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Bilateral injury of deep peroneal nerve in the patient after heart transplant

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In March 2015, a 62-year-old patient with advanced heart failure underwent a failed radiofrequency ablation, followed by paresis of muscles in the anterior compartment of the leg. After rehabilitation, partial recovery of the paresis was achieved. Orthotopic heart transplantation was performed 9 months after ablation at the at the Institute of Cardiology, followed by a bilateral paresis of muscles in the anterior leg compartment. Rehabilitation was implemented. The possible cause of paresis is most likely to be due to food shortages, mainly related to a folic acid deficiency.

Key words: heart transplantation, bilateral peroneal nerve paralysis.

Relationships and Activities: none.

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Clinical situation

In September 2015, a 62-year-old patient with dilated cardiomyopathy, NYHA class III heart failure (HF), left ventricular ejection fraction (LVEF) of 44%, severe mitral regurgitation, history of asystole in 2012, followed by implantation of a cardioverter-defibrillator was admitted to the National Institute of Cardiology. Since 2010, ventricular tachycardia is observed. There was a failed radiofrequency ablation in March 2015, followed by palsy of lower limb extensor muscles (Fig. 1A, B, C). Physiotherapy was carried out, after which the muscle function gradually recovered. Later, a thrombus on the cardioverter lead was detected, which required the administration of oral anticoagulants.

In mid-November 2015, HF exacerbation was recorded.

On December 23, 2015, a heart transplant was performed at the National Institute of Cardiology.

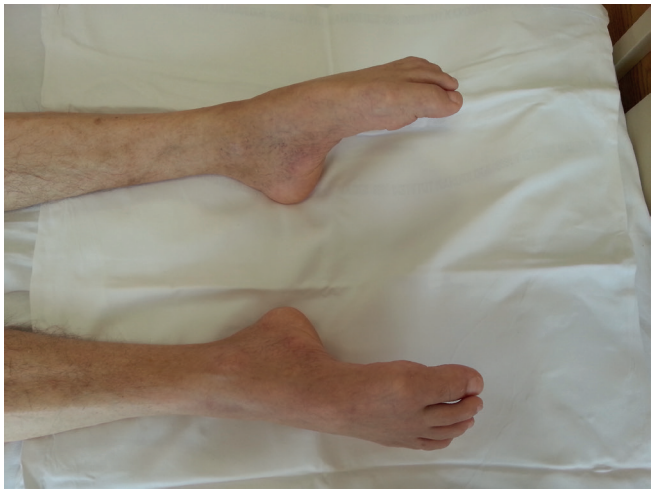
On the 5th day after surgery, palsy of muscles in anterior compartment of the leg was recorded. On day 13, the patient was examined by a neurologist, who revealed normal, symmetrical reflexes in the lower extremities without sensory disturbances. Hypotrophy of the lower limb muscles was noted. Neurologist supposed that a possible cause of palsy is fibular nerve compression at the fibula head. Electrical stimulation of the fibular nerves was ordered.

In parallel with postoperative rehabilitation, physiotherapy was carried out aimed at recovering the fibular nerve function. Despite the improvement of neuromuscular transmission, the full restoration of the feet functional activity was not achieved.

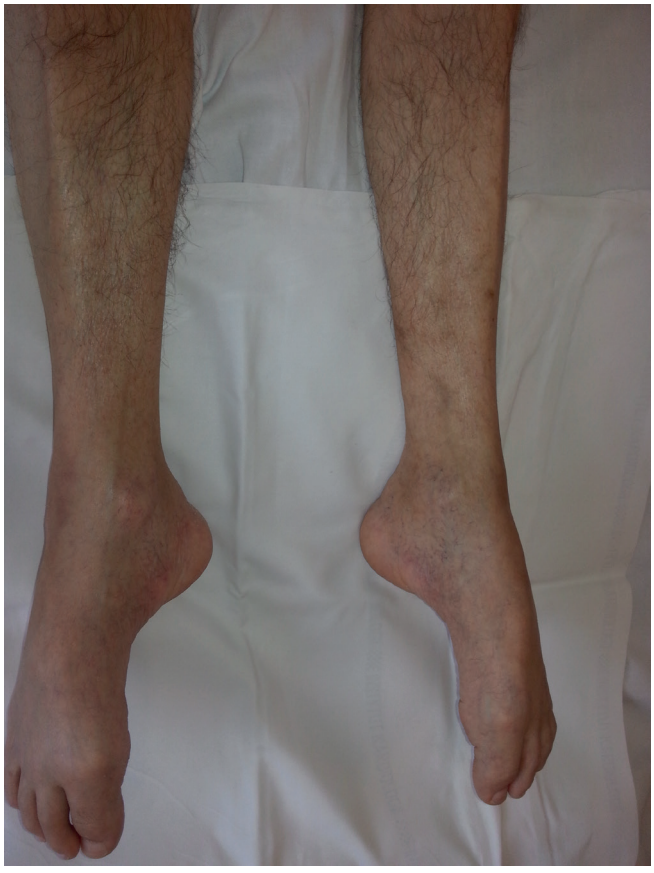
The ability to walk with walkers became possible only on the 52nd day after treatment. The patient's gait had typical features of fibular nerve palsy, which was also characterized by forward inclination of body while walking with walkers.

This type of gait did not appear immediately, but during walking.

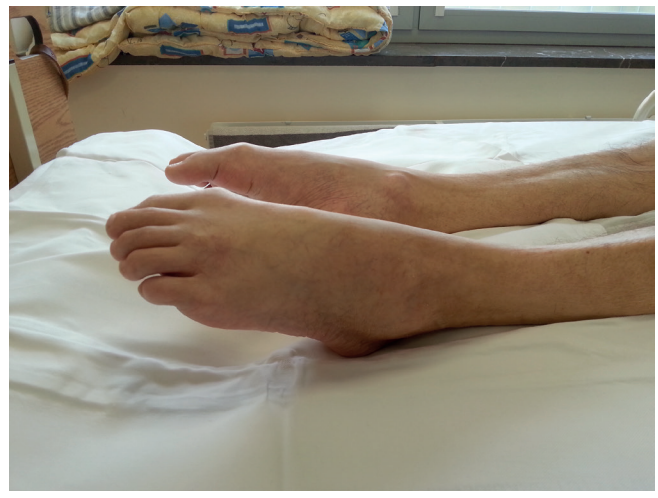
On the 57th day after heart transplant, levels of vitamin B12 and folic acid were determined due to



A



B



C

Figure 1 (A, B, C). Bilateral injury of deep peroneal nerve. Falling feet.

persistent high-colour-index anemia. According to results, vitamin B12 levels were normal, but folic acid values were reduced (2,58 ng/ml with acceptable values of 4,6-18,7 ng/ml).

On the 125th day after heart transplant, the patient was discharged with recommendations to continue rehabilitation at home. On October 17, 2017, the patient was rehospitalized for a second postoperative examination, control of laboratory and instrumental tests. The patient was cachectic and moved with walkers or elbow crutches. There was a permanent paresis of mentioned muscles of both lower limbs. According to the patient, a full course of rehabilitation was performed, including kinesiotherapy and physiotherapy.

Discussion

Bilateral peroneal nerve paralysis after cardiac surgery is a very rare case described in the literature [1]. The dysfunction is occurred due to a partial or complete nerve impairment, followed by leg muscle dysfunction. Paresis is the result of deep peroneal nerve injury. The compensating gait was typical: in order not to touch the ground with the toe, the patient raises his leg high, and when lowering it, first touches the ground with the toe, then the outer edge of the foot and finally the sole. In addition, there was a forward inclination of body while walking with walkers.

On the lower extremity, unilateral paralysis of branches of the common peroneal nerve is most often observed, the most common cause of which is the compression of it by the fibula head. According to literature data, there is a description of one case with similar characteristics obtained by us [2].

One of the possible reasons for paresis after heart transplant could be mechanical compression during surgical positioning, as well as low body and limb weight, which is associated with more superficial location of the common peroneal nerve and its branches. However, it should be remembered that compression by fibula head often leads to injury of the common peroneal nerve, which will cause clinical symptoms related to both the superficial and deep branches [3]. But in this patient, the symptoms of deep branch injury were determined.

The second cause of paresis can be an ischemic stroke. However, strokes and vascular disorders of the central nervous system most often is associated with unilateral symptoms. Clinical manifestations in this patient indicates peripheral nerve injury, but not central.

Also, intervertebral disc disease can be considered the cause of paresis of deep peroneal nerve. But the given patient did not have this pathology, and such a disorder is often manifested by unilateral symptoms.

Nerve injury due to metabolic diseases (diabetes) can also often occur. But this patient was not diagnosed with metabolic diseases (diabetes) [4].

The injury of peripheral nerves as a result of vitamin or mineral deficiency is of great clinical importance for the formation of bilateral peroneal nerve paresis. In favor of this opinion, folic acid levels are indicated.

Folic acid is an indispensable vitamin for the synthesis of nucleic acids (DNA), the deficiency of which can cause nerve injury. Long duration of HF and cachexia could lead to changes in cell membranes, and folic acid deficiency could aggravate this process, which could also cause peroneal nerve paresis. The only argument against this is the absence of data on folic acid levels before surgery and there is no detection of nerve injury before cardiac ablation.

Conclusion

There are many possible causes that can lead to nerve injury. One of the most likely causes is folic acid deficiency. Despite the treatment and rehabilitation measures, the condition of the presented patient did not improve. One of the ways to prevent such a complication is the enhanced laboratory tests at the admission and preparation for surgery, as well as systematic monitoring after surgery, systematic rehabilitation and high compliance of patient are the only factors that are necessary for the rapid recovering.

Perhaps, due to the difficulties of postoperative complications, the use of immunosuppressive drugs, including tacrolimus, and the too short observation and treatment period did not lead to positive dynamics.

Relationships and Activities: none.

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