Invited Comment

Management of peripheral arterial disease in patients with end-stage renal failure

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Introduction

It is uncertain whether uraemia per se promotes the genesis of arterial lesions, but undoubtedly the risk of peripheral arterial complications is increased in end-stage renal failure (ESRD) and their evolution is more rapid than in patients without ESRD. It follows that arterial monitoring using non-invasive methods is important in patients with ESRD. Such patients may present with occlusive arterial disease, aneurysmal arterial disease, or a combination of both. In principle, the lesions are accessible to the same therapeutic interventions as in the patients without ESRD, but at the price of a definitely higher operative risk, which nevertheless remains acceptable. While dialysis and transplantation have transformed the prognosis of ESRD in establishing the indication for intervention, it is still important to consider that life expectancy remains reduced in ESRD patients. The indication must therefore balance the risk of the natural evolution of the disease versus the risk of intervention, taking into account not only of life expectancy, but also of the quality of survival.

In the current situation of shortage of renal allografts, grafts are given with preference to the recipients with the longest life expectancy. In this context it should be considered that arterial lesions are amenable to treatment prior to or at the time of transplantation. Such patients should therefore not be considered an absolute contraindication against renal transplantation.

Patterns of arterial lesions

Atherosclerotic plaque

This is by far the most frequent lesion. Amongst patients with ESRD it is often diffuse, extended, and circumferential. It may produce either stenosis or complete occlusion. The haemodynamic consequences depend, inter alia, on the development of collateral circulation. Plaques are often calcified in ESRD patients, but such calcified atherosclerotic plaques must be clearly distinguished from non-obstructive medial calcification of the Mönckeberg type. Atherosclerotic plaques carry a high risk of thromboembolic or atheroembolic complications. The topography of atherosclerotic plaques is the same as in patients without ESRD. The most frequent localizations include cerebral arteries (especially at the carotid bifurcation), aortoiliac, visceral, and limb arteries. One should not forget that ESRD may have been caused by atherosclerotic renal arterial occlusive disease, either as a result of thromboembolism or of compromised renal perfusion.

Aneurysms

These are reported with increasing frequency in the general population and in patients with ESRD as well. Their most frequent locations are aortic bifurcation, thoracoabdominal aorta, iliac arteries, and femoropopliteal arteries. Occasionally they develop as a long-term complication in the arteries feeding arteriovenous fistulae. Again, one should consider that thoracoabdominal aneurysms may have caused ESRD by thromboembolism.

Management

Amongst the variable clinical presentations, certain patterns can be distinguished, knowledge of which impacts on patient management.

Cerebrovascular lesions

As in the general population, cerebrovascular lesions, especially at the level of the carotid bifurcation, may cause stroke or TIA by thromboembolism or compromised blood flow. The latter mechanism is by no means rare, given the fact that stenoses are often tight and involve several vessels in series. Particularly typical is the occurrence of seizures or symptoms of vertebrobasilar insufficiency during haemodialysis sessions. The diagnosis of the lesions is easy with duplex scanning and CT. As in the general population, tightly stenotic, symptomatic, or irregular lesions are the best candidates for revascularization. Carotid endarterectomy has transformed the neurological prognosis of these
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patients. Morbidity is only slightly higher than amongst patients without ESRD [1].

Aortoiliac aneurysms

As in patients without ESRD, a natural history of aortic aneurysms carries the risk of major complications, the most important of which is rupture. Amongst patients with ESRD, the evolution is more rapid, and this is at least in part due to coexisting hypertension. Amongst transplant recipients, evolution may be further accelerated by steroid therapy. Aneurysms are most frequently located in the infrarenal aorta. Results of emergency surgery after rupture remain dismal with mortality rates of at least 50%. In contrast, at least in patients without ESRD, mortality is only around 4% after elective surgery. The factors that increase operative risk are now well known. In patients with ESRD, mortality after elective surgery is higher, approximately 8%, than in patients without ESRD. This is accounted for by complicating factors, since in the absence of unstable coronary disease, congestive heart failure, or severe respiratory disease, surgical results are quite good [2,3]. In poor-risk patients with ESRD, endovascular devices promise to become the solution of choice.

The use of renal allografts is widespread and thus the discovery of an aortic aneurysm above a functional renal allograft is not unusual. The aneurysm is easily

Fig. 1. Bilateral carotid endarterectomy specimen in an ESRD patient. Note the necrotic structure of the plaque.

Fig. 2. Bilateral total occlusion of both internal carotids in an ESRD patient with seizures during haemodialysis.

Fig. 3. Aneurysm of the left brachial artery feeding an angioaccess arteriovenous fistula established 12 years before.

Fig. 4. Thoracoabdominal aneurysm above a renal transplant.
accessible to surgical treatment using a modified technique.

*Infrainguinal occlusive disease*

Most often patients are seen for severe claudication. An increasingly diversified therapeutic armamentarium permits variations in the approach to the patients’ condition. Surgical revascularization is the most invasive procedure, but it gives the best and most durable results. It should be reserved for good-risk patients with ESRD. For higher-risk patients, endovascular methods and extra-anatomical bypass are available, and in selected cases permit treatment of severe claudication to save the limb.

Fig. 5. External iliac stenosis causing renal transplant failure and claudication.

In the presence of infrainguinal occlusive disease, evaluation by the vascular surgeon proceeds in three steps (i) it must be clarified that tissue necrosis is the result of ischaemia; (ii) the arterial lesion responsible must be identified and patency of outflow vessels must be established; (iii) revascularization procedures must then be initiated.

The first step requires clinical examination, demonstration of absent pulses, and duplex scanning; more sophisticated methods are seldom necessary. The gold standard for identifying the lesion remains the arteriogram. High-quality images are necessary. In the absence of aortoiliac occlusive disease, conventional arteriograms, using direct femoral puncture, provide the best results. Most frequently, concomitant presence of femoral, popliteal, and infrapopliteal stenoses is responsible for tissue necrosis. This explains why most frequently the treatment is best achieved through distal bypass. It becomes increasingly rare that no revascularization procedure is feasible. Sufficient run-off for a bypass may be created even for diseased arteries as distal as those of the foot. The odds of success in the short and especially in the long run are very much dependent upon the availability of a suitable venous autograft. Other materials give dismal results in this setting. The development of reversed or *in situ* saphenous bypass has reduced the rate of primary major amputation. Distal minor amputations, and debridement for infection still are often necessary, even after successful revascularization. This extends the duration of the stay in hospital. In this setting, a multidisciplinary approach of foot care centres is desirable. The reported results of limb salvage surgery are less favourable amongst patients with ESRD than in those without, especially in diabetic patients. Operative mortality is approximately 9% with a range of 2–15%, depending essentially upon the type of recruitment and selection of patients. The reported rates of limb salvage at 2 years vary from 70 to 90%. If severe infection is present a number of amputations remain necessary despite successful revascularization [5–9].
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Fig. 7. (a) Gangrene of the foot in an ESRD patient. (b) The preoperative angiogram did not seem to show a suitable outflow for a bypass. (c) A saphenous bypass was, however, possible and achieved limb salvage.

Conclusion

Peripheral arterial disease may threaten the life expectancy and the quality of life of patients with ESRD. It is more often amenable to revascularization than generally thought.

References