure. Disadvantages are the need for general anaesthesia and the high costs of the equipment (32). In a clinical study ultrasonography was compared with MRI for diagnosing epicondylitis in man. The sensitivity for detecting epicondylitis ranged from 64% to 82% for sonography, and from 90% to 100% for MRI. Specificity ranged from 67% to 100% for sonography, and from 83% to 100% for MRI. Sonography seems adequate for diagnosing epicondylitis in the majority of the patients, allowing MRI to be reserved for patients with symptoms whose sonographic findings are normal (23).

Although computed tomography (CT) is not used as a standard diagnostic tool in human medicine to diagnose epicondylitis, it can be of interest in diagnosing lesions of the medial epicondyl and the flexor muscles using either a bone or soft tissue window (36). As with MRI, CT requires general anaesthesia, but image acquisition is faster and the images are more detailed (36).

Treatment

Asymptomatic cases radiographically discovered as incidental findings are supposed to be left untreated, although follow-up studies of these cases are not available. Symptomatic cases can be treated either conservatively or surgically. Due to the small number of cases and the short follow-up period, the results of the treatment should be interpreted with caution.

Conservative

The optimal medical treatment for medial epicondylar lesions has not yet been documented in veterinary literature. A treatment of four weeks of pentosan polysulphate sodium did not have any effect (10). Prednisolone and mefenamic acid were reported to be effective, but lameness recurred when treatment was stopped (8).

In human medicine, conservative therapy is the first step in treating medial epicondylitis. It has been described as highly successful, although there is not any information about the long-term results. The treatment is initiated with the application of ice to the affected elbow for a period of 15–20 minutes three to four times a day, combined with oral nonsteroidal anti-inflammatory medication for a period of 14 days. In cases of little response to this treatment, a period of night splinting combined with local corticosteroid injection around the affected tendon insertion is suggested. As soon as the symptoms have improved, a guided rehabilitation program can start (37).

Surgical

In dogs, traumatic avulsion of the medial epicondyloide is treated surgically by fixing the chip with a lag screw (6). Only when the fragment is too small or brittle it is suggested to remove it. Postoperative external support using a supportive bandage, a modified Robert-Jones bandage or a half splint is recommended in order to prevent excessive elbow abduction and hyperextension of the digits (7). Information on the results is limited: in two dogs in which a lag screw was used, lameness had disappeared at six months and one year respectively after surgery. One dog was still sound two years after removal of the chip (7).

In contrast to dogs, treatment of little leaguer’s elbow consists of complete rest, ice-packs and analgesic medication; surgery however is rarely necessary (13). Minimally displaced fractures can be treated with splint immobilisation, and fractures with more than 5 mm displacement should be treated surgically by internal fixation (13).

Several papers report on the surgical treatment of lesions other than a traumatic avulsion in dogs that do not benefit from conservative therapy (8, 10, 12). A standard surgical procedure for the removal of the calcified bodies has not been described in veterinary literature, although all papers report on a similar surgical approach. Via a medial incision, the superficial and deep fascia are separated, the origin of the different flexor muscles is dissected, the involved flexor muscle is identified, and the calcification is isolated and removed. Any bony spur formation is removed using a rongeur. The remaining muscle or tendon stump is sutured to the proximal part of the involved muscle (5, 8–10, 12). Because the calcifications can be associated with other forms of elbow dysplasia, the arthroscopic or surgical inspection of the elbow joint for cartilaginous or bony fragments is recommended (9, 10, 12). A good outcome has been reported following surgery: the largest and also most recent study reports on the treatment of 22 elbows: 15 dogs did not have any lameness at all, five dogs were only lame after heavy exercise and two dogs were still lame after an average follow-up period of 18 months (12). Three dogs with spur formation and one dog with spur formation and calcifications improved significantly after an average postoperative period of four months (8). The need for surgical removal of the calcification and the good outcome of that treatment can be questioned since no long-term follow-up studies have been done. However, in a study about mineralisation of the supraspinatus muscle, reformation of the fragment five years after surgical removal has been described without recurrence of lameness (38).

In human medicine, a standard surgical procedure for medial epicondylitis consists of the excision of the pathological tissue, stimulating the healing response by improving local vascularity, reattaching any elevated tendon to the epicondyle, and repairing the remaining defect. Postoper-

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atively a splint is placed (37). The prognosis seems good: A review of 35 human patients with medial epicondylitis who underwent surgical treatment reported 88% good to excellent results after an average follow-up period of six years (39). Another report of 26 patients mentioned 87% success after an average follow-up period of seven years (40).

Conclusion

Pathological changes in the area of the medial humeral epicondyle are most frequently described as calcified bodies near the medial epicondyle while spur formation is only mentioned in one report (2, 5–12). Several terms have been used to describe these lesions: an ununited medial epicondyle, dystrophic calcification of the flexor tendon origins, traumatic avulsion of the humeral medial epicondyle, medial humeral condylar osteochondritis dissecans, and development of a preformed ossification centre (2, 4–7, 9–12). The varying description and illustrations (Fig. 4 to 8) of the reported cases suggest a different aetiology, although there are several similarities between the different classifications. Equally, histopathological examination of the resected calcified tissue revealed similarities: all calcifications or fragments contained bone surrounded by cartilage or fibrocartilaginous tissue, as well as a spur consisting of compact bone with clusters of cartilage. However, histopathological findings do not explain the cause of the problem.

A comparison of the findings in dogs with similar pathology in man supports either a traumatic cause or an overuse lesion with an insidious onset in most cases. Although in some cases a developmental problem could be the cause, the term ‘ununited medial epicondyle’ does not reflect the aetiology of the different forms and should therefore be replaced by a more appropriate term. Therefore the authors propose the term ‘flexor enthesopathy’ to describe the presence of pathological changes within the flexor muscles and their attachment to the medial epicondyle, without referring to the cause of the problem.

Changes at the medial epicondyle are often considered as clinically unimportant lesions, although they were the cause of lameness in the reviewed reports. It is also the authors’ experience that these lesions should be included in the differential diagnosis of elbow problems as a primary cause of lameness. When the first radiographic screening reveals epicondylar changes, further diagnosis should include ultrasonography and eventually should be followed by MRI.

Conservative treatment does not appear to be successful in dogs, whereas in the majority of reported cases surgical removal of the calcifications led to a significant improvement. In man however, conservative treatment is the standard treatment. The good results may be explained by the early diagnosis and appropriate treatment. An early and correct diagnosis might improve the results of conservative treatment in dogs.

A study of a large series of dogs affected with medial epicondylar lesions is being carried out by the authors and should reveal more information on the diagnosis and management of the different types.

References