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Stinger Lesion: An Atypical Lesion of a Professional Rugby Player, Overview of a Poorly Known Pathology

Delvaque JG[1], Rubio E[2] and Masmejean EH[1]*

[1]*Hand, Upper Limb and Peripheral Nerve Surgery Service, George Pompidou European Hospital, France*

[2]*Sports Health Medical Institute (IMSS), France*

Abstract

The traumatic plexopathy of the brachial plexus, called Stinger lesion, is little known to the orthopedic surgeon, yet it is a very frequent and common injury in high-level contact athletes. Its quick identification in its typical presentation, the elimination of the most severe alternative diagnosis and its management must be known in order to be adapted to the athlete's sporting objectives. Atypical forms with slow recovery are rare and represent a real diagnostic challenge. We report the case of a professional rugby player who presented an atypical Stinger injury and detail its management. This original case is an opportunity to provide a comprehensive review of the different forms and presentations of these traumatic plexopathies and their management.

Keywords: Plexus brachial; Sport trauma; Stinger; COVID-19

Introduction

Two main types of brachial plexus injuries are traditionally described: Obstetric brachial plexus paralysis and adult stretch injuries. Both are serious and require specialized care. A third case exists: The traumatic plexopathy of the brachial plexus, better known as Stinger injury or Burner injury. It is little known to the orthopedic surgeon, yet it is a very frequent and common injury in high-level contact athletes. Owen Farrell, England rugby halfback, said when he refused to come out on suspicion of a knockout during a match at the 2021 Six Nations Tournament: "It's just a stinger; it has nothing to do with me."

The classic injury mechanism is a direct impact on the shoulder involving the upper limb on one side and the head on the opposite side. This corresponds to the classic move made by a tackler in rugby. This result in compression and stretching of the brachial plexus, which in some cases can lead to a transient plexopathy of the brachial plexus called a stinger injury [1]. Stinger in French "dard" refers to the symptomatology of the injury. The Stinger injury is described as an electric discharge running through the upper limb. The symptoms are generally resolved in 1 min to 2 min. They are characterized by an immediate sensation of burns and electric discharges going through the entire upper limb associated with an important weakness, even with a total paralysis of the upper limb. In some cases, weakness of the upper limb persists over time, especially in the muscles innervated by the proximal roots (C5 and C6); these symptoms may persist for several days or weeks [2].

The 2020/2021 COVID-19 epidemics highlight a certain neurotropism of this type of coronavirus. This tropism can be manifested by mononeuropathy or multifocal neuropathy [3]. Beside the known attacks of the central nervous system and their symptoms such as anosmia or ageusia, a tropism of this virus for the nervous elements composing the brachial plexus has been highlighted [4,5].

Case Presentation

We present the case of a professional rugby player with a long recovery traumatic plexopathy in an athlete who was infected with the COVID-19 virus.

We then propose, based on the literature data, an update on the Stinger lesion in high-level athletes.

We present the case of a professional rugby player of the “Stade Français” evolving as second line in the French professional league. On November 29th, 2020 in a match following a tackle on the right shoulder (Figure 1 and Video 1), he presented a Stinger injury characterized by a transient paralysis

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Correspondence:
Jean-Gabriel Delvaque, Hand, Upper Limb and Peripheral Nerve Surgery Service, George Pompidou European Hospital, 20 rue Lebanc 75015, Paris, France, Tel: 0664895554; E-mail: delvaque.jg@gmail.com

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Figure 1: The Tackle. Top 14 rugby match, "Stade Français" (Pink) *vs* "La Rochelle" (Black) 15/02/2020.

Video 1: On November 29, 2020 in a match following a tackle on the right shoulder.

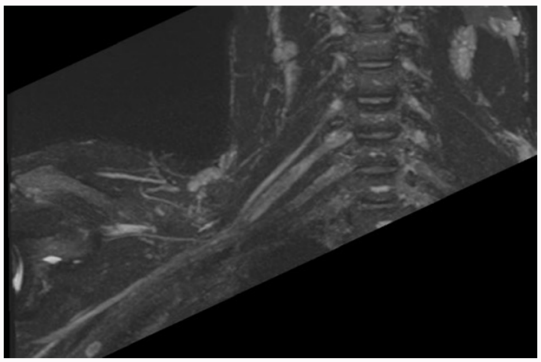

Figure 2a: Plexus brachial MRI, Right C5C6 roots injury. C5C6 enlargement and hypersignal *vs.* controlateral. A) Neurographic 3D SPACE STIR, MIP reconstruction.

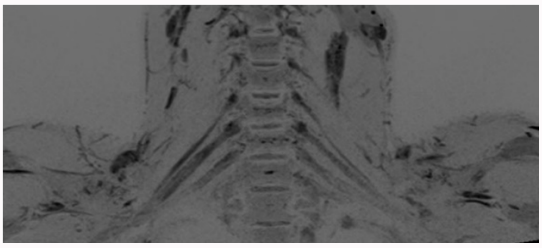

Figure 2b: Plexus brachial MRI, Right C5C6 roots injury. C5C6 enlargement and hypersignal *vs.* controlateral. b). Neurographic 3D SPACE STIR, MIP reconstruction (Black/White inversion).

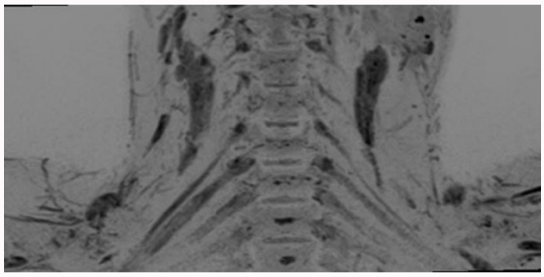

Figure 2c: Plexus brachial MRI, Right C5C6 roots injury. C5C6 enlargement and hypersignal *vs.* controlateral. c) Neurographic 3D SPACE STIR, MIP reconstruction (Black/White inversion).

of the entire right upper limb associated with significant paresthesias. Within minutes of the accident, an almost complete regression of symptoms was noticed. Unlike the usual evolution of the Stinger injury, the patient presented persistent discomfort during training and weakness of the right upper limb motivating a consultation 3 months after the trauma. During our clinical examination, we observed: Hypoesthesia of the shoulder stump, pain and muscle weakness major on the external rotators of the right shoulder, we also noted an irritative syndrome (pseudo-Tinel sign of the supra-clavicular region), the overall being compatible with incomplete C5, C6 involvement.

Standard X-ray of the cervical spine, dynamic bilateral positional Doppler ultrasound, electroneuromyogram of both upper limbs and MRI of the right brachial plexus were performed in order to document the lesions and to eliminate a differential diagnosis (Parsonage Turner syndrome and thoracic-brachial outlet syndrome). The paraclinical evaluation was normal except for the brachial plexus MRI showing diffuse hyperfixation of the right brachial plexus (Figure 2). The diagnosis slow recovery Stinger lesion is therefore retained. The treatment consisted of rehabilitation in order to open the cervico-thoraco-brachial clamp associated with the prescription of low-dose pregabalin to treat the irritative syndrome. At 6 months after the trauma, the patient was seen in consultation. He presented a total regression of symptoms with, in particular, a strength in external rotation at 5/5 (symmetrical isometric test) as well as a normal sensitivity of the shoulder stump. However, there is still a mild supraclavicular irritation syndrome. The return to competitive sport was performed at 6 months with a "pad" on the right shoulder as protective gear.

Discussion

Focus on the Stinger injury in high-level athletes

Kawasaki et al. [6] have shown that 33.9% of professional rugby players experience at least one Stinger injury in a season, with a recurrence rate of 37.0% [6]. Nevertheless, as these lesions are well known and commonplace among contact athletes, their incidence is probably underestimated [7].

The mechanism involved in most cases combines downward traction of the upper limb and forced lateral flexion of the head on the side opposite the injury. This mechanism leads to a stretching of the brachial plexus associated in certain cases with a foraminal compression of the nerve roots of the brachial plexus. These traumatic stretch injuries of the brachial plexus are classified according to the Seddon Sunderland classification [8]. They are in the majority of cases type 1 (Neurapraxia) and rarely of type 2 (Axonotmesis). Type 3 lesions (Neurotmesis) are not part of the Stinger lesions and belongs to the well-known management of tear out lesions of the brachial plexus [9].

Grade 1 lesions lead to a demyelination process without axonal

damage. In grade 2 lesions, the axons and myelin sheath are affected leading to a process of Wallerian degeneration, but the Schwann cells as well as the perinerve and epinerve remain intact. As previously mentioned, the Stinger injury corresponds to an immediate sensation of burning and electrical discharges running through the entire upper limb associated with significant weakness or even total paralysis of the upper limb. In some cases, weakness persists in the upper limb, more marked in the muscles innervated by the proximal roots (C5 and C6), but it can also affect any of the muscles of the upper limb; these symptoms can persist for several weeks or even months [10].

Clinical examination and management

The context, the description of the symptoms, the spontaneous resolving character in less than two minutes and the experience of the lesions by the athlete easily allows to make the diagnosis of Stinger injury. In case of atypical symptoms (bilateral damage, damage to the lower limbs) or in case of doubt, it is needed to consider the trauma as a cervical spine damage (immediate immobilization with a cervical collar and exit on a stretcher) [9]. The acute clinical examination should initially focus on eliminating differential diagnoses, such as cervical spine trauma or a shoulder girdle injury. Secondly, a complete motor and sensory examination of the upper limb concerned must be carried out, paying particular attention to the muscles innervated by the C6 and C7 roots, which are the most frequently affected.

As a matter of course, even in the typical injury cases, a complete clinical examination must be repeated after the game and regularly during the first two weeks. Indeed, a normal clinical examination in the acute phase does not prevent the occurrence of symptoms in the hours or days following the trauma [1]. In case of typical lesions, there is no indication to carry out further investigations. On the other hand, if the symptoms persist, the clinical examination must be repeated. It will try to asses' pain in the cervical spine, a sensory or motor deficit in the upper limb, the presence of a supra or infra clavicular pseudo Tinel's sign, an asymmetry of the radial pulse or a muscular atrophy especially in the shoulder (Deltoid, supra and infraspinous fossa) in order to eliminate a differential diagnosis.

After careful palpation of the cervical spine, a Spruling test is performed. If there is associated neck pain, the diagnosis can be guide to a cervical radicular damage [11]. The Spruling test consists of applying compression, rotation and homo-lateral tilt to the lesion. The maneuver is positive when it triggers radicular symptoms in the upper limb. Patients with Stinger injury have no neck pain and a negative Spruling test [9].

The value of complementary investigation in Stinger injury is controversial. In cases of typical lesions with quick resolving symptoms, no further investigation is indicated. On the other hand, in case of persistent or recurrent symptoms or in case of doubt, imaging and electro-neuro-myograms can help to document the lesion and eliminate differential diagnoses.

Standard and dynamic cervical X-rays, as well as cervical MRI, are used to identify a spine facture, as well as cervical roots damage. Literature data seem to highlight anatomical predispositions of the cervical spine to the occurrence of Stinger injuries [12]. Indeed, a higher incidence of foraminal or cervical canal stenosis has been found in athletes with repetitive Stinger injuries [11].

In case of persistent symptoms, we perform a dynamic arterial and venous positional Doppler ultrasound in order to rule out a cervicothoracic outlet syndrome.

The ENMG performed after 6 weeks in case of "long stinger" allows on the one hand to eliminate a pre-ganglionic damage of the brachial plexus and on the other hand to specify, if any, the level of the damage [13].

Brachial plexus MRI is also only performed in cases of "long Stinger" and if all other investigations are normal. Hypersignal of the involved roots is currently found in stretch lesions of the brachial plexus [14].

Management and return to sport

The management of these injuries is mainly based on physical rest, pain management and rehabilitation.

Feinberg et al. [15] propose an algorithm of management and return to sport according to the type of injury following the Sunderland classification [15].

Grade 1 lesions (Neurapraxia) are divided into three categories related to the severity of the symptoms. The lesion known as "mild grade 1" is defined by an immediate regression of the symptoms. In this context the sports activity can be continued immediately. In the case of "moderate grade 1" injuries, the symptoms persist during the sporting activity and end after the match, while in the case of "severe grade 1" injuries, the symptoms persist for over 24 h. In moderate or severe injury, the sport activity must be stopped, the clinical exam must be repeated regularly and the paraclinicals investigations in order to eliminate a trauma of the cervical rachis must be carried out. The return to sport can be considered as soon as the symptoms disappear.

For Sunderland's grade 2 lesions, sports activity must be stopped immediately for 15 days minimum. The clinical exam must be repeated.

Paraclinical investigation are performed for cervical spine trauma, an EMG should be performed if symptoms persist for more than 15 days. The return to sport can be considered as soon as the symptoms regress and paraclinicals investigations are normal.

In order to reduce the incidence of spine and nerve injuries, sports federations have progressively changed the rules of high-risk sports by being more and more restrictive and by sanctioning risky behaviors more severely. For example, at rugby, head-on tackles, high tackles and tackles above the waist are now prohibited in France and in most international competitions.

Regarding equipment, although their benefits are not scientifically validated, wearing "pads" on the shoulders in order to absorb shocks and the use of medical devices limiting hyperextension such as the "Cowboy-Collar" for American soccer players are becoming more and more common [16].

Conclusion

The damage to the nervous system due to the neurotropism of COVID-19, although still unknown, seems to be more and more described and documented, the most common disorders are anosmia and agueusia. In addition to these, mononeuropathy or multifocal peripheral neuropathy have been described. Plexus lesions in patients with severe covid-19 infection were initially analyzed as "resuscitation neuropathies" and consequences of prone sessions. The existence of a few cases of the upper limb deficits in patients who have not had any prone sessions has suggested the existence of a COVID-19 tropism for the nervous elements of the brachial plexus.

In our case the athlete had a positive COVID-19 PCR test on 9/11/2020, during a systematic screening of an official competition. He had no symptoms and was able to return to training and competition as soon as the regulatory isolation was over.

The occurrence of a stinger lesion in a professional athlete who played rugby intensively and had never presented this symptomatology, brought us to search an additional subjacent lesion. The MRI showed diffuse damage to the roots of the brachial plexus.

We are unable to state that the lesion developed by the player is related to his contamination by COVID-19. But this leads us to wonder: "Did a neurotropism of the COVID-19 virus in the brachial plexus facilitate the occurrence of a Stinger lesion and slow down his recuperation?

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