Case Report

Central venous catheter related Superior Vena Cava Syndrome after renal transplantation in two cases

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Abstract

Vena cava superior syndrome is a specific clinical disease; very important obstruction of the large central veins. Catheter-related deep vein thrombosis (DVT) and vena cava superior (VCS) syndrome are rare but serious complications that require specific care in these selected renal transplant recipients. Thrombosis formation, is a major complication of central venous catheter (CVC) attempts, however, arterial puncture, hematoma, and pneumothorax are more common complications. Mechanical complications of (CVC) have been reported in 5% to 19%. Incidences of CVC thrombosis have been reported in up to 66% of patients in venographic studies, however; VCS syndrome is a rare complication. After evaluating the catheter-related thrombosis risk, hemodialysis catheters were found to be the highest risk in comparison with other catheters. Catheter-related DVT and VCS syndrome are rare but serious complications require specific care in these renal transplant recipients. We report two cases of vena cava superior syndrome due to CVC thrombosis after kidney transplantation in two chronic renal disease patients with histories of previous catheter thrombosis.

Keywords: end stage renal disease, vena cava superior syndrome, central venous catheter, catheter related deep vein thrombosis

1. Introduction

Catheter-related deep vein thrombosis (DVT) and vena cava superior (VCS) syndrome are rare but serious complications that require specific care in these selected renal transplant recipients. Catheters with large diameters increase the risk of thrombosis because blood flow is obstructed. The coagulation pathway has started, and permanent inflammation due to the existence of a catheter can cause intimal hyperplasia. The catheter’s tip being parallel to the longitudinal axis of the VCS’ course, avoidance of the direct contact with the vessel’s wall are major factors in preventing the development of thrombus. In addition, central venous catheter (CVC) complications are more frequent in cases of chronic kidney failure, multi-organ failure, and sepsis, which lead to a compromised immune system.

We report two cases of vena cava superior syndrome due to CVC thrombosis after kidney transplantation in two chronic renal disease patients with histories of previous catheter thrombosis.

2. Case Report

2.1. Case 1

A 41-year-old female patient diagnosed with end stage renal disease (ESRD) with an unknown etiology received a cadaveric kidney transplantation (KT). She had a history of thrombosis related with the hemodialysis (HD) access both in the left brachial arteriovenous fistula (AVF) and the HD catheter during an eight years chronic HD program. We performed right radial arterial cannulation and right internal jugular venous catheter placement intra-operatively. The patient received induction immune-suppressant treatment with anti-thymocyte globulin (ATG) Fresenius for three days. Swelling of the right arm developed on postoperative day four, and Doppler ultrasound (US) revealed thrombosis of the right axillary and distal brachial veins. Thrombi were detected by magnetic resonance angiography (MRA) in both the intersection of the right brachiocephalic vein and VCS and in the brain. A simultaneous ventilation/perfusion scintigraphy (V/Q Scan) revealed resorbing acute emboli in the lungs. Excluding the presence of Heterozygote MTHFR A1298C, Heterozygote F5 Leiden, and Heterozygote PAI-1 mutation, all other thrombus panel tests were normal. Anticoagulation was performed by low molecular weight heparin enoxaparin sodium. On postoperative day six, the patient’s creatinine erose to 3.1 mg/dl, and urine output decreased. The renal Doppler US was consistent with acute rejection, and the allograft biopsy was postponed because the patient received anticoagulation and plasmapheresis treatment for presumed antibody rejection. On postoperative day thirteen, the Control Doppler US revealed no thrombi in the upper extremity and

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jugular veins. The delayed allograft biopsy showed healing acute antibody mediated rejection. On postoperative day thirty-two, the patient was discharged after normalization of the kidney functions.

2.2. Case 2
A 41-year-old male patient diagnosed with ESRD with an unknown etiology received a cadaveric kidney transplantation (KT). And he received HD treatment for twelve months. Intra-operatively right radial arterial cannulation and a right internal jugular venous catheter placement were performed. The patient received induction immunosuppressant treatment with ATG Fresenius for three days. The rest of his hospital stay was uneventful, and the patient was discharged on postoperative day six with three immunosuppressant medications, including tacrolimus (TRL), mycophenolate mofetil (MMF), and a steroid. He was readmitted to the ward for swelling and erythema of the neck on postoperative day ten. The right jugular vein was completely thrombosed, and it was accompanied by a widening of the superficial veins and the development of collateral veins. An upper extremity MR revealed similar results. Anticoagulant treatment with low molecular weight heparin (enoxaparin) was initiated. All blood tests regarding thrombi and the V/Q scan of the lungs were negative. The rest of his hospital stay was uneventful; he was discharged with a prescription for warfarin.

3. Discussion
Mechanical complications of CVC have been reported in 5% to 19% of patients who receive central venous catheters. Thrombosis formation is decidedly a major complication of CVC insertions; however, arterial puncture, hematoma, and pneumothorax are more common complications (1). Incidences of CVC thrombosis have been reported in up to 66% of patients in studies of venography however, VCS syndrome is a rare complication (2). It has also been reported that patients have tolerated subclavian catheters better than left internal jugular catheters. The risk of thrombi development is high in the left internal jugular vein than the right internal jugular vein, because it is related with two strong angulations of the catheter that are required to reach to right atrium. In addition, the risk of thrombus development is higher in the subclavian vein than the right internal jugular vein (42% vs 10%) (3). In a study reporting 2128 CVC days internal jugular vein catheterization was determined to carry the highest risk of thrombosis development, followed by the femoral (36%), upper limb (27%), and subclavian (9%) routes. After evaluating the catheter-related risk of developing thrombosis, hemodialysis catheters were found to present the highest risk in comparison with other catheters (4).

The physiological period of ESRD patients differ from the healthy population. Their serum creatinine level is generally higher and their hematocrit level, platelet production and properties (adhesion, aggregation are generally decreased), and life cycles are under the normal activity and because of the uremia (5,6). Therefore; the uremia, anemia, and thrombocytopenia in this group of patients prevent thrombus formation and increase bleeding risk compared with the healthy population.

Living-donor renal transplantation procedures are more successful than cadaveric kidney transplantation, because cadaveric kidney takes longer to reach to a normal value of suitable renal function and serum creatinine level. This delay diminishes the risk of thrombus formation. Post-operative heparin administration should only be used in hypercoagulable manifestations suggested by some author, such as collagen vascular disease, deep venous thrombosis history, anti-thrombin 3 (AT3) deficit, diabetes, multiple miscarriages, protein S deficit, or protein C resistance (7,8). In thrombophilia cases, observe to clarify the anti-coagulant therapy is more beneficial than standard anti-coagulant therapy. One of the major complications resulting from graft loss is renal allograft vascular thrombosis. The rate of thrombosis varies between 0.5% and 6% (9). Researchers suggest the use of anti-coagulant therapies to reduce the risk of complication in all renal transplant recipients. Low molecular weight heparin (LMWH), per-oral anti-coagulants (aspirin, warfarin), heparin have been used together or separately in the anti-coagulant therapy. Some studies have suggested that LMWH reduces the possibility of post-operative thrombosis and does not raise the surgical bleeding risk (7,10). According to some author, prophylactic heparinization can cause increased morbidity in kidney transplant patients. Mohan et al proved that intra operative heparin administration increases the post-operative transplant demand and does not decrease the frequency of graft thrombosis. (11) Likewise, another study defined the increase the risk of bleeding caused by LMWH and aspirin in used together in the early post-op. period (12). Unlike; The risk of vascular thrombosis increased by heparin induced thrombocytopenia (HIT). HIT caused by an immunological reaction and the patients using heparin have an up to 5% increased risk of vascular thrombosis caused by HIT (13).

We do not administer heparin routinely after renal transplantation except for patients with risk factors. In patients diagnosed with the ESRD, fibrinogen and inflammatory markers were elevated and these are the risk factors for the development of thrombosis. In other epidemiologic studies, hemodialysis and kidney transplantation were suggested to increase the risk for thrombosis. Cessation of prophylactic anticoagulant treatment ten days prior to surgery to decrease peri- and post-operative bleeding might increase the risk of thrombosis beginning on the day of cessation of the treatment. Anticoagulation treatment should be initiated in the early postoperative period.

Development of VCS syndrome due to CVC placement is related to multiple factors. Recurrent catheter placement
attempts and leaving the catheter catheters remaining in the same site for long durations lead to the development of recurring endothelial injury and chronic inflammation, which result in the development of thrombus and injury to the vein wall.

VCS syndrome is rarely caused by CVC-related thrombosis. Malignant tumors are significant causes of VCS syndrome. Physical examinations are the simplest way to detect thrombus or VCS syndrome in patients with a history of VCS placement. The venous blood flow resistance rises as the SVC syndrome becomes compressed, and the symptoms and signs occur. The most common signs and symptoms are neck and facial swelling, hoarseness, headache, dyspnea, pain, and other symptoms (14).

Mean treatment options for VCS syndrome include treatment of the original malignancy, anti-coagulation, endovascular standing, and surgery in patients diagnosed with VCS syndrome (14). The first signs observed during physical examinations in our study were swelling and erythematic on the right side of the chest and face. Ultrasound (US) revealed increased vein diameter, cessation of the blood flow, and images compatible with thrombi. Computerized tomography (CT) is a valuable tool for the evaluation of the compressing masses on the central veins. Although Doppler US has a sensitivity of 94% and a specificity of 96% for the recognition of the thrombi, magnetic resonance (MR) performed with contrast media is the most valuable tool in detecting thrombi development. Thrombolysis and heparinization are the suggested treatment modalities for catheter-related thrombus. Initiating the treatment immediately after the development of symptoms may result in a success rate of up to 70%. When the treatment is delayed, the success rate decreases, and the treatment is considered ineffective after ten days.

In conclusion, catheter-related DVT and VCS syndrome are rare but serious complications that require specific care in these selected renal transplant recipient. That minimizing the duration of the procedure can potentially cause complications, so an experienced physician is recommended to perform the procedure. Since thrombus development has a multi-factorial etiology, careful risk assessment and individualized treatment strategies are required for minimization of complications.

Conflict of interest
All authors declare no conflict of interest regarding this manuscript

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References