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Research Article

Shoulder Impingement after Low Velocity Car Accidents - Is there a "Whiplash" for the Shoulder Joint?

E Pietsch*

Consultant Orthopaedic Surgeon and Trauma Specialist, Department of Trauma and Orthopaedics, The-Expert-Witness.de, Hamburg, Germany

***Corresponding Author**: E Pietsch, Consultant Orthopaedic Surgeon and Trauma Specialist, Department of Trauma and Orthopaedics, The-Expert-Witness.de, Hamburg, Germany.

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Abstract

Road traffic accidents with an acceleration/deceleration pattern to the neck can lead to associated pain in the shoulder. More specifically, patients can develop symptoms suggesting impingement. The mechanism is unclear. 64 patients with impingement symptoms after a whiplash injury in a medico legal setting were recruited for a prospective study to determine the findings after a rear-end collision. Magnetic resonance imaging (MRI), x-rays or Ultrasound could be obtained in 42 patients. A differentiated view excludes the idea that the common whiplash injury can result in structural damage of the shoulder joint. Our findings revealed pre-dating conditions that become relevant through an altered proprioception causing impingement symptoms.

Keywords: *Whiplash Injury; Shoulder Pain; Impingement; Road Traffic Accidents*

Introduction

Shoulder pain after "whiplash injuries" in rear-end collisions is common and can be long-standing despite physio treatment [1,2]. It is our understanding that symptoms can be attributed to referred pain from the trapezius or the neck. A minority of Clients, however, presents symptoms in their shoulder joint suggestive of an impingement. The cause for their symptoms is poorly understood, as with the given accident mechanism, injuries appear less likely to occur if "injury" is defined as a structural damage. Results from available studies were compared with our findings from subjects in a medico legal setting. We assumed that an altered proprioception can lead over an altered muscular balance to a functional impingement.

Methods

We included all individuals from our medico-legal Clinic that have been involved in an accident as occupant of a transport vehicle and for which a report had to be prepared. Over a period of 3 years, 811 Clients were examined who had presented to the author for a medico-legal report following a "whiplash" type of trauma to the neck. Whiplash was considered when the individual sustained an acceleration/deceleration movement to the neck and complained of symptoms secondary to the accident. All Clients were assessed by the author for the clinical evidence of pathologies to the shoulder joint. Individuals with neck or shoulder related symptoms prior to the accident were excluded.

A full examination of the neck and shoulder was carried out. This involved tests for Neer impingement sign [3], Hawkins-Kennedy impingement sign [4], the painful arc sign and muscle strength tests. Documentation included details of any available imaging before or after the medico-legal report. Further imaging following the medico-legal assessment was requested in order to confirm the diagnosis.

A review of the medical records took place in all cases. In particular, the findings from the physio notes were correlated with the Client's history. Their findings were used where symptoms to the shoulder joint were suspected and where symptoms had resolved themselves until the medico-legal examination.

Fulfilment criteria were a new onset of shoulder pain following an acceleration/deceleration trauma to the neck injury as well as having four positive clinical tests as described above.

Results

64/811 Clients (7,9%) presented with impingement signs following their accident. The overwhelming number of Clients was female (42/64). The average age at the time of the accident was 46 years (22 -77 years). In 67% (43/64) cases, the nearside shoulder was affected. In one case, bilateral symptoms occurred 28 days after the trauma. Only one of the Clients complained of a visible bruise as a result of a seatbelt trauma. None of the symptoms started insidiously after impact but developed gradually within hours, i.e. within one day after the accident. In 4/64 Clients, symptoms started 4 weeks after the accident.

In 70% of the Clients (45/64), shoulder symptoms coincided with neck pain and in 81% (52/64) with symptoms to the trapezius. 85% of the Clients (55/64) described their symptoms in their shoulder joint.

Within the follow-up of 13,7 months (3 to 48 months) after their accidents, i.e. the time of the examination for a medico legal report, 81% (52/64) complained of persisting symptoms. 86% had documented characteristic findings of an impingement syndrome (55/64), 9% symptoms of a subacromial bursitis or unspecific findings (9/64). 17% of the Clients (11/64) had a history of a shoulder condition with treatment that included a subacromial decompression (4/64), rotator cuff lesions (7/64), ACG degeneration (3/64), calcifying bursitis (4/64), clavicle fracture (1/64), shoulder dislocations (2/64) or a frozen shoulder (1/64).

6% Clients (4/64) had undergone x-rays as a result of the accident prior to their examination for their medico-legal report with unremarkable findings.

40% of the Clients (26/64) had job requirements that involved lifting or overhead tasks. 89% of the Clients (57/64) underwent physiotherapy with a minimum of 5,6 sessions. Treatment was regarded as helpful in only 9% of the cases (6/64). Clients described treatment for their necks with deep tissue massages, acupuncture, mobilisation, heat, TENS and advice for stretching exercises at home but no specific treatment for their shoulder condition None received specific instructions for strengthening exercises for their shoulder girdle.

In 25% of the Clients (16/64), symptoms resolved within a maximum of 10 months (0,25 to 10 months). This applied mainly to the manual labourers whose jobs involved heavy lifting, e.g. builders or dock workers (10/16).

In 72% Clients (46/64), further investigations were requested after the preparation of a medico legal report. The most common findings with 30% were focal intrasubstance tears involving the supraspinatus tendon (14/46). 4 of them had an isolated tear (1/4 full thickness and 3/4 partial tears). More common were supraspinatus tears with 35% (16/46) in association with further pathologies (4/16 ACG degeneration, 6/16 anatomical impingement, 1/16 bursitis, 6/16 tendinosis, 2/16 infraspinatus tendon tears, and 1/16 calcifying tendinosis). Among those with isolated findings were 7/46 with criteria for an isolated impingement of the cuff tendons, 11/46 with a subacromial bursitis, 5/46 with a tendinosis of the cuff tendons, 3 with evidence for a calcifying bursitis.

17% of the subjects had no further imaging as their symptoms had settled at the time of the examination (11/64).

At the time of the impact, 98% of the Clients had their hands on the steering wheel in a relaxed, i.e. bent position (63/64). Only one Client reported that his arm was twisted by the steering wheel.

Mean time to the final diagnosis was 23 months.

Discussion

The use of the term “whiplash injury” is used for the constellation of symptoms as a result of a road traffic accident involving the spine. The mechanism is understood as an acceleration-deceleration, which is associated with the onset of a plethora of symptoms, e.g. neck pain and stiffness, occipital headache, thoracolumbar back pain, paraesthesia in the upper limbs [5-8] or shoulder pain. The latter one is regarded as being attributable to referred pain from the neck but can also occur vice versa [9].

A small number of car occupants can experience symptoms in their shoulder joint. They present with clinical features of an impingement syndrome or even pain in their AC joint. Following the literature, there appears to be no consensus regarding the mechanism of the injury or its related symptoms.

Abbassian (2008) favoured the idea of an impact from the seatbelt as injury mechanism. They found an incidence of 5% of Clients with impingement symptoms in a background population of medico legal claimants. Symptomatic Clients were on average older than the asymptomatic Clients (57.5 years versus 36.9 years). In 83%, the seat belted shoulder was affected, but even in the remaining 17% of the population, signs of bruising were noticeable over the symptomatic shoulder or the upper limb. It was found that symptoms occurred within one week after trauma. The diagnosis could be confirmed in 5/11 cases only clinically and, unfortunately, there was no further reference to the underlying pathology. The authors suggested that in the overwhelming cases, a direct impact to the shoulder had occurred and that the most likely cause was the seatbelt trauma. It was also considered that the higher age can suggest pre-existing degenerative changes exposing the Clients to a higher risk of developing impingement.

Chauhan [10] found 9% of their Clients to be symptomatic of impingement signs. Most of them showed an alteration in their scapulothoracic rhythm. With the trapezius as a main stabilizer of the scapula, it was argued that an involvement of the trapezius can lead to impingement. They administered a steroid injection followed by specific physiotherapy aiming at correcting scapulothoracic and scapulohumeral rhythm showing a good response afterwards. However, Clients with an immediate onset of pain were excluded in order to exclude symptoms as a result of a direct seatbelt injury.

Our Clients revealed a heterogeneous distribution of findings. None of them reported findings that would have suggested a relevant impact from the seatbelt. None of them developed a seatbelt mark. It was also our observation that even in subjects with a clearly visible seatbelt mark, no impingement signs would develop. Besides, the onset of symptoms was in a timely close relationship of one day after the trauma and preferred the restrained shoulder joint, the older Client (46 years (25 - 77)) and the female gender (42/64).

The relevance of the trauma as a result of the accident is usually reflected in the severity of the physical damage. Our investigations in the symptomatic Clients revealed conditions that appeared degenerative in nature. No Client could be identified with findings that would have suggested a trauma-related pathology.

As our "older" Clients appeared to be preferred in developing symptoms, MRI and Ultrasound findings must be critically weighed against the natural process of degeneration in a comparable background population without trauma.

The specialist needs to take a few aspects into consideration when it comes to commenting on imaging. With age, there is a rising incidence of shoulder pain. Between 7% and 34% of adults have shoulder pain at times [11]. The incidence is estimated to be 19 per 1000 person-years-highest in women over 45 years and lower in young adults [12]. In certain sports or occupations, point prevalence can reach 40% or higher [13-15]. There is also an increase of degenerative findings that affect tendons, AC and glenohumeral joint alike, which mostly remain asymptomatic. Osteoarthritis, the most common cause of shoulder pain originating from the AC joint, is a frequent finding in patients older than 50 years of age [16]. Horvath [17] showed that 54 to 57% of elderly patients have radiographic evidence of degenerative arthritis of the AC joint. Evaluation of MRIs among asymptomatic subjects demonstrated the prevalence of AC joint osteoarthritis to be between 48% and 82% [18,19].

As such, the medical expert needs to differ between trauma-related changes and predating degenerative findings. The tools that assist in decision making consist of the accident mechanism, the Client's history of symptoms and imaging.

Firstly, tendon tears follow a distinct injury mechanism [20]. They result from a shoulder dislocation or a tensile force (fall onto the adducted or retroverted arm, internal rotation or abduction over 60° against resistance or the hyperextension) but not after a direct impact [21]. In the event of a rear-end or side collision with the arm in a "relaxed" position, the requirements for a tendon tear are usually not given.

Secondly, the presentation of symptoms in an acute traumatic tear leads to insidious symptoms and a loss of function. In the "younger" patient (< 40 years), it occurs without predating history of symptoms or signs of degenerative changes on XR or MRI.

Thirdly, MRI or Ultrasound can reveal a tendon tear. The location and the type of tear allow conclusions in relation to its cause. Biomechanically, the bursa-side layer has greater deformation and tensile strength than the joint-side layer. This makes the joint-side layer more vulnerable to a tensile load than the bursa-side layer [22]. A degenerative tear is usually a longitudinal tear and, with increasing size, also transverse. Signs of a predating degenerative condition can be sclerosis of the greater tuberosity, AC joint degeneration, fatty muscular degeneration, subacromial osteophytes, acromion type II and III, and increasing symptoms. The complexity of the rotator cuff makes isolated or focal tears more than unlikely. Hempfling [23] described in an anatomical review that a cuff tendon tears in combination with functionally associated tissue structures and has to occur as a detachment of its tendon footprint usually in combination with an injury to the deltoid. Punctional tears, l- shaped or semilunar tears within the tendon, in contrast, are usually degenerative in nature.

Finally, in the surrounding of an anatomical "impingement", isolated tendon tears are common and rarely due to the alleged trauma. Impingement is defined as compression, entrapment, or mechanical irritation of the rotator cuff structures and/or long head of the biceps tendon either beneath the coracoacromial arch (subacromial) or between the undersurface of the rotator cuff and the glenoid or glenoid labrum (internal). This is either due to anatomic changes in the coracoacromial arch or changes in the biomechanics. Predisposing factors can be degenerative spurs underneath the AC joint, a hooked acromion or a narrow joint space. In this setting, repetitive impingement is one of multiple proposed mechanisms for the development of rotator cuff disease, as well as progression to a partial or full-thickness rotator cuff tear [24]. A working history with the Client's job requirements can be helpful to differentiate further.

In our group, 50% showed smaller tears of the cuff tendons (32/64), usually the supraspinatus tendon, with one Client with a complete tear of the subscapularis. The relevance of these findings is questionable. The latter one recovered within 2 months after trauma, 8 Clients with a supraspinatus tendon tear on MRI or Ultrasound within 1 to 3 months. This supports the idea that Clients can have predating but asymptomatic tears with coincidental findings on imaging. Also, the findings did not have an impact on workers with overhead tasks. Their symptoms resolved quicker despite their underlying pathology than Clients without a demanding job.

Muddu., *et al*. [25] suggested the idea of a "whiplash injury to the shoulder". It should be understood as a separate entity rather than, for example, an impingement syndrome. In their series, 15 out of 18 patients had no significant shoulder pathology on MRI. Only 2/18 patients (11%) demonstrated rotator cuff tears and evidence of subacromial impingement. It is not clear, however, if their patients had positive clinical signs for subacromial impingement (despite their negative MRI) or if they were merely complaining of generalised shoulder pain following their neck injury.

Accepting that findings on imaging can be regarded as predating and that the mechanism of a rear shunt or side collision is unlikely to cause a structural damage, the question remains what mechanisms are likely to produce the Clients' symptoms.

A few authors argued that impingement symptoms are due to an impaired glenohumeral and scapulothoracic rhythm [26,27]. Chauhan (2003) found an altered trapezius function as symptoms responded well to specific physio and local injections. It was also accepted that

impingement can also follow after an injury of the supraspinatus nerve. However, this would require an injury at spinal level C5/C6 affecting the supra- and infraspinatus muscle whereas the trapezius is innervated form the accessory nerve from C2 to C4.

Following the findings of an altered trapezius function, it appears more plausible that the altered muscular pattern can occur as a result of a disturbed sensorimotor system, which results in an instability of the shoulder joint.

The sensorimotor system is defined as all of the sensory, motor, and central integration and processing components involved in maintaining joint stability. It includes several components, i.e. proprioception, joint position sense, kinaesthesia, sensation of force, and neuromuscular control. Of significance are sensory and mechanoreceptors that can be found beneath the synovial membrane and close to the humeral site of insertion of the ligaments, coracoacromial ligament, the musculotendinous junctions of the rotator cuff and in the capsule. Receptors are close to the labrum and the glenoid [28]. They are regarded as controlling stability of the shoulder musculature [29].

Mechanoreceptive and nociceptive afferents have their peripheral terminals in the joint capsule, cell bodies in the dorsal root ganglia, and terminal processes in the spinal cord. As such, biomechanical loading of these afferents can initiate nociceptive signalling in the peripheral and central nervous systems [30].

A joint injury with disruption or detachment of these ligaments would be regarded as suitable not only to destroy the mechanical restraints of the joint capsule, glenoid, labrum, etc., but also, the sensorimotor system resulting in a disrupted feedback mechanism. The result is an unstable shoulder with poor muscular control due to missing stiffness [31]. Myers [32] also found forces significant enough to cause disruption and the receptors to respond to the injury usually after shoulder surgery, shoulder dislocations or subluxation.

However, in low-speed shunts, these reflections would not fully apply. The forces would not be significant enough to disrupt the sensorimotor system. But they may be significant enough to cause stimulation of sensory receptors [33] that, in return, can modify the muscle tone around the shoulder joint. Similar considerations have been given to the cause for the whiplash associated disorders and a stimulation of the proprioceptors of the facet joint capsule [34-36].

It would therefore be reasonable to consider impingement symptoms of the shoulder as an own entity, which follows an acceleration/deceleration trauma and creates its own "whiplash" to the shoulder joint. Pre-dating conditions, e.g. tendon tears, become symptomatic and clinically relevant as a result of an altered proprioception with a disturbed scapulothoracic rhythm. This would explain why symptoms can occur without signs of direct trauma, bilaterally or in the far side shoulder. Besides, it would also be in keeping with the fact that symptoms in manual labourers are short-termed as a muscular dysbalance is unlikely to develop or persist. The poor response to physiotherapy indicates that treatment was not specific enough to restore the scapulothoracic rhythm and would support Chauhan (2003) that specific physio can be supportive to restore muscular balance. Patients should therefore be carefully assessed and selected for their appropriate treatment.

Conclusion

Symptoms of a shoulder impingement after whiplash injuries of the neck are less frequent than referred pain from the neck into the shoulder. Findings suggest that predating conditions can become clinically apparent due to an altered scapulothoracic rhythm as a result of an altered proprioception. It appears as if the acceleration/deceleration of the shoulder joint creates its own "whiplash", which should respond well to more specific physio treatment.

Limitations

This study has limitations. It includes patients in medico-legal proceedings, which may not truly reflect the general population. Subacromial injections of local anaesthetic would have been useful in the further assessment of the cohort.

Conflict of Interests

The author declares that there is no conflict of interest.

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