

# Exhausting Multiple Hemodialysis Access Failures

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## ABSTRACT

**Introduction:** Vascular access is often considered the **Achilles heel** of hemodialysis because of its impact on morbidity, all cause mortality and finally costs of these patients. The most common complication of permanent hemodialysis (HD) vascular access is thrombosis, with some cases being related to hypercoagulability states. Antiphospholipid antibody syndrome (APAS) is a cause of increased thrombotic tendency, and this may complicate the management of such patients on HD. **Case report:** We describe a 41-year-old woman with end stage renal disease (ESRD) from Adult Polycystic Kidney Disease who was referred to our tertiary care center for treatment and selection of renal replacement therapy form. It was thought to initiate with peritoneal dialysis considering her actual conditions. She was putted on hemodialysis for several sessions, and a subclavian catheter was her first vascular access. The surgeon created an arterio-venous fistula which did not mature. After the implantation of the peritoneal catheter she started peritoneal dialysis and continued living with that for 2 years. She felt exhausted and because of a grave peritonitis episode accompanied with procedure failure and a long hospitalization she was transferred to hemodialysis. Renal transplantation was not possible because she didn't have a kidney donation. She was maintained on regular HD, but her dialysis care was complicated by recurrent vascular access failures. She had multiple interventions for arterio-venous fistulas and grafts but almost all of them failed due to thrombosis to the extent that only one access site was available for her routine renal replacement treatment. A thorough thrombophilia screen confirmed the presence of antiphospholipid antibodies. A diagnosis of APAS was made and she was anticoagulated with warfarin. The AVG made in this last available site is still working from 18 months. If it fails we have no answers and solutions for her. **Conclusion:** The presence of APAS can complicate HD management by causing recurrent vascular access thrombosis and failure, and nephrologist must remain alert to this possibility. Checking and treating as soon as possible it's our future challenge.

**Key words:** hemodialysis, recurrent thrombosis, access failure, antiphospholipid antibody syndrome (APAS).

## 1. INTRODUCTION

The most common complication of permanent hemodialysis (HD) vascular access is thrombosis, accounting for 80 to 85 percent of arterio-venous (AV) access loss. Anatomic problems, mainly venous stenosis, are by far the major predisposing factors for thrombosis, being responsible for 80 to 85 percent of all cases (1, 2). Arterial stenoses and non-anatomic problems such as excessive post-dialysis fistula compression, hypotension and hypovolemia account for the remaining cases, with some cases being related to hypercoagulability states (3, 4, 5, 6).

In this case report, we describe a patient with the primary antiphospholipid antibody syndrome (APAS) complicated by recurrent AV fistula and vascular access thromboses. We outline her management and conclude by summarizing an approach to the care of such problematic cases.

## 2. CASE REPORT

A 41-year-old woman with end stage renal disease (ESRD) from ADPKD was referred to our tertiary care center for urgent renal replacement therapy. Considering

her actual state, the very long distance from hemodialysis centers and the possible opportunity for a future renal transplantation we concluded to start with peritoneal dialysis. Before starting PD we applied several sessions of hemodialysis using a subclavian temporary catheter. Two weeks after the peritoneal catheter implantation she started peritoneal dialysis. She continued on PD for two years but after a grave episode of peritonitis accompanied with a septic state, dialysis failure, long hospitalization, she was transferred urgently to hemodialysis. Her transplantation plans failed because her mother, the only possible donor died from a heart attack. She was maintained on regular HD, but her dialysis care was complicated by recurrent vascular access thrombosis. The first fistula functioned only 3 months and the second, third and fourth fistula were immature and not functioning. Both grafts didn't function and were clotted till the first days. During this time the catheters were her vascular access of necessity with all the difficulties and hazardous situations that they bring with them. The third graft that was performed in Turkey, was clotted till the first days then was done the thrombectomy and then clotted again. The

salvage procedures failed again. During this time the ongoing of hemodialysis was compromised from the recurrent septic states with seizures and bacteremia especially in the first hour of hemodialysis sessions. We usually used the catheter antibiotic locking in the end of hemodialysis with cephazolin or gentamicin but she gradually went on severe malnutrition due to long term infectious states and inefficient hemodialysis. She was supported with parenteral nutrition, more frequent HD sessions and intravenous antibiotics. This long calvary of suffering continued to the extent that only one access site was available for her routine renal replacement treatment.

A thorough thrombophilia screen confirmed the presence of antiphospholipid antibodies, while antinuclear antibody and anti ds-DNA antibodies were negative. A diagnosis of APAS was made and she was anticoagulated with warfarin. The surgeon created the left femoral graft. It was used only after 4 weeks with extreme precautions. From 1.5 years it is still working and functioning well. Now she has a better quality of life, better hemoglobin levels, URR and she is free of temperature, seizures and rigid catheters that were her nightmare. Now she is not anxious, but for how much time? What if this AVG doesn't function any more? What to do with her? A lot of answers?

### 3. DISCUSSION

APAS is a disorder characterized by arterial and venous thrombotic events associated with the presence of the so called antiphospholipid antibodies, which are auto-antibodies directed against phospholipids or phospholipids binding proteins. APAS can either be primary or secondary; in the latter case it is associated with other autoimmune diseases, particularly systemic lupus erythematosus (SLE), or related to acute infections or certain drug exposures. Four types of antiphospholipid antibodies have been characterized: anticardiolipin antibodies, antiprothrombin antibodies, anti-ethanolamine antibodies and anti-beta2-glycoprotein I antibodies. It is not clear whether the presence of antiphospholipid antibodies is involved in the pathogenesis of APAS or is an epiphenomenon. However, the presence of these antibodies is strongly associated with lupus anti-coagulant activity of serum and to the risk of arterial and venous thrombosis, spontaneous abortion and thrombocytopenia (7).

According to the Sapporo criteria for the diagnosis of the APAS, which are adopted by the American College of Rheumatology, the isolated detection of antiphospholipid antibodies in a patient's serum is not enough to make the diagnosis of APAS (8). For a diagnosis of APAS to be made, the patient must have at least one clinical criterion and one laboratory criterion for APAS. Clinical criteria include one or more confirmed episodes of vascular thrombosis, or three un-explained consecutive abortions, or an unexplained late pregnancy loss, or a premature birth due to preeclampsia or placental insufficiency. Laboratory criteria include the detection of anicardiolipin antibodies in high titer or lupus anticoagulant activity in the patient's serum on at least two occasions, six weeks apart (8).

Renal complications occur in as many as 25% of patients with the APAS (9). For the most part, these complications are directly related to thrombotic occlusions of glomeru-

lar capillaries; however, they occasionally involve the renal veins and renal arteries. Clinical manifestations include acute and subacute renal failure, active urinary sediment, the nephrotic syndrome and hypertension (9, 10). Pathological entities include thrombotic microangiopathy, focal segmental glomerulosclerosis, ischaemic interstitial nephritis with fibrosis and renal infarction (9, 10). In addition, the presence of antiphospholipid antibodies in patients with ESRD often complicates their management. The presence of the antiphospholipid antibodies doubles the frequency of thrombotic occlusion of vascular access sites in HD patients (4).

The development of these antibodies may be related to the type of HD access used, since they are more frequent in patients with AV grafts compared to patients with AV fistula. In a cross-sectional study that evaluated HD patients at a single dialysis facility, 22% of patients with AV grafts had a raised titer of anticardiolipin antibodies, while only 6% of patients with AV fistula had similarly raised titers (5). However, another likely explanation for the higher incidence of antiphospholipid antibodies in patients with AV grafts is that patients who already have the antiphospholipid antibodies are more likely to suffer from repeated vascular access failure and are more likely to require the placement of a synthetic AV graft. The development of antiphospholipid antibodies may also be related to the type of dialysis membrane used, with a greater incidence being associated with frequent use of cuprophane membranes (68% versus 34%,  $P<0.05$ ) (6).

Vascular thrombosis is usually recurrent in APAS and all patients who have major or recurrent thrombotic event should receive life-long warfarin therapy with a target INR of 3.0 or higher (11, 12). In a retrospective study of 147 patients with the APAS, recurrent thrombosis affected 30% of patients per year in those who did not receive long-term anticoagulation therapy. Aspirin alone was of no benefit, while low intensity warfarin (INR<3) with or without low dose aspirin offered only modest protection against recurrent thrombosis (23% of patients per year). High intensity dose warfarin (INR>3) with or without low dose aspirin markedly reduced the incidence of recurrent thrombosis (1.3% of patients per year) (11). Treatment with warfarin may also be successful in increasing AV graft survival in HD patients with elevated anticardiolipin levels and a history of vascular access failure (11, 12).

### 4. CONCLUSION

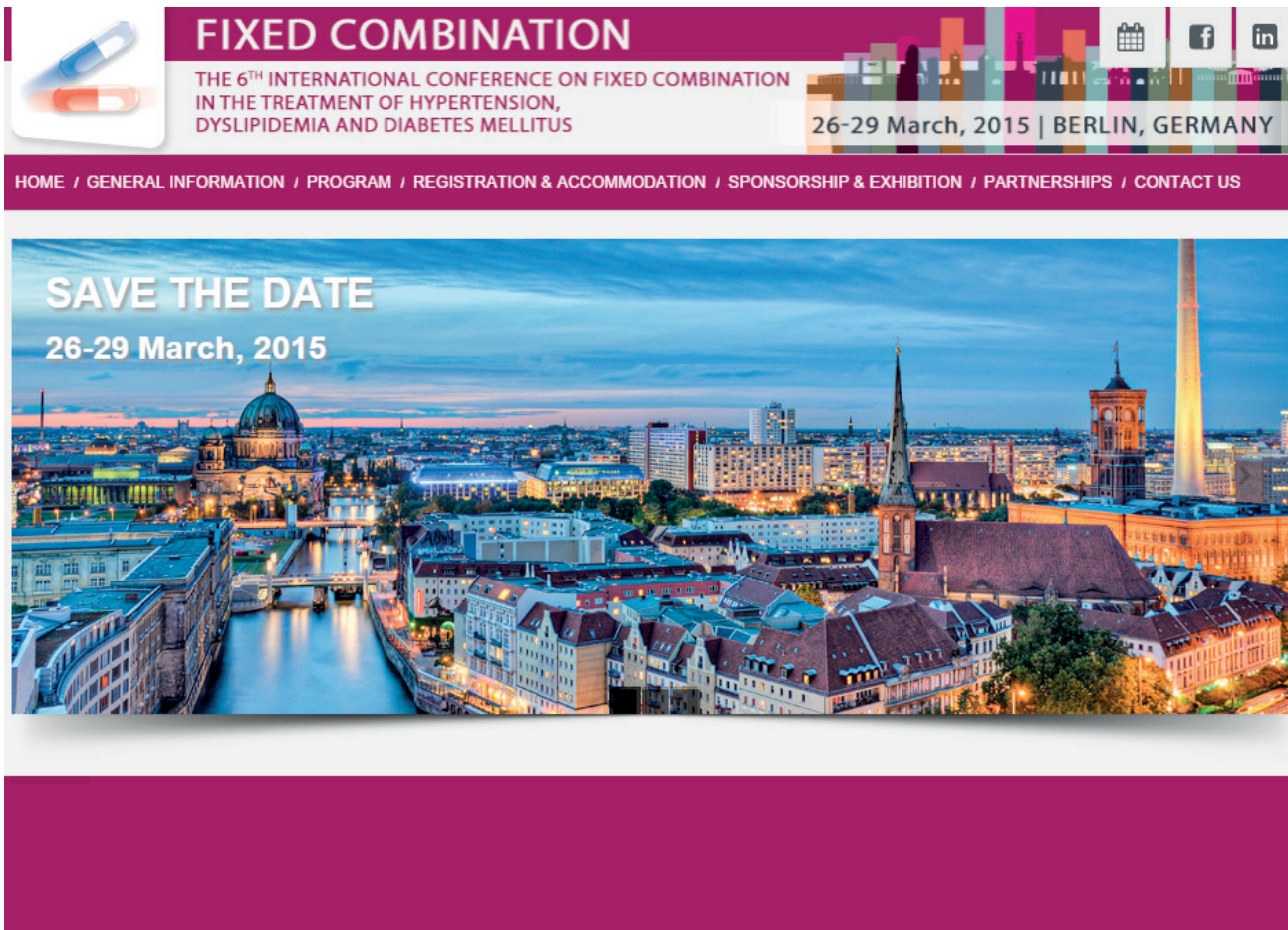
Nephrologists must remain alert to the possibility of APAS being the cause of repeated vascular access thrombosis and failure in HD patients. Checking and treating as soon as possible remains our challenge in these cases. Anticoagulation management of these patients must be handled with extra care to avoid bleeding complications.

**CONFLICT OF INTEREST: NONE DECLARED.**

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