Journal Of Harmonized Research (JOHR)

Journal Of Harmonized Research in Medical & Health Sci.
7(2), 2020, 07-10

ISSN 2395 – 6046

Short Communication

ATRIAL FIBRILATION MIMICKING ACUTE RENAL REJECTION

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Abstract: Purpose: Atrial fibrillation in renal transplant patients is a cardiac pathology that should be treated. A year ago, the patient underwent cadaveric transplantation. The patient was discharged normally. During the 1-year follow-up period no rejection was observed. However, the patient was hospitalized because of the 3.3 mg / dL creatinine value in the first follow-up year

Material and method: Pulse steroid therapy was initiated with pre-diagnosis of rejection. Beta blocker therapy, due to the high value of creatinine, started low dose because the patient had ECG in atrial fibrillation. On the second day of the treatment, the patient requested Dopler USG. It was stated that the residual index (RI) value was slightly increased when the posterior artery and vein were open. In the meantime, due to the lack of urine output and the increased potassium value of the patient, nephrology has planned dialysis treatment. But the patient did not accept the dialysis treatment. The patient was scheduled for a biopsy on the third day of the pulse steroid, assuming no response to treatment. Cardiac enzymes, ECG and cardiology consultation were requested on the patient with severe respiratory distress and retrosternal pain due to increase in volume load and exacerbation of atrial fibrillation. It is recommended that chronic atrial fibrillation needed full-dose Beta blockers in the acute phase if there was not an obstacle

Findings: Treatment of cardiology has begun. Effective therapy resulted in patient fibrillation to normal rhythm. On the next day, the patients creatinine level reached 1.4 and the urine output increased to normal level. The patient was discharged after 1 week.

Conclusion(s): It should be remembered that patients with atrial fibrillation may have increased creatinine levels due to hypoperfusion. Early and effective treatment is important**.**

Keywords: Atrial Fibrilation, Rejection, Renal Transplantation

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Received on: December 2019
Accepted after revision: April 2020
DOI: 10.30876/JOHR.7.2.2020.07-10

Introduction: Heart pumps blood all over the body, and firstly the atria of the heart and secondly the ventricles contract. The electrical system in the heart causes this contraction. Electrical stimulation begins at the aright atrium in the sinoatrial node (SA node), and then the electrical signal travels to the atrioventricular

(AV) node. Through this electrical conduction, the atria and the ventricles contract, and blood is pumped throughput the body [1].

Sinus rhythm occurs when the electrical conduction of the heart begins under the SA node control. Then, the heart beats regularly at a rate of 60 to 100 per minute [2].

AF is the most common heart rhythm in which the atrium is irregularly reflected in electrocardiography (EKG). The SA node cannot control the electrical signal, many different warnings prevent the contraction of the atria, and blood in the atria cannot flow into the ventricles. As the electrical signal is abnormal, many signals travels through the atria and are controlled by the AV node. The AV node reduces the number of electrical signals by transmitting signals into the ventricles, which is a very quick process. The number of signals transmitted in the atria can range from 300 to 600 per minute. In this way, the heart beats regularly at a rate of 60 to 100 per minute [3].

Myocardial insufficiency might occur because of cardiomyopathy, prolapse and stenosis in the aortic valve, pericarditis, open heart surgeries, chronic lung disease, pulmonary embolism, AF with irregular and rapid heart rhythm, renal insufficiency and elevated serum creatinine levels [4].

Patients with AF might experience tachycardia, respiratory disorder, pressure in the chest area, pain, activity intolerance, fear of death and anxiety. AF might decrease the contractile power of the heart, and heart failure might occur. Thrombus which can occur as a consequence of AF might negatively affect brain, lung, heart and renal veins [5-6].

For the treatment of AF, antiarrhythmic drugs such as amiodarone, dofetilide, propafenone, procainamide and disopyramide; drugs to reduce and regulate the rate of ventricles such as digoxin, beta blockers, verapamil, diltiazem and calcium channel blockers; and anticoagulant drugs to prevent any potential embolism like warfarin are used. Cardioversion can also be performed [5-7].

For the treatment of AF, pulmonary vein ablation or AV node ablation can also be performed along with a cardiac pacemaker. If these methods are not effective, surgical treatment can be preferred [6].

In this article, the experiences of our kidney transplant patient monitored by our organ transplant service and diagnosed with AF findings four years after the transplant surgery are analysed.

A possible relationship between the activation of the renin-angiotensin-aldosterone system (RAAS) and AF is possible. It is known that there is a role of the RAAS in the pathogenesis of AF. Angiotensin II can increase atrial pressure, lead to atrial fibrous and modulate ion channels, all of which play a role in the structural and electrical change of the atrium, resulting in AF. In addition, polymorphism in genes increases the susceptibility to AF. In renal failure, it might lead to oxidative stress and impaired intracellular calcium homeostasis, hyperkalaemia and elevates serum creatinine and atrial stretch and dilatation [8]. AF can also be seen in transplant rejections [9].

Case report: The patient is a 38-year-old woman. She was diagnosed with essential hypertension, renal failure and had a kidney transplant after haemodialysis. The patient was discharged with normal vital findings after the transplant surgery. Transplant rejection was treated through an immunosuppressive therapy.

The patient who had a kidney transplant because of renal failure and essential hypertension was hospitalised with complaints of weakness and pain. The serum creatinine of the patient who had a transplant surgery a year ago was found to be 2.8 mg/dl. The findings of her physical examination are: TA: 140/90 mmHg; her conjunctival respiration was normal; in cardiovascular evaluation, S1 (+) and S2 (+), but the rhythm was 110 per minute; there was no distension pain in the abdomen; graft sensitivity (+), pain (-) and sensitivity (-). The size of the radiodiagnostic right pelvic localised transplant kidney was 116x54 mm,

and the renal contour was uniform. The parenchyma thickness and the echogenicity were found to be normal. The pelvicalyceal ectasia in the transplanted kidney was not monitored, and there was no solid or cystic space occupying lesion. The systole was 70 cm/second, while the diastole was 6 cm/second. There was no evidence of stenosis in the renal artery. The renal vein was openly monitored. The renal parenchymal index values were found to be 0,88, which was higher than the normal values. It was reported that rejection might have occurred.

The patient had 150 mg of ecopirin (coraspin). The prothrombin time, prothrombin activity and apt times were normal. The INR was high (it was 4, whereas the normal value is 1-1,14). Vitamin K was given to the patient in order to prevent any potential bleeding. The patient was also given two fresh frozen plasmas. There was no evidence of bleeding. The blood glucose, pancreatic and liver enzymes, total protein and albumin levels were normal. Liquid and electrolyte balance was normal. There was no glucose, protein or albumin in the urine. The erythrocyte, leukocyte, platelet values were normal, there was no decrease. The tacrolimus blood level was normal. The patient was had been monitored a year ago when the creatinine was 2 mg/dl. Then, the patient had been discharged when the creatinine levels had been normal. In our last analysis, the patient was found to have pain in the graft area. The increased creatinine levels suggested rejection (BUN = 79,90, creatinine=3.3); however, biopsy could not be performed due to high INR. The pulse rate of the patient was 110 per minute, and the renal vein was open. In the cardiologist consultation, the patient was diagnosed with AF after the electrocardiogram. And transesophageal echocardiography of patient showed no thrombus. Beloc was recommended for the treatment of AF.

For the treatment of the rejection, 1000 mg Prednol (cortisone) pulse therapy was started. Then, 200 mg ATG (immunosuppressive) was used. Later, as the patient did not respond to the Beloc treatment, a cardiology consultation was conducted again and the patient was given a Cordorone (amiodarone) infusion. The patient was cardioversion and given digoxin 5 mg and Beloc 50 mg for the treatment of AF. Coraspin treatment was continued. Hemodynamic follow-up, heart rhythm monitoring and oxygen saturation were performed for the patient whose ejection/fraction was normal and who had no fluid loan in the lungs. The tissue perfusion, consciousness and peripheral nutrition of the patient were monitored by the nurses. The daily life activities and skin integrity of the patient were maintained, and the blood gases, hemogram, laboratory results, bowel sounds and distension status of the patient were also monitored and recorded by the nurses. The laboratory values, hemodynamic and respiratory results, signs of infection, findings of the cardiac and renal system, data of the lungs and the liver were evaluated by the surgeons and the nurses. Certican 2x0.75 mg immunosuppressive, prolonging the life of the new kidney, was started. The clinical condition of the patient improved after six days. The patient was discharged when the clinical findings were found out to be normal. Information regarding what should be done at home, infection control, how to use the medication, AF findings and other necessary explanations were repeated through the training brochure by the nurses. Any potential rejection that might occur and the blood values, drug use, AF findings and signs of infection of the patient were continued to be monitored.

Discussion: Atrial Fibrillation (AF) is a cardiac pathology that must be treated in renal transplant patients. This report, in this respect, gives information about the treatment of the patient and the provision of care in the hospital. The AF that triggered AF in the patient might have been caused by a history of hypertension, increased creatinine level after the transplant, calcium in the kidneys of the patient, impaired potassium homeostasis, atrial stretch and

dilatation [7]. Early intervention for changes in vital findings after transplant, management of cardiological findings like AF, maintenance of fluid support, simultaneous immunosuppressive and cortisone treatment might prevent transplant rejection. Therefore, monitoring and diagnosing any negative situation after organ transplant as a team are significant and vital [10].

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