

## INFECTIOUS COMPLICATIONS OF ARTERIOVENOUS ePTFE GRAFTS FOR HEMODIALYSIS

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**Background.** Insufficient venous vasculature disallows autologous arteriovenous fistula creation. In this case an arteriovenous conduit of expanded polytetrafluoroethylene (ePTFE) interponed between artery and vein is used for hemodialysis. Although arteriovenous graft infection is an infrequent complication, infected grafts cannot be used for hemodialysis and can cause infection, sepsis and bleeding. Treatment options remain limited but the general approach is to maintain functional angioaccess and to eradicate infection.

**Aim.** to summarize current knowledge of the prevention and treatment of arteriovenous graft infection.

**Methods.** literature review

**Conclusions.** ePTFE graft present an unreplaceable material used for angioaccess in patients with an insufficient venous vasculature. A number of risk factors causing graft infection is known. Since hemodialysis patients are a high-risk group, an effective strategies for graft infection prevention and early diagnosis should be determined. Among the most important risk factors belong surgical procedure, recurrent venipuncture and other infection disease. The prostheses should be removed when infected, especially in the presence of sepsis. In case of “localized infection”, the prostheses can be removed partially only under the condition of careful patient selection and subsequent follow-up.

### INTRODUCTION

Chronic hemodialysis treatment requires vascular access. This can be provided by central venous catheter, native arteriovenous fistula or arteriovenous graft interponed between arterial and venous blood vessel. A native arteriovenous fistula is considered the most favourable in terms of function, duration and absence of complications<sup>1,2</sup>. There are always however, a number of patients with insufficient autologous venous vasculature where the native superficial venous vessels are hypoplastic or attrited due to previous surgical procedures. For this reason a central venous catheter is introduced or an an arteriovenous conduit is implanted in these patients<sup>2</sup>. Various materials – autologous, homologous and heterologous – are used as a graft. Various evaluations of these materials have been reported but the common view is that all are associated with a large number of complications<sup>3–5</sup>. Currently, the ePTFE conduit is preferred for its availability. A central venous catheter vascular access is indicated if acute hemodialysis procedure is necessary and all other possibilities for angioaccess have been exhausted. Despite associated complications, central venous catheters are increasingly common in Europe as well as in USA and Canada<sup>2</sup>.

Initially, natural materials were used as autologous, homologous and heterologous vein and arteries and later artificial materials such as modified polyester and ePTFE

vascular prostheses began to be used. Of these, ePTFE interponate best meets the requirements of sufficient arteriovenous graft: availability and handling, material inertness, rapid healing, low incidence of post venipuncture bleeding, resistance to infectious complications and thrombosis, and long term good cumulative access function. Conduits made of ePTFE have been used for some time. Initially they were used in surgical procedures performed on the venous system<sup>6</sup>. The first experience with ePTFE conduits in human arterial circulation were published in 1975 (ref.<sup>7</sup>). In 1973, Volder was the first to use the ePTFE conduit as an arteriovenous graft for hemodialysis<sup>8</sup>. In the course of time ePTFE conduit has become the gold standard for arteriovenous graft<sup>5,9–12</sup>. Currently, there are many ePTFE conduits producers and various product modifications are available. The ePTFE conduits vary in length of fibrils, wall thickness and wall reinforcement, number of wall layers, the cover of the internal surface and shape of the conduit. Conduits have been compared in a number of studies but no substantial benefits between different types have been reported<sup>4</sup>.

Above all, the function and duration in terms of the emergence of stenotic and thrombotic complications have been monitored<sup>13–16</sup>. These complications are essentially repairable. A more serious complication is graft infection<sup>17</sup>, whose spread in the organism can cause general infection. In hemodialysis patients infection is associated with high

morbidity and is the second most common cause of death in this group of patients<sup>18-21</sup>.

Studies have shown that a vascular access is one of the most important risk factors for infection and bacteremia in hemodialysis patients<sup>22,23</sup>. The hospitalization rate for these patients is double the general population and in 20% of cases the cause for hospitalization is infection<sup>24,25</sup>. Epidemiologic studies have shown that angioaccess type affects the risk of infection or bacteremia emergence<sup>22,26</sup>. At highest risk are patients with central venous catheter followed by patients with tunelized catheter and arteriovenous graft, while native arteriovenous fistula present low risk of infection<sup>23,26,27</sup>. In patients with central venous catheter infection, complications occur in 20 to 50% and the risk of infection is associated with the duration of catheter positioning<sup>28-31</sup>. The lowest risk of infectious complications is in patients with native arteriovenous fistula, where the incidence is from 2 to 3% (ref.<sup>13,29,32</sup>). In patients with arteriovenous graft the incidence of infection is from 11 to 35% (ref.<sup>13,32-35</sup>), and infection complications associated with arteriovenous graft are approximately one third more than arteriovenous fistula<sup>23</sup>. Also later published studies have shown that the incidence of infection in arteriovenous graft is ten times more common than autologous fistula and significantly lower than central venous catheter<sup>13,36</sup>. In Europe, infected graft present a problem demanding complex solution as patients autologous vasculature is usually attrited and arteriovenous graft was the final chance for angioaccess without central venous system cathetization. Arteriovenous graft infection manifests locally or can spread to the organism.

Local arteriovenous graft infection manifests as skin affection, in addition, infected grafts tend to thrombosis, perigraft hematoma and pseudoaneurysm. These complications are the leading cause of the access loss in 60%, while infection causes graft failure in 35% (ref.<sup>37</sup>). The incidence of graft infection increase with length of function: there is a higher incidence in the second and third year<sup>36</sup>.

Serious overall symptoms of infection are sepsis, metastatic infectious complications such as endocarditis, arthritis, pulmonary embolism and osteomyelitis<sup>38</sup>. Hospitalization due to sepsis is associated with increased incidence of myocardial infarction, congestive heart failure, cerebral stroke and peripheral artery ischemic disease in the following years<sup>39</sup>. Arteriovenous graft infection management includes treatment of local and general complications as well as maintaining the angioaccess. The task is to maintain functional arteriovenous graft through local treatment and antibiotics administration as long as possible.

A number of risk factors for arteriovenous graft infection have been recognized. Risk factors result from graft localization – infection is more common in lower extremities<sup>40,41</sup>, insufficient antisepsis during surgical procedure<sup>34</sup>, technique of venipuncture with risk of hematoma creation or infection contamination<sup>42</sup>. Clearly, the longer the arteriovenous graft is used for hemodialysis the higher is the risk of graft infection emergence<sup>33,36,43</sup>.

The incidence of arteriovenous graft infection is affected by a large number of factors such as: impaired immuno-

surveillance in hemodialysis patient caused by neutrophils dysfunction in uremia<sup>44-46</sup>, obesity, diabetes mellitus<sup>47</sup>, hypalbuminemia<sup>36,48</sup>, insufficient personal hygiene<sup>49</sup>. Another risk factor is HIV infection<sup>50,51</sup>. The arteriovenous graft as well as all vascular prostheses are compromised by other infectious diseases that can spread through blood circulation and colonize the conduit<sup>52</sup>. As in vascular surgery, the administration of antibiotics can suppress symptoms of arteriovenous graft infection. However, infection cannot be eradicated and presents a permanent risk as source for local or general infection. A considerable risk factor for bacteremia is its existing presence<sup>23</sup>.

### Microbiology

Usually an angioaccess is infected with common skin microorganisms represented by grampositive bacteria. In most cases, the causative organism of the angioaccess infection is *Staphylococcus aureus* or other grampositive pathogens, like coagulase-negative staphylococci. *Staphylococcus aureus* is demonstrated in almost 68% cases of angioaccess infection. Other grampositive bacteria have been found in 20 to 60% (ref.<sup>36,53</sup>).

Less commonly, gramnegative bacteria are the cause of infection with the demonstration in 28% of cases<sup>36,54</sup>. Their occurrence is associated with the transmission from hemodialysis machines<sup>55</sup>. The most common route of infection entry is an angioaccess, followed by urinary tract, gastrointestinal tract and respiratory tract.

Infections caused by *Staphylococcus aureus* occur frequently often and are associated with more complications and worse outcomes than other infections. Metastatic skeletal infections, endocardial infections and brain abscesses are among the most serious infectious complications. Infected arteriovenous graft is the source for septic emboli as has been confirmed in 12% of infected grafts<sup>36</sup>. The morbidity rate in patients with bacteremia caused by *Staphylococcus aureus* ranges from 13 to 44% (ref.<sup>38,56,57</sup>). Methicilin resistant *Staphylococcus aureus* (MRSA) is an increasing problem in dialysis centers. A risk factor for MRSA is a previous use of antibiotics<sup>58,59</sup>. An infection caused by MRSA increases the risk of 90-days mortality of 70% in comparison with an infection caused by methicilin sensitive pathogen<sup>60</sup>. The use of linezolid may be beneficial in MRSA soft-tissue infection treatment<sup>61</sup>. The eventual drug penetration into infected grafts has not been confirmed and needs to be evaluated in further studies. Vancomycin resistant enterococci infections are also more often diagnosed in hemodialysis patients. In the USA the incidence of such infection has at least doubled<sup>62,63</sup>. The possible cause is excessive use of antibiotics, especially vancomycin and the third generation of cephalosporins<sup>62,64</sup>.

### Prevention

Compliance with the rules of aseptic surgical techniques and accurate operating technique are the main prevention of early infectious complications following graft implantation<sup>65</sup>. An early infection complication is almost always associated with microbial contamination. Early infection in association with hematogenous contamina-

tion is extremely rare: an incidence of 1.1% is reported<sup>42,66</sup>. Besides inadequate asepsis practices in the operating room, an unqualified wound dressing can cause infection. Such complication is prevented by prophylactic use of antibiotic, e.g. cefazolin<sup>67</sup>. Vancomycin single administration after graft implantation was found to lead to significant decrease in early infectious complications<sup>34</sup>. However, the use of vancomycin increases the incidence of the vancomycin resistant enterococci<sup>68,69</sup>. Also prophylactic use of rifampicin decreases the incidence of *Staphylococcus aureus* infection, but its administration is associated with the risk of toxic reactions and with bacterial resistance after short-term administration<sup>70,71</sup>. The perigraft reaction needs to be distinguished from infectious complication. The former manifests with edema, skin erythema and heat and the presence of pain around the vascular prosthesis. The local finding can mimic graft infection. Initially, fluid collects around the prosthesis, cyst formations and later, infiltrates having a fish meat structure on the cut can occur. The perigraft reaction can arise anytime after prosthesis implantation (a few days to months). Histologically it has the structure of a chronic seroma<sup>72-74</sup>. In patients with renal end-stage disease the perigraft reaction incidence is higher<sup>66</sup>. Due to the local finding graft cannulation is not possible. The perigraft reaction was present in the type of prosthesis Diastat<sup>75</sup>.

In practice, the usual rules for medical personnel apply. These include hand washing and, scrubbing, rinsing and disinfection of the forearms. The aseptic technique for the cannulation is also important<sup>44</sup>.

### Diagnosis

Clinical findings such as warmth and redness of the skin, local pain, edema, serous or purulent secretion from the wound (after cannulation) or abscess usually lead to a diagnosis of infection. There may also be general symptoms of temperature and chill. Tunnel infection is extremely serious. It is associated with pus around the graft and sometimes the graft floats. A chronic fistula with purulent secretion occurs on the side of the cannulation. The early manifestation of graft infection can present as bleeding from the anastomosis. Infected arterial anastomosis of the graft on the side of the brachial artery can cause massive haemorrhage<sup>76</sup>. If the extent of the involvement is unclear, ultrasonography examination should be performed to detect fluid around the graft<sup>53</sup>. If wound or puncture secretion is present it is necessary to examine a sample of the secretion. If the patient is septic, a hemoculture tests should be done.

In terms of the infection, dysfunctional thrombosed grafts that are left in the patient are a special issue. This can be infected and become a source of infection, causing serious complications<sup>50,77</sup>. In the presence of sepsis of unknown etiology, we should be aware of this potential source of infection and the patient should undergo labeled leukocyte scintigraphy<sup>78</sup> or positron emission tomography scanning (FDG-PET)<sup>79</sup>. The PET CT is considered as an easier and more accurate diagnostic method than the Indium 111-leukocyte imaging<sup>79,80</sup>.

### Graft Infection Therapy

Treatment for the graft infection can be conservative, surgical or, most commonly, a combination of both. In terms of surgical treatment, the principal is removal of the infected prosthesis or infected part of the prosthesis – total graftectomy (TGE), subtotal graftectomy (SGE) or partial graftectomy (PGE)<sup>53</sup>. In the presence of sepsis, tunnel infection or bleeding from the anastomosis, total graftectomy is indicated<sup>81,82</sup>. After removal of the graft, surgical treatment of the anastomosis is required – venous anastomosis is re-sewn and arterial anastomosis is treated with a venous patch. Brachial artery ligation is indicated only when there is bleeding from the anastomosis. Exceptionally, brachial artery ligation can lead to hand ischemia. In such cases, venous bypass performed on the intact subcutis may be necessary to maintain sufficient hand circulation.

If the autologous venous circulation is extremely sclerotic, the bypass cannot be performed. Hand ischemia is preferable to life-threatening arterial bleeding. We know from practice, that the artery can be ligated, if the hand had not been in danger of ischemia while the graft was functional<sup>83,84</sup>. The brachial artery can be ligated below the branching point of the deep brachial artery. The ligation is performed on the intact tissue: Generally it is safe and the results are good without ischemic complications or bleeding<sup>85</sup>. When the arterial anastomosis is not affected by infection, it is acceptable to leave a small piece of the prosthesis (2 to 3 mm) at the side of the arterial anastomosis and sew the lumen of the prosthesis<sup>86,87</sup>. This procedure is also known as subtotal graftectomy. It is indicated in patients with infected prosthesis, but without clinical symptoms of sepsis. The procedure is beneficial as it reduces the risk of arterial and nerve injury and the risk of arterial bleeding. After the graft removal, the necrotic tissue must be removed and lavages are performed<sup>53</sup>.

The procedure, when the brachial artery is sutured end-to-end, or when small pieces of left prosthesis are sutured, is not always radical, while the potentially infected tissue surrounding the graft is left in situ. From here the infection can spread and can cause arterial bleeding. The risk of new arterial bleeding in patients with positive hemoculture is 20%. The resection of the artery at the site of anastomosis and the replacement with a venous graft is recommended. Thus a complete infected tissue debridement is allowed. This procedure is radical in the elimination of infection and it also prevents arterial stenosis<sup>88</sup>. It should be followed by a microbiological examination of the prosthesis and, subsequently by targeted antibiotic treatment<sup>19</sup>. Before the microbiological test results are known, patients are treated empirically, usually with a combination of gentamicin or vancomycin and the third generation of cephalosporins<sup>50,53,54</sup>. Sequential antibiotic therapy is set up according to the test results. The dosage of antibiotics is renal function dependent and the treatment duration is 3 to 6 weeks on average<sup>50,53</sup>.

Partial graftectomy is indicated if only a part of the prosthesis is infected. The intraoperative finding is fundamental – all the parts of prosthesis, except the affected

part, must be fixed in the subcutis and free of infection. In such situations, the affected part of the prosthesis is removed, the intact parts of the prosthesis are ligated and the subcutis is oversewn through-and-through at the side of the anastomosis. The infected subcutis is partitioned and a new prosthesis is implanted in the intact subcutis, connected only with the unaffected sections of the original prosthesis. This procedure allows us to dialyse the patient through the original vasculature so he or she does not need to be catheterized. However, this procedure remains controversial, while the original graft often works as a source of infection. The infection involvement is not exactly clear during the surgery and the PGE carries a risk of further infectious complications and bleeding. The PGE has been evaluated in many single-centre studies and good results have been reported in 74–80% (ref.<sup>37, 53, 81, 82, 89</sup>). Although, this approach is not generally accepted. Some centers prefer the TGE since the PGE is associated with a large number of infectious complications<sup>77</sup>.

In some cases there is only a “local infection” presented as a skin necrosis, abscess or fistula at the point of entry of infection. The patient is generally symptom free. A graft may be infected, but the local finding is discreet and can be treated with local surgical therapy using drainage, elution and suitable antibiotic administration<sup>81, 90</sup>. With this approach, the majority of infections can mitigate but are not treated.

In general, prompt treatment of skin or distant infection is fundamental for preservation of the graft. Only well-timed and potent antibiotic therapy can positively affect the incipient skin and subcutis involving local infection. The infected graft that cannot be treated by antibiotic therapy, must be resected or completely removed. The next step is to provide an alternative angioaccess, usually catheterization of the central venous system, and intravenous antibiotic therapy. After the resolution of infection, a sequential procedure providing a permanent angioaccess by graft placement is scheduled. Preferably the graft is placed in another location than the previous one. Treatment of the infected graft is associated with hospitalization and thus high care cost<sup>36, 56</sup>.

Typical therapeutic models for graft infection treatment mentioned above have obvious disadvantages. The main disadvantage is that no alternative localization exists for the placement of the graft. This is the situation when attempts for angioaccess have been repetitively performed on the opposite upper extremity and all have failed, or the central venous system is obturated. Other materials were used as a substitution for autologous vessel circulation to solve this problem.

In 2000 Matura et al.<sup>91</sup> published a prospective study outcome. The authors used a cryopreserved femoral vein instead of prosthesis in the infected field, or when all others modalities for angioaccess were attrited. The relative resistance to infection of this material was confirmed. The material was used in the field of infection in 38 patients. In 20 patients, infected ePTFE prosthesis was replaced by the cryopreserved vein in one session. No new infection

nor any other complication such as bleeding or pseudoaneurysm of the cryopreserved graft appeared. One year primary cumulative function was 49% and secondary patency was 75%. In comparison with a group of 68 patients with implanted ePTFE prosthesis, the patency of the graft was similar and the resistance to infection was higher in the group with the cryopreserved graft<sup>91</sup>.

In 20 patients at high risk for infection Bolton et al.<sup>92</sup> used the cryopreserved femoral vein. The localization of the vein was the thigh, the upper extremity and the chest. They failed to confirm the positive results of Matura et al.<sup>91</sup> There was sepsis in 15% of patients in their group, local infection in 40% and 6 patients experienced massive hemorrhage from infection. Bolton et al.<sup>92</sup> consider the risk of infection in the cryopreserved vein high, especially when placed in the subcutis of the thigh. They believe that replacement of the infected ePTFE graft by the cryopreserved vein is indicated only if there is no other option available.

In another study published in 2002 further experiences with cryopreserved femoral vein or great saphenous vein harvested from the regularly examined organ transplant donors were discussed. The vein was used in 38 patients and the one year cumulative function was 68%. In this group of patients, no infection of the cryopreserved vein was seen, the aneurysmatic dilatation of the vein was found twice. The clinical signs of allograft rejection did not occur<sup>93</sup>. The potential of cryopreserved vein grafts were also evaluated in 2004 and 2005 by Madden et al.<sup>94, 95</sup>. In their first work they compared the outcomes of ePTFE grafts and cryopreserved veins in infection-free field. The graft patency was similar, cryopreserved veins were more resistant to infection but tended more to pseudoaneurysm creation. The study followed up with emphasis on stenotic and thrombotic complications since the authors expected the cryopreserved grafts to be more resistant in this respect than ePTFE grafts. The study was interrupted in 2003 when FDA (Food and Drug Administration) imposed security restrictions on tissue processing to the producer. The study showed similar outcomes in terms of stenotic and thrombotic complications in both groups but no infectious complications.

Since cryopreserved veins are more expensive they were not recommended for usage, irrespective of the FDA restriction.

Considering the expense of ePTFE grafts, formaldehyde-fixed arterial allografts began to be used<sup>96</sup>. Femoral arteries were harvested from cadaver organ transplant donors. Graft patency was comparable to other materials, the incidence of infectious complications was low – one patient out of 43, in a total of 68 grafts.

Data shown suggest that ePTFE prosthesis remains the only material for use. The cryopreserved graft appeared to be acceptable for complicated angioaccess in hemodialysed patients with infection complications. It is not clear from current literature findings if cryopreserved veins were further developed or utilized after the FDA restriction. Clinical importance of the cryopreserved veins is related only to single-centre experience.

## CONCLUSION

The arteriovenous graft infection has a significant impact on the morbidity and mortality in hemodialysed patients. A complex approach is required, including prevention, diagnosis and therapy. All measures to decrease the incidence of infection are beneficial and lower the costs of angioaccess care. Observations made during the last thirty years have brought nothing substantially new. The ePTFE prosthesis remains the most appropriate substitute. Prior to implantation, it is necessary to suppress all source of infection. Prosthesis implantation requires compliance with sterile safeguards. Diagnosis of infection is usually established according to clinical findings. The specification in accordance with PET CT examination seems to be a perspective. The principal of therapy remains unchanged. The surgical procedure is fundamental. Type of surgical procedure depends on experience. Apparently it is safer to remove all the infected prosthesis and provide access to dialysis using a central venous catheter. Functionality and length of use of prosthesis can also influence decisions. Prostheses that are repeatedly examined for stenotic and thrombotic complications with suspicion of infection should be removed. Simultaneous antibiotic therapy should correspondent to the results of the susceptibility tests.

## REFERENCES

- National Kidney Foundation: Kidney Disease Outcome Quality Initiative (NKF K/DOQI) clinical practice guidelines for vascular access. *Am J Kidney Dis* 2001; 37:137-181.
- Ethier J, Mendelssohn DC, Elder SJ, Hasegawa T, Akizawa T, Akiba T et al. Vascular access use and outcomes: an international perspective from the Dialysis Outcomes nad Practice Patterns Study. *Nephrol Dial Transplant* 2008; 23:3219-3226.
- Bachleda P et al. Cévní náhrady v chirurgii arteriovenózních spojek k hemodialýze. Olomouc: Prodos; 2001. p.12-16.
- Scher LA, Katzman HE. Alternative grafts materials for hemodialysis access. *Seminars in Vascular Surgery* 2004; 17:19-24.
- Berardinelli L. Grafts and graft materials as vascular substitutes for haemodialysis access construction. *Eur J Vasc Endovasc Surg* 2006; 32:203-211.
- Norton L, Eiseman B. Replacement of portal vein during pancreatectomy for carcinoma. *Surgery* 1975; 77: 280-294.
- Campbell CD, Brooks DH, Webster MW, Bahnson HT et al. The use of expanded microporous polytetrafluoroethylene for limb salvage: A preliminary report. *Surgery* 1976; 79: 485-496.
- Volder JGR, Kirkham RL, Kolff WJ. AV shunts created in new ways. *Trans Amer Soc Artif Intern Organs* 1973; 19:38-42.
- Baker LD, Johnson JM, Goldfarb D. Expanded polytetrafluoroethylene (PTFE) subcutaneous arteriovenous conduit: an improved vascular access for chronic hemodialysis. *Trans Amer Soc Artif Intern Organs* 1976; 22:382-387.
- Kaufman JL, Garb JL, Berman JA, Rhee SW, Norris MA, Friedmann P. A prospective comparison of two expanded polytetrafluoroethylene grafts for linear forearm hemodialysis access: does the manufacturer matter? *J Am Coll Surg* 1997; 185:74-79.
- Hurlbert SN, Mattos MA et al. Long-term patency rates, complications and cost-effectiveness of polytetrafluoroethylene (PTFE) grafts for hemodialysis access: a prospective study that compares Impra versus Gore-tex grafts. *Cardiovasc Surg* 1998; 6:652-656.
- Sorom AJ, Hughes CB, McCarthy JT, Jenson BM, Prieto M, Panneton JM et al. Prospective, randomized evaluation of a cuffed expanded polytetrafluoroethylene graft for hemodialysis vascular access. *Surgery* 2002; 132:135-140.
- Schild AF, Perez E, Gillaspie E, Seaver C, Livingstone J, Thibonnier A. Arteriovenous fistulae vs arteriovenous grafts: a retrospective review of 1 700 consecutive vascular access cases. *J Vasc Access* 2008; 9:231-235.
- Young EW, Dykstra DM, Goodkin DA, Mapes DL, Wolfe RA, Held PJ. Hemodialysis vascular access preferences at outcomes in the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Kidney International* 2002; 61:2266-2271.
- Anel RL, Yevzlin AS, Ivanovich P. Vascular access and patient outcomes in hemodialysis: questions answered in recent literature. *Artificial Organs* 2003; 27:237-241.
- Schwab SJ, Harrington JT, Singh A, Rohen J, Shohain SH, Perrone RD et al. Vascular access for hemodialysis. *Kidney Int* 1999; 55:2078-2090.
- Anel RL, Yevzlin AS, Ivanovich P. Vascular access and patient outcomes in hemodialysis: questions answered in recent literature. *Artificial Organs* 2003; 27:237-241.
- Sarnak MJ, Jaber BL. Mortality caused by sepsis in patients with end-stage renal disease compared with the general population. *Kidney Int* 2000; 58:1758-1764.
- Butterly DW, Schwab SJ. Dialysis access infection. *Curr Opin Nephrol Hypertens* 2000; 9:631-635.
- The United States Renal Data System 1999 annual data report. *Am J Kid Dis* 1999; 34: 168-176.
- Lafrance JP, Rahme E, Leloir J, Iqbal S. Vascular access related infections: definitions, incidence rates, and risk factors. *Am J Kidney Dis* 2008; 52:982-983.
- Taylor G, Gravel D, Johnston L, Embil J, Holton D, Paton S. Incidence of bloodstream infection in multicenter inception cohorts of hemodialysis patients. *Am J Infect Control* 2004; 32:155-160.
- Hoen B, Paul-Dauphin A, Hestin D, Kessler M. EPIBACDIAL: A multicenter prospective study of risk factors for bacteremia in chronic dialysis patients. *J Am Soc Nephrol* 1998; 9:869-876.
- US Renal Data System. *USRDS 2007 Annual Data Report*. The national institute of health, national institute of diabetes and digestive and kidney disease, Bethesda, MD, 2007.
- US Renal Data System. *USRDS 2006 Annual Data Report*. The national institute of health, national institute of diabetes and digestive and kidney disease, Bethesda, MD, 2007.
- Stevenson KB, Hannah EL, Lowder CA, Adcox MJ, Davidson RL, Mallea MC et al. Epidemiology of hemodialysis vascular access infections from longitudinal infection surveillance data: predicting the impact of NKF-DOQI clinical practice guidelines for vascular access. *Am J Kidney Dis* 2002; 39:549-555.
- Roberts TL, Obrador GT, St Peter WL, Pereira BJ, Collins AJ. Relationship among catheter insertions, vascular access infections, and anemia management in hemodialysis patients. *Kidney Int* 2004; 66:2429-2436.
- Hung KY, Tsai TJ, Yen CJ, Yen TS. Infection associated with double lumen catheterization for temporary hemodialysis: experience 168 cases. *Nephrol Dial Transplant* 1995; 10:247-251.
- Powe NR, Jaar B, Furth SL, Hermann J, Briggs W. Septicemia in dialysis patients: incidence, risk factors and prognosis. *Kidney Int* 1999; 55:1081-1090.
- Kairaitis LK, Gottlieb T. Outcome and complications of temporary haemodialysis catheters. *Nephrol Dial Transplant* 1999; 14:1710-1714.
- Mokrzycki MH, Zhang M, H, Cohen H, Golestaneh L, Laut JM, Rosenberg SO. Tunnelled haemodialysis catheter bacteraemia: risk factors for bacteraemia recurrence, infectious complications and mortality. *Nephrol Dial Transplant* 2006; 21:1024-1031.
- Zibari GB, Rohr MS, Landreneau MD, Bridges RM, DeVault GA, Petty FH, et al. Complications from permanent hemodialysis vascular access. *Surgery* 1988; 104: 681-686.
- Raju S. PTFE grafts for hemodialysis access. Techniques for insertion and management of complications. *Ann Surg* 1987; 206:666-673.

34. Zibari GB, Gadallah MR, Landreneau M, McMillan R, Bridges RM, Costley K et al. Preoperative vancomycin prophylaxis decreases incidence of postoperative hemodialysis vascular access infections. *Am J Kidney Dis* 1997; 30:343-348.
35. Padberg FT, Lee BC, Curl GR. Hemoaccess site infection. *Surg Gynecol Obstet* 1992; 174:103-108.
36. Minga TE, Flanagan KH, Allon M. Clinical consequences of infected arteriovenous grafts in hemodialysis patients. *Am J Kidney Dis* 2001; 38:975-978.
37. Schutte WP, Helmer SD, Salazar L, Smith JL. Surgical treatment of infected prosthesis dialysis arteriovenous grafts: total versus partial graft excision. *Am J Surg* 2007; 193:385-388.
38. Sexton DJ. Vascular access infections in patients undergoing dialysis with special emphasis on the role and treatment of *Staphylococcus aureus*. *Infect Dis Clin North Am* 2001; 15:731-742.
39. Foley RN, Guo H, Snyder JJ, Gilbertson DT, Collins AJ. Sepsis in the United States dialysis population, 1991 to 1999. *J Am Soc Nephrol* 2004; 15:1038-1045.
40. Taylor SM, Eaves GL, Weatherford DA, McAlhany JC Jr, Russell HE, Langan EM 3rd. Results and complications of arteriovenous access dialysis grafts of the lower extremity: a five year review. *Am Surg* 1996; 62:188-191.
41. Khadra MH, Dwyer AJ, Thompson JF. Advantages of polytetrafluoroethylene arteriovenous loops in the thigh for hemodialysis access. *Am J Surg* 1997; 173:280-283.
42. Kessler M, Hoen B, Mayeux D, Hestin D, Fontenaille C. Bacteremia in patients on chronic hemodialysis: a multicenter prospective survey. *Nephron* 1993; 64:95-100.
43. Allon M, Depner TA, Radeva M, Bailey J, Beddhu S, Butterly D et al. Impact of dialysis dose and membrane on infection-related hospitalisation and death: Results of the HEMO study. *J Am Soc Nephrol* 2003; 14:1863-1870.
44. Jaber BL. Bacterial infections in hemodialysis patients: Pathogenesis and prevention. *Kidney Int* 2005; 67:2508-2519.
45. Vanholder R, De Smet R, Glorieux G, Argilés A, Baurmeister U, Brunet P, Clark W, Cohen G et al. Review on uremic toxins: Classification, concentration, and interindividual variability. *Kidney Int* 2003; 63:1934-1943.
46. Girndt M, Sester U, Sester M, Kaul H, Köhler H. Impaired cellular immune function in patients with end-stage renal failure. *Nephrol Dial Transplant* 1999; 14:2807-2810.
47. Dhingra RK, Young EW, Hulbert-Shearon TE, Levey SF, Port FK. Type of vascular access and mortality in US hemodialysis patients. *Kidney Int* 2001; 60:1443-1451.
48. Miller PE, Carlton D, Dierehoi MH, Redden DT, Allon M. Natural history of arteriovenous grafts in hemodialysis patients. *Am J Kidney Dis* 2000; 36:68-74.
49. Kaplowitz LG, Comstock JA, Landwehr DM, Dalton HP, Mayhall CG. A prospective study of infections in hemodialysis patients: Patients hygiene and other risk factors for infection. *Infect Control Hosp Epidemiol* 1988; 9:534-541.
50. Nassar GM, Ayus JC. Infectious complications of the hemodialysis access. *Kidney Int* 2001; 60:1-13.
51. Curi MA, Pappas PJ, Silva MB Jr, Patel S, Padberg FT Jr, Jamil Z et al. Hemodialysis access: Influence of the human immunodeficiency virus on patency and infection rates. *J Vasc Surg* 1999; 29:608-616.
52. Bonomo RA, Rice D, Whallen C, Linn D, Eckstein E. Risk factors associated with permanent access-site infections in chronic hemodialysis patients. *Infect Control Hosp Epidemiol* 1997; 18:757-761.
53. Ryan SV, Calligaro KD, McAfee-Bennett S, Doerr KJ, Chang J, Dougherty MJ. Management of infected prosthetic dialysis arteriovenous grafts. *J Vasc Surg* 2004; 39:73-78.
54. Lentino JR, Baddour LM, Wray M, Wong ES, Yu VL. *Staphylococcus aureus* and other bacteremias in hemodialysis patients: antibiotic therapy and surgical removal of access site. *Infection* 2000; 28:355-360.
55. Wang SA, Levine RB, Carson LA, Arduino MJ, Killar T, Grillo FG, Pearson ML et al. An outbreak of gram-negative bacteremia in hemodialysis patients intraced to hemodialysis machine waste drain ports. *Infect Control Hosp Epidemiol* 1999; 20:746-751.
56. Engemann JJ, Friedman JY, Reed SD, Griffiths RI, Szczech LA, Kaye KS et al. Clinical outcomes and costs due to *Staphylococcus aureus* bacteremia among patients receiving long-term hemodialysis. *Infect Control Hosp Epidemiol* 2005; 26:534-539.
57. Troidle L, Eisen T, Pacelli L, Finkelstein F. Complications associated with the development of bacteremia with *Staphylococcus aureus*. *Remodial Int* 2007; 11:72-75.
58. Kleven RM, Morrison MA, Nadle J, Petit S, Gershman K, Ray S et al. Invasive methicillin-resistant *Staphylococcus aureus* infections in the United States. *JAMA* 2007; 298:763-771.
59. Shorr AF. Epidemiology of staphylococcal resistance. *Clin Infect Dis* 2007; 45:171-176.
60. Shurland S, Zhan M, Bradham DD, Roghmann MC. Comparison of mortality risk associated with bacteremia due to methicillin-resistant and methicillin-susceptible *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 2007; 28:273-279.
61. Sharpe JN, Shively EH, Polk HC. Clinical and economic outcomes of oral linezolid versus intravenous vancomycin in the treatment of MRSA-complicated lower extremity soft-tissue infections caused by methicillin-resistant *Staphylococcus aureus*. *Am J Surg* 2005; 189:425-428.
62. Berns JS. Infection with antimicrobial-resistant microorganisms in dialysis patients. *Semin Dial* 2003; 16:30-37.
63. Hadley AC, Karchmer TB, Russell GB, McBride DG, Freedman BI. The prevalence of resistant bacterial colonization in chronic hemodialysis patients. *Am J Nephrol* 2007; 27:352-359.
64. Barbosa D, Lima L, Silbert S, Sader H, Cendoroglo M, Draibe S et al. Evaluation of the prevalence and risk factors for colonization by vancomycin-resistant *Enterococcus* among patients on dialysis. *Am J Kidney Dis* 2004; 44:337-343.
65. National Kidney Foundation: KDOQI Clinical Practice Guidelines for Vascular Access. *Am J Kidney Dis* 2006; 48: 176-247.
66. Davidson IJA. PTFE bridge grafts. In: Davidson IJA, ed. *On call vascular access*. Austin: RG Landes. Company; 1996.
67. Almonacid PJ, Pallares EC, Rodriguez AQ, Valdes JS, Rueda Orgaz JA. Comparative study of use of Diastat versus standard wall PTFE grafts in upper arm hemodialysis access. *Ann Vasc Surg* 2000; 14:659-662.
68. Tokars J, Finelli L, Alter MJ, Arduino MJ. National surveillance of dialysis-associated diseases in the United States, 2001. *Semin Dial* 2004; 17:310-319.
69. Atta A, Eustace J, Song X, Perl TM, Scheel PJ Jr. Outpatient vancomycin use and vancomycin-resistant enterococcal colonization in maintenance dialysis patients. *Kidney Int* 2001; 59:718-724.
70. Falagas ME, Fragoulis KN, Bliziotis IA. Oral rifampicin for prevention of *S. aureus* carriage related infections in patients with renal failure - metaanalysis or randomized controlled trials. *Nephrol Dial Transplant* 2006; 21: 2536-2542.
71. Yu VL, Goetz A, Wagener M, Smith PB, Rihs JD, Hanchett J et al. *Staphylococcus aureus* nasal carriage and infection in patients on hemodialysis. Efficacy of antibiotic prophylaxis. *N Engl J Med* 1986; 315:91-96.
72. Kaupp HA, Matulewicz TJ, Lattimer GL, Kremen JE, Celani VJ. Graft infection or graft reaction? *Arch Surg* 1979; 114:1419-1426.
73. Bolton W, Cannon JA. Seroma formation associated with PTFE vascular grafts used as arteriovenous fistulae. *Dialysis and transplantation* 1981; 10:61-65.
74. Ahn SS, Machleder HI, Gupta R, Moore WS. Perigraft seroma: clinical, histologic, and serologic correlates. *Am J Surg* 1987; 154:173-178.
75. Bachleda P et al. Cévní náhrady v chirurgii arteriovenózních spojek k hemodialýze. Olomouc: Prodos; 2001. p.56-58.
76. Bachleda P et al. Cévní náhrady v chirurgii arteriovenózních spojek k hemodialýze. Olomouc: Prodos; 2001. p.78-81.
77. Schild AF, Simon S, Prieto J, Raines J. Single-center review of infections associated with 1574 consecutive access procedures. *Vasc Endovasc Surg* 2003; 37:27-31.
78. Palestro CJ, Vega A, Kim CK, Vallabhajosula S, Goldsmith SJ. Indium 111 labeled leukocyte scintigraphy in hemodialysis access-site infection. *J Nucl Med* 1990; 31:319-324.

79. Musso GC, Reynaldi J, Navarro M, Vallone M, Enz P, Vazquez P et al. Hidden clotted vascular access infection diagnosed by fluorodeoxyglucose positron emission tomography. *Nephrology* 2008; 13:2648-2655.
80. Lawrence PF, Dries DJ, Alazraki N, Albo D Jr. Indium-111-labeled leukocyte scanning for detection of prosthetic vascular graft infection. *J Vasc Surg* 1985; 2:165-173.
81. Bhat TJ, Tellis VA, Kohlberg WI, Driscoll B, Veith FJ. Management of sepsis involving ePTFE grafts for hemodialysis access. *Surgery* 1980; 87:445-450.
82. Taylor B, Sigley RD, May KJ. Fate of infected and eroded hemodialysis grafts and autogenous fistulas. *Am J Surg* 1993; 165:632-636.
83. Haug M. Der komplizierter av shunt infolge arterieller Probleme. *Angioarchiv* 1991; 22:15-17.
84. Humphries AL Jr, Nesbit RR Jr, Caruana RJ, Hutchins RS, Heimburger RA, Wray CH. Thirty-six recommendations for vascular access operation: lessons learned from our first thousands operations. *Am. Surg* 1981; 47:145-151.
85. Schanzer A, Ciaranello AL, Schanzer H. Brachial artery ligation with total graft excision is a safe and effective approach to prosthetic arteriovenous graft infection. *J Vasc Surg* 2008; 48:655-658.
86. Gifford RR. Management of tunnel infections of dialysis PTFE grafts. *J Vasc Surg* 1985; 2:854-858.
87. Calligaro KD, Veith FJ, Valladares JA, McKay J, Schindler N, Dougherty MJ. Prosthetic patch remnants to treat infected arterial grafts. *J Vasc Surg* 2000; 31:245-252.
88. Wu MY, Ko PJ, Hsieh HC, Chu JJ, Lin PJ, Liu YH. Repair of arteriotomy after removal of infected hemodialysis access by venous graft. *Chang Gung Med J* 2003; 26:911-918.
89. Raju S. PTFE grafts for hemodialysis access. Techniques for insertion and management of complications. *Ann Surg* 1987; 206:666-673.
90. Albers FJ. Clinical considerations in hemodialysis access infection. *Adv Ren Replace Ther* 1006; 3:208-217.
91. Matsura JH, Johansen KH, Rosenthal D, Clark MD, Clarke KA, Kirby LB. Cryopreserved femoral vein grafts for difficult hemodialysis access. *Ann Vasc Surg* 2000; 14:50-55.
92. Bolton WD, Cull DL, Taylor SM, Carsten CG 3rd, Snyder BA, Sullivan TM et al. The use of cryopreserved femoral vein grafts for hemodialysis access in patients at high risk for infection: a word of caution. *J Vasc Surg* 2002; 36:464-468.
93. Lin PH, Brinkman WT, Terramani TT, Lumsden AB. Management of infected hemodialysis access grafts using cryopreserved human vein allografts. *Am J Surg* 2002; 184:31-36.
94. Madden RL, Lipkowitz GS, Browne BJ, Kurbanov A. Experience with cryopreserved cadaveric femoral vein allografts used for hemodialysis access. *Ann Vasc Surg* 2004; 18:453-458.
95. Madden RL, Lipkowitz GS, Browne BJ, Kurbanov A. A comparison of cryopreserved vein allografts and prosthetic grafts for hemodialysis access. *Ann Vasc Surg* 2005; 19:686-691.
96. Liu Z, Zhu B, Wang X, Jing Y, Wang P, Wang S, Xu H. Clinical studies of hemodialysis access through formaldehyde-fixed arterial allografts. *Kidney International* 2007; 72:1249-1254.

