Case Report

Reflex sympathetic dystrophy following vascular access surgery for haemodialysis: influence of peripheral ischaemia?

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Introduction

Reflex sympathetic dystrophy (RSD) is a descriptive term meaning a complex condition of the affected body parts that is painful and disabling. The diagnosis is primarily clinical. Several authors have proposed diagnostic criteria [1–4], which are all more or less covered by the definition of the American Association for Hand Surgery (AAHS).

The AAHS defined three criteria, necessary for diagnosis: (i) diffuse pain; (ii) loss of hand function; and (iii) sympathetic dysfunction. The latter meaning objective evidence of significant autonomic dysfunction, as reflected by skin, soft-tissue, or blood flow changes, such as temperature increase or decrease, sweating increase or decrease, hair growth increase or decrease, nail growth increase or decrease, atrophy of skin or subcutaneous tissue, oedema, bloodflow increase or decrease, Sudeck’s osteoporosis, or characteristic bone scan [5]. In this case report three patients will be described who developed RSD following vascular access surgery for haemodialysis.

Case reports

Patient 1

A 71-year-old woman, with a 7-year history of hypertension, non-insulin dependent diabetes mellitus, intermittent claudication and a 4-year history of renal failure underwent placement of a Brescia/Cimino AV fistula in the left wrist. The fistula did not mature sufficiently and the patient underwent a second operation for implantation of a Gore-tex® AV graft in the left forearm. Postoperatively there was pain in the left hand, which did not subside. Digital blood pressure values were 30 mmHg for the first digit, 40 mmHg for digits two to four and 20 mmHg for the fifth digit. When the fistula was closed with manual compression the blood pressure for the second digit rose to 100 mmHg. An angiography showed a stenosis of the left subclavian artery, but following percutaneous angioplasty the pain remained. Subsequently the BC fistula was closed and during this operation, thrombosis of the Gore-tex® AV graft was noticed and thrombectomy was carried out. However, hand ischaemia did not improve and 10 days later the AV graft was also closed.

After this operation digital blood pressure values returned to normal. Altogether, the period of ischaemia in the left hand had lasted 7 weeks. A jugular catheter was placed and haemodialysis could finally be initiated. To date the left hand remains painful, afunctional, and shows sympathetic dysfunction (Figure 1).

Patient 2

A 59-year-old woman with a 40-year history of hypertension and renal failure due to phenacetin abuse, underwent placement of a brachial AV fistula in the right elbow. After 7 months the fistula thrombosed and a Gore-tex® AV graft was implanted in the left forearm. Subsequently, ischaemia of the left hand developed, illustrated by digital blood pressure values of 40 mmHg for the first digit and 45 mmHg for digits two to five. Two weeks following implantation, the graft was tapered to diminish the fistula flow. While ischaemia subsided, as proven by normal digital blood pressures, diffuse pain, sympathetic dysfunction and loss of function of the left hand remained. Fifteen months later the fistula thrombosed and a new graft had to be placed in the left forearm. Following pulmonary embolism in the past, this patient was a chronic user of coumarin derivatives. These had caused prolonged post-puncture bleeding with the Gore-tex® graft, and therefore a dacron graft was implanted. This fistula was revised four times and eventually removed after 6 months as a consequence of recurrent infection and thrombosis. A Diastat® AV graft was then placed in the left upper arm, but had to be removed after 6 months for comparable reasons as the dacron graft.
RSD following AV bridge graft surgery

Changes in the skin and bone of the affected extremity. Sixty-five per cent of cases are precipitated by trauma, mostly a fracture. Operation has been reported to be the preceding factor in 19% of cases [6]. RSD following placement of an AV graft for haemodialysis has been described only once [7]. This should not mean that it is a rare complication of vascular access surgery. Cases of unexplained limb pain and swelling after AV bridge graft placement are often seen. When the criteria for diagnosis are strictly followed these patients might suffer RSD more often than is realised.

The mechanism responsible for RSD is not known. However, nowadays most scientists agree that the syndrome is started by nerve injury. This would cause secondary changes in the dorsal horn and/or pain maintaining sympathetic complications [8].

As well as the aetiology, a successful therapy is not known. The only therapy, effective in large studies is aimed at the interruption of the activity of the sympathetic nervous system by means of regional intravenous sympathetic blockade (RIS) or stellate ganglion blockade (when RSD affects the arm) [9,10]. The interventions are not curative, but are aimed at relieving the pain. Nowadays the efficacy especially of RIS blockades is in discussion, since studies failed to show any change following this form of treatment [12].

Trying to prevent RSD therefore seems the only appropriate way to handle this syndrome. For patients, who need haemodialysis, prevention of RSD is especially important. Loss of hand function for these patients would mean (besides from problems it would give to any patient) exclusion from CAPD in the future.

We believe there are possibilities to prevent RSD in some cases. When the operation is based on well-defined criteria and performed using normal operative techniques, as is normally the case, nothing more can be done to prevent RSD. Patient 3 is an example of such a case. Patients 1 and 2, however, developed RSD not consequent to direct nerve trauma, but following a period of ischaemia. Probably the ischaemia caused nerve trauma and thus RSD. For all kinds of reasons, the period of ischaemia lasted 2 weeks in Patient 2 and even 7 weeks in Patient 1. While the ischaemia could be treated, RSD remained. Trying to diminish delay in treating the ischaemia could have been an important factor in the prevention of RSD in these patients.

In conclusion, we think it is important when signs of ischaemia occur following vascular access surgery, not to hesitate to reoperate as soon as possible, to prevent the development of RSD.

Discussion

Reflex sympathetic dystrophy is a syndrome of burning pain, hyperalgesia, swelling, hyperhidrosis, and trophic skin changes in the skin and bone of the affected extremity. Sixty-five per cent of cases are precipitated by trauma, mostly a fracture. Operation has been reported to be the preceding factor in 19% of cases [6]. RSD following placement of an AV graft for haemodialysis has been described only once [7]. This should not mean that it is a rare complication of vascular access surgery. Cases of unexplained limb pain and swelling after AV bridge graft placement are often seen. When the criteria for diagnosis are strictly followed these patients might suffer RSD more often than is realised.

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References


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**Fig. 1.** Photograph of the left hand of Patient 1. Note the trophic skin changes, oedema and increased nail growth. The hand is completely afunctional.

From then on the patient was dialysed by a CAPD catheter. Eight months later she died as a consequence of heart failure.

**Patient 3**

A 70-year-old woman with a 6-year history of hypertension was diagnosed to suffer renal failure. Diagnostic tests revealed Kahler’s disease. She underwent placement of a Brescia/Cimino fistula in the left wrist for haemodialysis. Following this operation reflex sympathetic dystrophy developed in the left hand. There were no signs of ischaemia and digital blood pressures were normal. Because of insufficient maturation of the BC fistula, a jugular catheter was placed to enable haemodialysis. Two months later she died, in spite of four chemotherapeutic courses with vincristin, adriamycin, and dexamethasone (VAD) following diagnosis.

All three patients underwent an examination by a neurologist, who could not identify a neurological cause for the clinical image.

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