Bilateral leg symptoms – The T10 syndrome?

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**Abstract**

Prior studies have investigated the role of the sympathetic nervous system and the thoracic spine related to the upper extremities (known as the T4 syndrome). However, there is only little known about the role of the thoracic spine related to the lower extremities. In this case report, a patient with a heavy, tired feeling in both legs and hypomobile thoracic segments was treated with passive mobilisations of the thoracic spine.

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**1. Introduction**

Patients with bilateral leg symptoms are commonly seen by a manual therapist in clinical practice. These symptoms can vary from pain, a heavy feeling, pins and needles and signs of loss of neurological conduction. The possibility of involvement of the sympathetic nervous system has already been described for the upper extremities. This phenomenon is called “the T4 syndrome” (Grieve, 1988; Maitland et al., 2005). However, there is little known about possible mechanisms involved in bilateral leg symptoms and its relationship to the thoracic spine (Grieve, 1988; Bogduk, 2002; Perry and Green, 2008). The effectiveness of treatment with passive mobilisation techniques in restoring sympathetic function was already proven in several studies (Wright, 1995; Perry and Green, 2008; Jowsey and Perry, 2010).

This case report describes a patient with symptoms that gave suspicion of a sympathetic outflow dysfunction in the lower extremities with co-existing hypomobile thoracic segments. The clinical presentation, treatment and response will be presented in this case report.

**2. Subjective examination**

A 52-year old female was referred to the physiotherapy practice by the general practitioner (GP) with bilateral leg symptoms (area 1, Fig. 1) and low back pain (LBP, area 2, Fig. 1). The patient worked as a secretary at the hospital. She exercised on the powerplate and crosstrainer three times a week for 1 h. The symptoms were localised in both legs and consisted of a heavy, tired deep feeling, which was intermittent with the right leg being worse. The central LBP was nagging and intermittent.

On waking up in the morning her legs felt stiff. During the day they got more tired. At the end of the day both legs felt heavy. She had difficulty sleeping and needed to get out of bed sometimes to walk around and ease her symptoms, which could take up to 30 min. Activities that provoked her symptoms were sitting for more than 60 min, getting up from a chair or bed and starting to walk, switching sides in bed, bending forward and doing sports. When the symptoms got worse she was able to reduce them by shaking the legs or walking.

The symptoms started without a clear cause somewhere between six and twelve months previously. Later she noticed that this corresponded to the period when her office desk was changed. As a result, she needed to bend over and rotate more to the right. The symptoms began with a tired feeling in her right leg at the end of a day at work. After one month her left leg showed the same symptoms. The LBP began three months prior to the leg symptoms. Over time the symptoms got worse, interrupting her sleep and interfering with her sports. She reduced her level of sports to 30 min twice every week. She decided to go to the GP who referred her to physiotherapy.

To the GP further investigation was unnecessary. An X-ray performed a couple of years ago revealed minor degenerative changes at the level of L4-5. The radiographic report did not reveal anything about the thoracic spine. At the time of referral no medication was
used. She had experienced LBP and neck pain before, but could not remember when.

The patient considered her general health and fitness as good and had not had fever for the last two years. She had no weight loss in the last month. Her bowel and bladder functions were unchanged. Coughing or sneezing did not provoke her symptoms.

3. Physical examination

At the start of the physical examination the patient reported no pain being present. Overall observation revealed a flat back posture. The skin of the right leg was more red than on the left. When sitting, the patient preferred to sit slumped with the left leg crossed over the right. On palpation in prone lying, the right leg seemed colder. Skin movement of the lower thoracic and lumbar spine seemed normal and no increased sweating was noticed. Forward flexion, left rotation and left lateral flexion were normal in range and painless. Extension was limited to 10°. Right rotation was limited to 30°. On lateral flexion to the right, no movement was noticed below L3. However, none of the movements could reproduce pain. Combined movements revealed that the quadrant test of the lumbar spine on the right side was painful locally at the level of L4. This pain was not similar to the patient’s LBP. In full flexion, right rotation was limited to 20° and painful at the thoracolumbar junction. When sustaining them, none of the combined movements reproduced the patient’s symptoms. No signs of neurological conduction deficits were found when testing sensibility, key muscles and reflexes. Straight leg raise tests were normal. The sympathetic slump test revealed a rotation limited to 10° and the patient’s LBP was provoked. On the left side no abnormalities were found. At this time a short reassessment was carried out. The patient reported no present pain, active flexion/right rotation was unchanged. Passive accessory movements (PAIVM’s) of the thoracic spine revealed loss of range-of-motion (ROM) and tenderness on a unilateral posterior-anterior movement (PA) at T10 (see Fig. 2). ROM was decreased at T11 and T12. PAIVM’s of the lumbar spine revealed an increase in ROM and pain at L4 at the unilateral PA
movement. Passive physiological movements (PPIVM’s) revealed a loss of right rotation at L3–4 and T10–11.

Screening tests for the hip revealed nothing significant. These tests included squatting, internal and external rotation, passive flexion/adduction and Patrick’s sign.

4. Clinical interpretation

Non-dermatomal distribution of heaviness can be indicative of a thoracic disorder (Maitland et al., 2005). The close relationship between the thoracic spine and the sympathetic nervous system is well documented in literature (Grieve, 1988; Wright, 1995; Bogduk, 2002). During the physical examination, none of the tests provoked the bilateral leg symptoms. The LBP could be reproduced with the sympathetic slump. Since the main symptom could not be reproduced, indirect signs had to be treated. The working hypothesis based on available information was: ischaemia of the sympathetic nervous system arising from prolonged flexion-rotation postures which loaded the sympathetic chain especially at the hypomobile segments T10–12.

Other possible sources could be of visceral, neurological or vascular origin. Serious pathology was not expected because questions on red flags were all found to be negative (Greenhalgh and Selfe, 2010). Sources of visceral origin were not expected. Bowel and bladder functions were unchanged and the self-reported level of fitness was normal (Maitland et al., 2005). No further signs, indicating lumbar disc pathology, were found.

5. Treatment

The treatment programme consisted of four sessions and a follow-up three months later by telephone. Each treatment session lasted 30 min and a moment of ‘reflection on action’ by the therapist.

5.1. Treatment 1 — day 3

The physical examination revealed a loss of flexion-right rotation range-of-motion. The first treatment consisted of physiological right rotation mobilisation grade III+ focused on the thoracolumbar junction in side-lying. It is suggested that a thoracic dysfunction with an ischaemic component responds well to great range-of-motion mobilisation techniques (Maitland et al., 2005). After 2 min reassessment was carried out. The active tests and PPIVM’s showed no increase in range. Another session of the rotation mobilisation was applied, only this time as a grade IV+. After 1 min, on reassessment range of flexion-rotation had improved with 10° and present pain had increased. As present pain had increased, a grade III+ mobilisation was preformed for 1 min in the same direction to ease treatment soreness. An explanation was given on the working hypothesis and pain mechanisms.

5.2. Treatment 2 — day 9

The patient reported an increase in pain post-treatment which disappeared waking the next day. The heaviness in both legs was still present at the same intensity. Flexion-right rotation was still as limited as it was during the physical examination. The physiological rotation technique did not improve the patient’s main complaints. A unilateral PA movement grade III+ on the right side at the levels T10–11–12 was performed in neutral prone lying for 2 min. Reassessment showed an increase in range of rotation (20°) in flexion. The technique was repeated twice. At this point, rotation to the right was equal to the left in flexion. The sympathetic slump test showed an increase in ROM of rotation but could still reproduce the LBP. The patient was instructed to avoid sustained flexion-rotation positions which repeatedly occurred at work.

5.3. Treatment 3 — day 16

On subjective reassessment the patient reported no treatment soreness. Directly post-treatment the patient experienced a warm feeling in both legs. The heaviness in both legs had decreased. There was no need to walk around at night because of the symptoms. The tenderness on the low back had decreased. The same mobilisation technique (unilateral PA T10–12 grade III+) was used, though progression was made on duration, by treating with three repetitions of 3 min. On reassessment, PAIVM’s showed an increase in resistance-free range-of-motion (increase in distance between A and R1, Fig. 2). The instruction of avoiding the sustained flexion-rotation positions was repeated and self management strategies were trained to preserve mobility. She was asked to perform 10–15 thoracic rotations from left to right and lumbar extension when sitting every hour. The patient was asked to delay the self management strategies until two days post-treatment to note further improvement.

5.4. Treatment 4 — day 23

One week later, the patient reported no heaviness in her legs anymore. The lumbar spine was only tender after a sustained period of sitting (>60 min). With the trained self management strategies the patient could quickly ease this tenderness. Reassessment of the PAIVM’s revealed a slightly earlier onset of resistance but no differences of range-of-motion between right and left. The sympathetic slump test showed a similar rotation to the right as to the left. This treatment session consisted of repeating the advice and self management strategies only. No further mobilisation techniques were applied.

At the follow-up phone call she stated that she remained symptom free and she could use her self management strategies effectively when necessary.

6. Discussion

In this case report a patient with bilateral leg symptoms was treated successfully with passive mobilisation techniques of the thoracic spine, explanation on possible mechanisms causing the symptoms and self management strategies on easing the LBP and unloading the sympathetic trunk. Specific exercises were not introduced until the third treatment, when significant improvement was already achieved. The author is well aware of the fact that symptom relief was not solely due to passive mobilisation. A combination of mobilisation and patient education is of great importance in managing patients with longstanding symptoms (Twomey, 1992; Shacklock, 1999; Moseley et al., 2004).

It is stated that mechanical irritation of the sympathetic chain could cause symptoms of paraesthesia and pain in areas widely spread from the thoracic spine. The role of the thoracic spine is already investigated in clinical patterns involving the upper limb and neck also known as the T4 syndrome. In those cases mobilisation techniques were thought to be a valuable aid towards symptom relief (DeFranca and Levine, 1995). The legs are supplied by sympathetic fibres from the levels T10–L2 (Grieve, 1988). Hence, a dysfunction in the lower thoracic segments and sympathetic nervous system could induce symptoms in the lumbar spine and legs.

Evans (1997) suggested that ischaemia of the sympathetic chain is a result of sustained periods of extreme postures. A flexed and rotated posture is more likely to load the sympathetic trunk (Butler,
When the sympathetic trunk is loaded and ischaemia arises, its nerve fibres can stimulate a vasoconstrictive effect. Avoiding these postures for sustained periods might decrease local ischaemia and therefore stimulate sympathetic activity.

The effects of passive mobilisation have been widely investigated (Twomey, 1992; Wright, 1995; Zusman, 2004). Manual therapy may stimulate the sympathetic nervous system and activate a descending pain inhibitory mechanism. The study of Zusman (2004) suggested that noradrenaline was released by the dorsal periaqueductal grey resulting in sympathoexcitation and hypoalgesia. It could be that the unilaterally applied mobilisation technique directly stimulates local sympathetic fibres as the ganglia have a close anatomical relationship with the vertebral motion segment (Slater, 2002). The chosen technique, effective in this case report, was a slow oscillatory movement. These findings are consistent with the study by Jowsey and Perry (2010).

In this case report multiple components were treated. The effect of every individual component remains unclear even with extensive reassessment after each technique. However, in treating patients in clinical practice it is common that multiple components are treated within one session. The strength of manual therapy lies within treating the whole neuromusculoskeletal system fitted within the biopsychosocial framework (Olson, 2004).

7. Conclusion

There is little scientific evidence for the role of the sympathetic nervous system in relation to leg symptoms. This case report describes the observation that a hypomobile thoracic segment responded to passive mobilisation techniques increasing range-of-motion on active and passive tests and decreasing patient’s symptoms. The lower thoracic area could be considered to be included in physical examination and treatment in patients with bilateral leg symptoms. Could it be possible we are also dealing with a clinical entity ‘the T10 syndrome’? Further research needs be done to support this hypothesis.

References


