Severe ischaemic monomelic neuropathy after dialysis access revision surgery

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Introduction

Dialysis access surgery is frequently performed to allow long-term haemodialysis in patients with chronic kidney insufficiency. Although already first described in 1979 ischaemic monomelic neuropathy (IMN) is still an underappreciated and barely known cause of pain and limb dysfunction after dialysis access surgery.⁴ Even though IMN is a rare complication, it imperatively requires early recognition to intervene in time. IMN is a form of dialysis access associated ischaemic syndrome, a so-called steal syndrome of the Vasa nervorum. It is defined as an acute infarction of a distal extremity nerve of one limb due to acute interruption of the blood flow.⁵ Accordingly it affects as well sensory as motor branches of the affected nerve in the concerned limb. The clinical manifestation of IMN can widely vary but usually consists of heavy, burning pain with an abrupt onset and persistence of the pain without ease. Additionally, IMN leads to motor dysfunction. Nerve conduction studies show distal sensory and motor damage and signs of axon loss.⁶ In contrast to dialysis-associated shunt-steal-syndrome (DASS) ischaemic monomelic neuropathy is diagnosed only if there is no steal detectable in ultrasonography.⁷ As soon as IMN is diagnosed distal perfusion of the Vasa nervorum should immediately be improved, since delay can lead to severe and irreversible limb dysfunction. Although in literature the beginning of symptoms is characterized to be briefly after dialysis access surgery, delay of treatment is described from some hours up to 5 months. The most common treatment is primary closure of dialysis access to stop the steal syndrome of the Vasa nervorum. Risk factors to develop IMN after dialysis access surgery are especially preexisting occlusive arterial diseases as well as diabetic neuropathy.⁸

Case summary

We introduce the case of a 44 years old female patient that suffered from ischaemic monomelic neuropathy after dialysis access surgery. In a first attempt a downsizing of the arterial anastomosis was performed. Still, only the subsequent ligation and partial removal of the graft led to normalisation of the symptoms.

Case report

In this case report the patient’s underlying disease is Wegener’s granulomatosis. Due to an occlusion of a twelve-year-old brachial-axillary prosthetic graft, we created a complete new brachial-jugular graft because of lack of any appropriate vein. Immediately postoperative the patient presented with rest pain, hyposensitivity and loss of strength in her hand. There were no clear signs of finger malperfusion. Even though we measured a high flow of 2.2l/min, the digital plethysmographic pulse wave did not show a pathologic result with or without graft compression. After exclusion of a steal syndrome [image 1+2], as defined by dialysis-associated shunt-steal-syndrome (DASS) we assumed an ischaemic monomelic neuropathy in which loss of sensory and motor function is seen. A downsizing of the arterial anastomosis was performed with no improvement of the symptoms. Therefore a ligation and partial removal of the graft was necessary. Afterwards the symptoms slowly diminished. Two months later the patient had nearly no symptoms left and the electromyoneurography showed normal results.

Conclusion

The number of patients that suffer from end-stage renal disease is worldwide steadily growing. Ischaemic monomelic neuropathy after dialysis access surgery is rare and often not or too late diagnosed. The incidence of IMN is not precisely described, probably because the entity may easily misdiagnosed for other vascular and neurologic disorders.⁹ If IMN is early recognized and instantly dealt with, morbidity can be reduced. Therefore, it is crucial to keep in mind this disease pattern. Also we suggest that every case of IMN should be documented and if possible published to enhance experience to this barely known disease.

Bibliography: