

Cardiorenal syndrome (chronic tip2) in decompensated cardiomyopathy: diuretics resistens and sensitive to ultrafiltration-case report

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Cardiorenal syndrome is a disorder of the heart or kidneys where acute or chronic dysfunction of one organ may induce acute or chronic dysfunction of the other consecutive. In cardiorenal syndrome (chronic tip 2) long-term abnormalities in cardiac function leads to decreased renal function.

However, high doses or excessive use of diuretics is associated with worsening renal function, at least in part by activating tubuloglomerular feedback and neurohormonal activation. Diuretic resistance is a hallmark of advanced heart failure and cardiorenal syndrome.

A 45-year-old patient with confirmed ischemic cardiomyopathy (coronary artery disease -prior re- myocardial infarction ; stent implanted LAD ,coronary artery bypass and circular ventrikuloplastic and mitral reconstruction) was admitted with a decompensated heart failure. He has one prior episode of cardiac decompensation. He is Diabetic, insulin-dependend. Two weeks before as a ambulatory patient he was put on high doses of diuretics(furosemide 750mg), ACE inhibitors, beta blockers, spironolotone.

Patient presented dyspnea, peripheral edema and ascites in the abdomen. Signs of elevated jugular venous pressure were presented as well. He was NYCHA class IV. Serum levels of creatinine and potassium at admission were similar to baseline. GFR was decreased. Echocardiography revealed global severe systolic dysfunction with 24% ejection fraction and moderate mitral and tricuspid regurgitation. Ultrasound of kidneys showed two kidneys with preserved shape, size and no signs of slowdown and calculosis. The patient was switch to intravenous diuretic therapy within 6 days. The lack of the effect of the diuretic stimulation, has led to change the therapy - dopamine stimulation(renal dose). In the period that followed the patient has presented an increased level of degradational products, has become olygo/anuric, due to which he has developed a diuretic resistance. The patient was referred and started with ultrafiltration. The ultrafiltration program was 3 times per week.

The two years follow-up show that the patient's condition was stable and he has no readmissions at the hospital for cardiac decompensation for the following period. He has returned to his "normal" routine prior to onset of illness.

Patients diagnosed for CRS should be immediately treat with ultrafiltration along with diuretics as an add-on therapy.

Ultrafiltration therapy can efficiently corrects volume overload in the acute and chronic conditions, improve cardiac function and quality od life and provide long-lasting benefits such as reduced HF-related readmissions.

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