An isolated double-crush-syndrome in posttraumatic thoracic outlet syndrome - A case report

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A B S T R A C T
Thoracic outlet Syndrome (TOS) includes all disorders caused by compression of all neurovascular Structures in the upper thoracic outlet (Ross and Owners, 1966, Bürger and Arterien, 2014, Sanders and Annest, 2017).

The Double-Crush-Syndrome (DBS) defines multilevel lesions along a neurovascular trunk caused by mechanical compression in different areas.
Pectoralis-minor-syndrome (PMS) is also classified in the disorders of the upper thoracic outlet and was also known as hyperabduction syndrome or subcoracidal syndrome.

Between 2015–2019 our department treated 488 patients suffering from neurological, vascular or combined TOS. Surgical treatment, depending on clinical and specific diagnostics was performed in 175 cases via transaxillary approach, including cervical rib resection, first rib resection, neurolysis of plexus brachialis, thoracic sympathectomy or vascular reconstruction. In all this year just a single patient with double crush syndrome was present.

CASE PRESENTATION AND METHODS: We report a case of a 28-years old female patient, reported in line with the SCARE criteria [13], suffering from neurovascular compression in the upper thoracic outlet after surgically treated clavicle fracture.

She developed typical symptoms of a Thoracic Outlet Syndrome.

CONCLUSION: Double-Crush-Syndrome in patients with Thoracic Outlet Syndrome are very rare, case reports seldomly exist. The diagnosis requires a specific clinical testing and x-ray radiography. Furthermore dynamic tests like ultrasound and angiography and neurophysiological testing requires a high degree of experience, so the compressed area can be detected.

Treatment includes an attempt of best medical and physical therapy, in case of failure a surgical treatment is necessary.

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1. Background

The symptomatic compression syndromes of the neurovascular bundle in the area of the upper thoracic aperture are summarized under the term “Thoracic Outlet Syndrome” (TOS) and are divided according to the compression mechanisms into neurological, arterial, venous or combined symptoms, depending on the location and structure being compressed.

PEET et al. was the first who used the collective term “thoracic outlet syndrome” for the compression of the neurovascular bundle in the area of the upper thoracic aperture in 1956.

This defines TOS as a one- or double-sided neurovascular compression, which is assigned to the corresponding symptoms as a result of compression of the brachial plexus, the subclavian artery and the subclavian vein [11].

The diverse clinical problems, missing diagnostic protocols and the non-standardized treatment lead to a controversial discussion regarding the optimal therapy.

2. Case presentation

2.1. First treatment

In 2018, a 28 years old female referred to our department after left sided clavicle fracture in 2016.

She showed typical symptoms of neurovascular compression with pain, weakness, paresthesia and loss of senso-motoric
agility, that occurred two years after a dislocated clavicle fracture and osteosynthetic restoration with already performed metal removal.

The fracture, which was fixed immediately after trauma by wire osteosynthesis, had been finally treated in the course of a plate osteosynthesis. Shortly after the treatment, stress-related complaints, pain in the left arm and hand, paraesthesia in overhead position began. Conventional X-rays showed that the osteosynthesis material was in the correct position. But later in the CT scan the suspicion of irritation of the brachial plexus by a implantet screw was expressed, so after a frustrating conservative therapy attempt, metal removal with satisfactory bone healing was performed.

The first consultation in our department took place in September 2018. We immediately recognized indicators for the presence of a compression syndrome in the upper thoracic outlet with prompt pulse suppression in abduction and a positive abduction-elevation-rotation test (AER).

The right side was free from any symptoms. The duplex ultrasound showed a compression of the left subclavian artery in the abduction and elevation positions.

We performed specific invasive diagnostic, e.g. by centralvenous angiography in in seated patient with different, particularly natural and triggering positions which confirmed the compression of the left subclavian artery in the abduction, external rotation and elevation position (Fig. 1) [12,4,2].

There was no evidence of damage to the brachial plexus in the context of our neurophysiological examinations. No cervical rib in the x-ray controls.

Based on our available diagnostics and results, we decided to perform surgical decompression via transaxillary approach with complete first rib resection and neurolysis in January 2019. The postoperative course run without any complication and the patient left our service with NSAR 7 days after surgery.

The follow-up, about 6 weeks after treatment, the patient reported persistent stress-related complaints in the left arm and hand. Even simple and daily actions could hardly be carried out. The professional situation very limited again and the quality of life has not improved.

About a year after first rib resection and despite accompanying best medical treatment therapy, consistent physiotherapy treatment, physical therapy and redone hospitalised pain therapy, no improvement occurred.

2.2. Second presentation

At the beginning of the second presentation at our department, a detailed medical history was carried out, in particular regarding the previous therapeutic approaches and medical consultations (Fig. 2).

In clinical examination we still provoked a pulse suppression on the wrist in hyper-abduction on the left. A paling of the left hand has also been demonstrated in the AER test with punching exercises. Arm fatigue promptly occurred in a few seconds.

The oscillography and ultrasound examination confirmed the suspicion and showed the image of a reduced perfusion in hyper-abduction. In normal position there were physiological curves without any evidence of ischemia (Figs. 4 and 5).
A central-venous angiography revealed a regular course of the left subclavian artery in all provocative positions, so that a selective transfemoral, intra-arterial angiography was performed for the detailed display of all left upper extremity vessels without wall morphological damage, but mild compression at proximal axillary artery without evidence of embolization (Figs. 3 and 4) [1,2,10].

Finally the targeted duplex ultrasound of the arm vessel performed in the provocation position showed a reproducible compression of the proximal axillary artery on the left by the pectoralis minor muscle in the abduction position (Figs. 4 and 5) [3].

The Pectoralis Minor Syndrome:

The pectoralis minor muscle has its origin on the 3rd to 5th rib. It pulls up to the processus coracoideus of the scapula. It causes the scapula to turn inwards and serves as an auxiliary breathing muscle when the upper arm scapula are fixed.

The pectoralis minor syndrome, however, rarely causing permanent damage, depending on anatomy, abduction and retroversion lead to compression and possible functional damage to the neurovascular bundle distal to the upper thoracic aperture.

The clinical symptoms occur similar to those of Thoracic Outlet Syndrome and must therefore be taken into treatment options.

Typically, there are recurring pain events, especially on the chest wall, in the subscapular area, in the axilla and on the shoulder, which can be accompanied by paraesthesia of the arm and hand. The ulnar fingers are usually also affected. The stress-related weakness occurs when the axillary artery is compressed in certain positions and the perfusion is restricted [3,5].

Causes are often secondary changes due to trauma (traffic accidents, falls) with osseous lesions of the clavicle or processus coracoideus of the scapula.

Repetitive, extreme stress-loads on the pectoralis minor, e.g. due to abnormal malposition, occupational stress (e.g. permanent assembly line work in the same position) and extreme muscle
building are typical causes for the development of a Pectoralis Minor syndrome. The therapy of the Pectoralis Minor Syndrome includes, in mild courses, conservative measures with targeted movement exercises to relieve the strain, but also defined stretching maneuvers to expand the elasticity of the pectoralis minor muscle under professional supervision. Progressive complaints should be treated surgically by myotomy and myectomy of the pectoralis minor muscle via transaxillary or axillary access (Fig. 5) [3,6,10].

3. Conclusion

For final decompression we performed a complete myotomy and partial myectomy of the pectoralis minor muscle using an axillary access on the left [5–9]. The clinical symptoms ceased, and exposure was possible again without restrictions in the further course.

4. Discussion

The compression syndromes of the upper thoracic aperture are rare, the symptoms varied, the causes different. Patients often suffer from the long and grueling way of securing the diagnosis, occasionally accompanied by frustrating surgical attempts at therapy.

In suspicious cases of a compression syndrome in the highly complex anatomy of the shoulder and upper thoracic outlet, it must be ensured that professional and adequate treatment is carried out. The time-consuming and special diagnostics assurance and the possibly necessary, technically complex surgical treatment should be reserved for special, experienced centers to ensure best results.

Declarations of Competing Interest

All authors declare that they have no competing interests.

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Ethical approval

The need for approval was waived.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution

Idea and findings initially by the author, during writing this paper HO, TB were involved constantly in discussion und improvements of the manuscript. Surgeries were performed by TB.

Registration of research studies

1. Name of the registry: An isolated Double-Crush-Syndrome in posttraumatic Thoracic Outlet Syndrome - a case report.
2. Unique identifying number or registration ID: researchregistry5903.
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): https://www.researchregistry.com/register-now#home/registrationdetails/5f3519810a19700016d9e364/.

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