Scapular winging

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ABSTRACT

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This review explores the causes of scapula winging, with overview of the relevant anatomy, proposed aetiology and treatment. Particular focus is given to lesions of the long thoracic nerve, which is reported to be the most common aetiological factor.

INTRODUCTION

Scapular winging is painful and incapacitating [1,2]. The composite movements of the scapula in rotation, abduction and tilting are essential for normal shoulder function and, if this anchored gliding of the scapula (against the posterior chest wall) is lost, then shoulder abduction can be limited to 90° or less and the ability to manipulate heavy objects is severely limited [3]. Daily tasks, including brushing hair and cleaning teeth, are difficult and the scapula prominence can be cosmetically distressing [4–7].

Scapula winging is characterized by a failure of the dynamic stabilizing structures that keep the scapula anchored to the chest wall, leading to a prominence of the scapula (Fig. 1). These stabilizers are comprised of three main muscle groups: serratus anterior, trapezius and rhomboids major and minor. The most common cause of scapular winging is serratus anterior palsy but, ever since Velpeau first described winging from isolated serratus anterior palsy in 1837, the true aetiology has remained an enigma [8]. Damage to the nerve supply, the muscles themselves or their attachments can cause winging, although serratus anterior palsy and lesions of the long thoracic nerve have received most attention in the literature. Furthermore, diffuse paralysis of scapula stabilizers from fascioscapulohumeral dystrophy or brachial plexus injury and scapula bony abnormalities, including osteochondroma or fracture, can produce winging. Numerous other secondary causes of winging have been described, including voluntary or habitual winging [2,9].

This review explores the causes of scapula winging, with an overview of the relevant anatomy, proposed aetiology and treatment. Particular focus is given to lesions of the long thoracic nerve, which is reported to be the most common aetiological factor.

SERRATUS ANTERIOR PALSY

The commonest cause of scapular winging is isolated serratus anterior paralysis producing a medial winging of the scapular (Fig. 1) [10]. In 1940, Overpeck and Ghormley [1] reported an incidence of 0.0026% (28 cases) at the Mayo clinic, contrasting with Gregg et al. [11] in 1979 who suggested that it is a more common entity, with 10 cases over 3 years amongst 20 surgeons at their institution.

Anatomy

The serratus anterior is composed of three main parts with a total of seven to 10 slips (the most common is eight slips found in 48%) and has its blood supply from the long thoracic artery superiorly and branches of the thoracodorsal artery inferiorly [12]. It has multiple cervical root innervations via the long thoracic nerve. The upper part is a cylindrical mass connecting the superior angle of the scapula to the first two ribs and it provides stability to the superior scapula angle, allowing rotation. The middle section connects the vertebral (medial) boarder of the scapula to the second, third and fourth ribs and contributes to scapular abduction. The lower serratus anterior is composed of thicker digitations and connects the inferior scapula angle to ribs 5 to 10. It has peak activity in the arc of arm abduction, combining with the upper trapezius to provide scapular abduction and upward rotation [3,12–14].

The sole innervation of the serratus anterior is the long thoracic nerve, first described by Bell and Bell in 1827 as the external respiratory nerve [15]. This motor nerve is unique, arising directly from the anterior rami of cervical roots five, six and seven. Anatomical variants include a contribution from C4 in up to 13% and an absence of the seventh root component in 8% [3,16,17].

Fig. 1 Medial scapula winging from serratus anterior palsy.

It has a mean length of 27 cm and diameter of 3 mm. Its course has considerable variation between individuals with direct penetration of C5 to the upper serratus in up to 65%, although it universally travels posterior to the axillary vessels and trunks of the brachial plexus and is found an average of 2.8 cm behind the clavicle [2,3,18]. In over half of individuals, the C5 and C6 contributions lie between the middle and posterior scalene, with the remainder travelling either through the middle scalene or anterior to the middle scalene [18]. The C7 contribution is always anterior to the middle scalene and, in the majority, joins C5 and C6 at the level of the second rib to complete the formation of the long thoracic nerve. The nerve then runs superficial to the serratus anterior for its entire course, giving branches to every digitation (Fig. 2) [5]. It intersects the most inferior and dominant serratus branch of the thoracodorsal artery at a point coined the 'crow's foot', forming a consistent and reliable landmark [12]. The relevance of this detailed anatomy will become evident as the sites of potential nerve damage are discussed.

Clinical presentation

The initial symptoms of serratus anterior palsy are often vague, with pain along the base of the neck and over the scapula and deltoid, which is then replaced by weakness and scapular winging after 10 days to 2 weeks [1,9,17,19–21]. In rare cases with definite acute trauma, the winging can be immediate [22]. The predominant features at presentation are a loss of shoulder power and limited abduction in two-thirds of individuals. A significant number also present with apparent shoulder instability, such as 17% in a series by Fery and Sommelet [4]. Warner and Navarro

Fig. 2 The long thoracic nerve with branches to each digitation of the serratus anterior.

report five patients with serratus anterior dysfunction who had 17 prior unsuccessful surgeries for incorrect diagnoses, including shoulder instability, impingement, acromioclavicular joint disease and biceps tendonitis [23]. The shoulder instability may result from a failure of the scapula to provide a stable platform for glenohumeral rotation and impingement may be a result of the coracoacromial arch being pulled downwards by the winged scapula [23]. The majority of cases are right-sided across the literature, although the reason for this remains a mystery [1,4,16,17,24].

On examination, there is medial winging of the scapula, made more prominent by forward arm flexion, with abduction limited to 90 °C or less. Manually stabilizing the scapula during examination can improve flexion and abduction and relieve pain (Fig. 3) [23]. Electromyography (EMG) is a valuable adjunct to clinical assessment in defining the involvement of the long thoracic nerve [10,23].

Aetiology

The largest series of isolated paralysis of the serratus anterior comprising 197 cases over 11 years, suggests several causative factors: strenuous work or sports (35%), acute trauma (26%), idiopathic (15%), invasive procedures (11%), infection (7%), anaesthesia (5%) and miscellaneous (sleeping position or cold) (1%) [25]. The next largest series reporting 111 cases over a 30-year-period adds 5% occurring post partum and 7% after injections [16].

The underlying pathology leading to serratus anterior palsy in the majority of cases is presumed to be long thoracic nerve injury, comprising the most common single nerve lesion in the shoulder region [16,26]. Three main theories exist to explain long thoracic nerve injury in isolated serratus anterior palsy. These are

Fig. 3 Arm abduction improves with manual stabilization of the medially winging scapula.

traction injuries, compressive injuries and neuralgic amyotrophy [17,27]. Apart from lesions to the long thoracic nerve, rare cases of scapular winging from traumatic avulsion of the serratus anterior or displaced inferior pole scapular fractures have been described [28,29]. EMG is valuable in demonstrating isolated long thoracic nerve injury as the cause rather than the aforementioned traumatic avulsion or secondary winging from another cause [10,23,29–31].

Historically, trauma has been considered to account for long thoracic nerve injury in over 50% of cases across the literature [11,16,22,25,32]. This trauma ranges from definite acute trauma with rapid onset of scapular winging to the more common scenario of presumed repetitive trauma from activities including repetitive occupational tasks and sports.

Different sites of proposed compressive neuropathy include the scalene muscles, between the coracoid and first or second rib, over the second rib, from inflamed bursae and at the anteroinferior scapular boarder [13,22,26,33–39]. Compression of the long thoracic nerve in the scalene muscles is a controversial theory because the C7 component does not join the nerve until after it passes through (or adjacent to) the middle scalene muscle and the dorsal thoracic nerve is also uninvolved despite running a similar course in the middle scalene [17,22,34]. This theory has support, however, including two series of microneurolysis of the long thoracic nerve in the middle scalene, with improvement of up to 98% for those with symptom duration less than 10 years [36–38,40].

Horwitz and Trocantins' large cadaveric series of 100 dissections found that the long thoracic nerve was angulated as it crosses the second rib and it was concluded that the likely site of injury was between the coracoid and the first or second rib [33]. Four bursae (subcoracoid, subscapular, accessory and supra-coracoid) were also identified that could cause compression if inflamed. All of these identified compression sites were exaggerated in a

supine posture, which may explain the cases presenting after prolonged obstetric or anaesthetic times [33,34]. A further report of four cadaveric dissections by Gozna and Harris demonstrated compression between the underside of the scapula and the second rib, although the site is inaccessible for surgical exploration [22]. As the nerve passes over the second rib, there is potential for bow stringing and traction injury because two separate cadaveric studies have shown a doubling in length of the long thoracic nerve between the scalene muscles and superior serratus anterior when the arm is raised and the head is turned [11,26,35]. Rupture of nerve fibres within a fascicle occurs with elongation of approximately 4%, although bow stringing was demonstrated in normal subjects, suggesting that this 30-cm long nerve has a tolerance for stretch [26,34].

A dissection of 21 cadavers failed to demonstrate a site of compression at the first and second ribs and, instead, suggested that the nerve is vulnerable to compression and traction both anterior to the lower scapula and at the level of the eighth and ninth ribs, where it lies relatively unprotected from external compressive forces such as crutches [6,13]. Compression in the lower anterior scapula corresponds to the 'crow's foot' landmark where the long thoracic nerve and dominant serratus branch of the thoracodorsal artery converge and the nerve is potentially tethered in the facial sling [12,13].

The diverse range of proposed sites of compression and multiple possible traumatic causes of long thoracic nerve injury is typical of many nerve palsies and neuritides and illustrates that the underlying pathology in many cases is something of an enigma [16]. It is easy to rationalize the damage caused to the nerve from an acute traumatic event such as direct blow in a road traffic collision with acute onset of scapular winging, or iatrogenic damage from needle thoracocentesis [11,41]. The mechanism of nerve injury from repetitive minor trauma where no specific event can be identified

is more difficult to appreciate, yet this supposed aetiology is more common across the literature [4,21,30,42–46,41]. In this scenario with no clear acute traumatic event, the classic presentation is of an initial onset of severe pain, which subsides over a period of approximately 2 weeks before the weakness and scapular winging becomes apparent. This clinical picture is also seen in cases of neuralgic amyotrophy, which was described by Parsonage and Turner in 1948 as brachial plexus neuritis, where isolated unilateral long thoracic nerve palsy was the most common finding involving 30 of their 136 patients [27]. Neuralgic amyotrophy may therefore account for a number of those cases presumed to have an insidious traumatic cause and also those developing scapular winging after an insult that may produce an immunological or inflammatory response such as after a routine immunization or post surgery [31].

Treatment

To plan effective treatment, an understanding of the natural history of scapular winging from isolated long thoracic nerve palsy is required because this involves spontaneous recovery in the majority of patients within 2 years [6,16,21,22,24,32,33,46,47]. Recovery includes regaining shoulder function and resolution of scapula winging for most patients, although many are left with a mild degree of endurance limitation in the affected shoulder over the long term [11,31,33]. A clear consensus has emerged that isolated long thoracic nerve palsy should therefore be treated expectantly for 18 months to 2 years in the majority of cases, with an avoidance of heavy precipitating activities during that time. Despite early apparent success, bracing the shoulder girdle as a supportive measure in conservative management has fallen from favour, with poor compliance and little proven benefit [1,11]. For the minority who do not recover well over this timescale, there is a trend towards them having had an initial acute traumatic insult [19,31].

For those unsuitable or who fail conservative management, the surgical options for scapular winging from serratus anterior paralysis include muscle transfers, neurolysis of the long thoracic nerve, scapulothoracic fusion and nerve transfers.

The first reported surgery for scapular winging from serratus anterior palsy was in 1904 where Tubby performed a pectoralis major transfer in a 7-year-old girl, restoring her ability to elevate the arm above the horizontal and 'thrust the arm forward as in fencing' [48]. This was refined by Marmor and Bechtol [44] to include a transfer of the sternal head only and others have used augmentation with a coiled fascia lata or hamstrings graft with good results [23,44,35,39,49–52]. More recent anatomical studies have shown that the split pectoralis major tendon is long enough for direct transfer without graft augmentation [10,53]. Early reports of successful muscle transfer surgery, however, were for patients who had a symptom duration of less than 18 months and it could be argued their good results simply represented the natural history of the condition [11,44,48,52]. Subsequently, more stringent reports, with a pre-operative EMG confirming chronic serratus anterior denervation after a period of conservative management, have demonstrated success with good or excellent outcomes in 60% to 100% of patients, which were sustained

at follow-up as long as 13 years post surgery [4,35,39,50,51,54]. Many of those with a successful reported outcome have some mild weakness of abduction, although mean arm abduction is improved by up to 75° C [35,39,54]. Reported complications include a failure of the pectoralis major muscle transfer attachment at the scapula, infection, unsatisfactory cosmesis [particularly breast asymmetry in women], glenohumeral adhesive capsulitis and fascia lata graft donor site seroma [23,35,39,49,50]. Other muscle transfers include pectoralis minor and the rhomboids, although neither are as effective as the above mentioned pectoralis major [55,56]. A possible predictor of successful surgery is symptom relief and improved function with manual stabilization of the scapula during examination pre-operatively (Fig. 3) [23].

Neurolysis of the long thoracic nerve in the scalene muscles has shown excellent results in the literature. Nath et al. reported 50 procedures where the nerve was decompressed as it traverses the middle scalene muscle with 98% improvement in those with symptoms of a duration less than 10 years [36]. A second series of 13 procedures by Nath and Melcher demonstrated an average improvement of arm abduction of 59° C with 24 hours of surgery [37]. A further four patients had supraclavicular neurolysis of the long thoracic nerve in the scalene muscles in a series reported by Disa et al. with resolution of symptoms in all four [38]. These outcomes are difficult to explain in light of the multiple possible sights of compression proposed, although they represent independent benefit beyond the natural history, with 10 patients in Nath's group having a pre-operative symptom duration of over 6 years [36].

Scapulothoracic fusion has generally been reserved for those with failed pectoralis major transfer surgery, scapular winging from diffuse neuromuscular disorders or when conventional muscle transfer is contraindicated [39,57–61]. Bizot et al. reported the results of scapulothoracic fusion in 10 patients as a primary alternative to pectoralis major transfer in scapular winging from isolated serratus anterior palsy [57]. Shoulder movement was limited to one-third of normal and a gain in abduction of only 20° C from before surgery was seen with complications of three non-unions, two pleural effusions and a frozen shoulder. It was concluded that difficulties in achieving fusion in an adequate position, poor mobility post surgery and high complication rates indicated that scapulothoracic fusion should not be the primary procedure in isolated serratus anterior palsy [57]. More recently, however, good results have been reported for scapulothoracic fusion as a primary surgical treatment for those who have failed conservative management, with high satisfaction levels in 82% of patients at mean follow-up of 5 years. This series of 42 patients included patients with dystrophic and nondystrophic scapula winging, including nine with isolated serratus anterior palsy [62].

Nerve transfer with microneurosurgical techniques is a developing area with reports of successful resolution of winging from transfers, including the thoracodorsal nerve to the long thoracic nerve [63]. The lateral branch of the thoracodorsal nerve is both the correct length and number of axons to provide successful transfer to the long thoracic nerve [64]. This work has arisen from strategies aiming to address brachial plexus injury, with restoration of long thoracic nerve function considered to be essential for an

overall return of shoulder function [65]. The proposed advantage of nerve transfer over muscle transfer is the preservation of muscle biomechanics [63].

For the rare cases of scapular winging from traumatic avulsion of the serratus anterior (from the scapular or displaced inferior pole scapular fractures), early surgical repair is suggested with excellent outcomes [28,29]. This contrasts with the traditional teaching of the conservative management of these scapular fractures because fibres of the serratus anterior attached to the inferior boarder of the scapula exert the greatest pull [28]. Early EMG studies are useful in this scenario to distinguish these injuries from long thoracic nerve injury [29].

TRAPEZIUS PALSY

Anatomy

Trapezius palsy is less common than serratus anterior palsy but can be equally debilitating. The trapezius has a diffuse medial attachment from the skull to the twelfth thoracic vertebra and spreads laterally to the spine of the scapula, lateral third of the clavicle and the acromion [63]. Its superior portion elevates the scapula, the middle portion adducts and retracts, and the inferior portion depresses the scapula and rotates the inferior angle laterally [7,14].

The spinal accessory nerve (cranial nerve XI) supplies both the sternocleidomastoid and trapezius. The spinal and cranial portions join to form the accessory trunk and pass through the jugular foramen to emerge as the external branch. The nerve then runs alongside the internal jugular vein and crosses it laterally to pierce and innervate sternocleidomastoid. It then enters the posterior triangle of the neck, covered only by skin and subcutaneous fascia, to supply trapezius [5,30,42,66].

Clinical presentation

Scapular winging after trapezius palsy is lateral because the superior angle of the scapula displaces and the shoulder droops. It can be minimal at rest but increases with arm abduction and also with resisted external shoulder rotation. Normal scapulohumeral rhythm is disrupted and forward arm flexion and abduction are weak with limitation of all overhead activities [7]. It can present with shoulder fatigue, pain from muscle spasm and radiculitis from traction on the brachial plexus [67]. Misdiagnosis is common, with 14 of 22 patients initially being misdiagnosed in a series by Bigliani et al. [68]. Incorrect diagnoses vary widely and include herniated nucleus pulposus, shoulder impingement or instability, serratus and rhomboid paralysis, scoliosis, neuromuscular disease, fracture malunion, scapula osteochondroma, stroke and herpes zoster infection [43].

Aetiology

The aetiology of scapular winging after trapezius palsy is most commonly iatrogenic spinal accessory nerve damage from cervical lymph node biopsy or radical neck dissection [67,69]. The accessory nerve is vulnerable in the posterior triangle of the neck both to these iatrogenic insults or trauma, including road traffic accidents,

assault, deep tissue massage, blunt trauma and penetrating trauma [5,61,70].

Treatment

Some controversy exists over the role of conservative management in scapular winging from trapezius palsy because a significant proportion are the result of iatrogenic injury [67,69]. The best evidence for the natural history of the condition is provided by Friedenberg et al. who reported the 50-month follow-up for 56 spinal accessory nerve palsies over a 22-year period at the Mayo clinic, with generally good outcomes after conservative management. Identified predictors of poor outcome were the severity of clinical features at presentation, including an inability to raise the arm above the shoulder and dominant extremity involvement [31]. Others dispute this as a result of unsatisfactory outcomes in seven of eight patients managed conservatively by Bigliani et al. and suggestions that conservative management should be reserved for the elderly and sedentary [43,67].

If diagnosed early, spinal accessory neurolysis or nerve repair can be considered and Teboul et al. reported good or excellent results in 16 of 20 cases, concluding that the procedure should be performed within 20 months of injury [67,71,72]. If this early window for nerve surgery is missed, then the best evidence is for reconstruction with an Eden-Lange procedure [10,67,68,71,73–75]. The basic premise of this operation and its modifications comprises a reconstruction of the trapezius using a transfer of the rhomboids and levator scapulae. Bigliani et al. reported adequate pain relief and functional improvement in 19 out of 22 patients at 7-year follow-up [68]; Teboul et al. reported good or excellent results in four out of seven patients [75]; and Romero and Gerber demonstrated excellent pain relief and function in nine out of 12 patients at mean follow-up of 32 years [75]. Predictors of poor surgical outcome are age over 50 years and concomitant long thoracic nerve palsy [71,75].

RHOMBOID PALSY

Anatomy

Scapular winging from rhomboid paralysis is rarely reported. The rhomboids are shaped as the name suggests (oblique parallelograms) and lie deep to trapezius. Rhomboid minor is a thick muscle lying superior to the rhomboid major, which is thin and flat. They run inferolaterally from the vertebrae to the medial scapular boarder and function to retract and rotate the scapula [76].

The dorsal scapular nerve arises from the ventral ramus of C5 and occasionally C4 to supply the rhomboids. Its course pierces the middle scalene, close to the long thoracic nerve, and then runs deep to the levator scapulae before descending along the medial scapula margin to innervate the rhomboids [2,7,42].

Clinical presentation

Winging from rhomboid paralysis can present with pain on the medial boarder of the scapular, although the functional loss is minimal [2,7]. The inferior scapular angle protrudes but this winging can sometimes be abolished with arm elevation [42]. Clinical testing of the rhomboids is performed with the hands on the hips and the patient pushing the elbows backwards against

resistance [76]. With these subtle clinical features, this condition is probably underdiagnosed.

Aetiology

As with serratus anterior and trapezius palsy, the main cause of rhomboid dysfunction is presumed to be neurological, from dorsal scapular nerve damage. Reported causes include recurrent dislocation of the shoulder, volleyball and C5 radiculopathy, although these lesions are found in association with other nerve involvement including the suprascapular and long thoracic nerves [77,78]. An entrapment injury in the middle scalene is proposed as the most common site of injury [7].

Treatment

Because the scapular winging caused by dorsal scapular nerve injury is mild with minimal functional impairment, conservative treatment is considered to be the mainstay of management [2,7].

RARE CAUSES

An uncommon cause of apparent primary scapular winging is a large osteochondroma of the scapula. These bony prominences can push the scapula away from the chest wall and inhibit muscle function. [2,9]. Cervical flexion myelopathy has also been reported to cause scapula winging, in a single patient, involving triceps and serratus anterior weakness with no sensory disturbance [79]. A more diffuse loss of scapula stabilization is seen in both fascioscapulohumeral dystrophy and, occasionally, with diffuse brachial plexus trauma. Both of these can lead to painful winging where scapulothoracic fusion has been shown to be a successful first line surgery. Microneurosurgical nerve transfers are also gaining prominence in the management of scapula winging in association with brachial plexus injury [63–65].

Painful winging from diffuse traumatic damage to the scapula stabilizing muscles also responds well to scapulothoracic fusion. After fusion, arm abduction can be improved by up to 70 °C with good pain relief and improved shoulder endurance [59–61]. Sprengel's deformity is often quoted as a cause of primary scapula winging and is characterized by a congenitally small, undescended scapula. If painful, it responds well to scapulothoracic fusion [9].

Overall, scapulothoracic fusion appears to be a successful treatment for winging from diffuse scapula pathology, irreversibly affecting multiple stabilizing muscles. Excellent union rates are seen with secure fixation and bone grafting of the scapula to multiple ribs. This fixation can be achieved by circlage wire fixation from the scapula around multiple ribs, augmented by a plate or rush pin to prevent wire cut-out, or by direct plate and screw fixation at multiple rib levels [58,60].

SUMMARY

Scapula winging most commonly presents in association with isolated injury to the long thoracic nerve and resultant serratus anterior paralysis. The true aetiology of long thoracic nerve injury remains an enigma in many cases. For those with acute trauma and acute symptom onset, the traumatic aetiology is evident. For those

without acute trauma, there are multiple divergent theories of nerve compression or traction injury. The clinical picture for many of these patients closely resembles that of neuralgic amyotrophy and this may be the more likely explanation. The natural history is spontaneous resolution within 2 years for the majority and the mainstay of treatment is therefore conservative. EMG studies are a useful adjunct for distinguishing long thoracic nerve palsy from other causes of scapula winging, and can therefore inform the choice to manage patients expectantly.

Trapezius palsy has a more clearly defined aetiology of iatrogenic spinal accessory nerve damage in most cases and, as such, is more likely to respond to early intervention. Neurolysis or nerve repair has proven effective if performed within 20 months, although, after this time frame, surgical reconstruction with an Eden-Lange procedure is the most reliable treatment.

For the few with dorsal scapular nerve injury, leading to rhomboid paralysis and scapular winging, the commonest cause is probably a traction injury from trauma. The scapular winging is mild and the proposed treatment is conservative.

Palsy of serratus anterior, trapezius or the rhomboids form the vast majority of cases of scapula winging. Rare causes of diffuse paralysis or trauma to multiple scapula stabilizers, however, can present with debilitating pain and functional loss. These cases can be well suited to scapulothoracic fusion surgery with good published outcomes. For those with brachial plexus injury, microneurosurgical transfer of the thoracodorsal to the long thoracic nerve can restore scapula function.

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