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ventricular myocardium. Consequently, dopamine and α-adrenergic receptors are not responsible for the decrease in strength of contractility of the ventricular myocardium.

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P128-T | Endovascular revascularization of occlusion renal artery in a patient with chronic kidney disease

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Background: The problem of revascularization of renal arteries in chronic kidney disease remains unsolved. We present the case of endovascular revascularization of renal arteries with positive dynamics and effect.

Materials and methods: Revealing renal arteries stenosis and performed endovascular stenting with the evaluation of the function in 6 months.

Results and discussion: Patient M, 57 years old, entered to the hospital with weakness, dizziness, arterial hypertension. Glomerular filtration rate (GFR) was 28 mL/min/1.73 m^2 (CKD-EPI). The daily blood pressure was 185/110 mmHg. Complete occlusion of the right renal artery and the proximal segment of the left renal artery to 85% were revealed by Doppler ultrasonography. Angiography of renal arteries with stenting occlusion of the right renal artery was performed. Stent in the left artery was emplaced in 3 months.

In 6 months after these procedures patient reported improvement in his general condition, blood pressure fixed at 140/80 mmHg. We received stabilization of renal function: serum creatinine concentration of the blood dropped to 67·8 mmol/L, GFR increased from baseline – 86 mL/min/1·73 m^2. The average systolic blood pressure fell by 32·1%, diastolic blood pressure decreased by 27·3% from baseline (75 mmHg). According to dynamic nephroscintigraphy, stabilization of renal function was with preservation of the perfusion volume of the right kidney, without significant increase in function, with improvement of left kidney function. The total GFR was 55·56 mL/min (left – 46·5 mL/min, right – 9.06 mL/min).

Conclusion: This case shows that percutaneous endovascular interventions can be effective and safe as a method of revascularization in patients with renal artery occlusions.

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P035-F | Ventricular or atrial epicardial fat secretome can be regulated by acetylcholine: new preclinical models on autonomic dysfunction

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Background: Atrial fibrillation (AF) is one of the most sustained arrhythmias. Recent studies suggest that the local amount of epicardial adipose tissue (EAT) around the atria is more associated with AF burden than obesity, ageing or left atria dimensions. Several authors have paid attention on EAT-released proteins as mediators of AF substrate. Besides, EAT shelters ganglionated plexuses and cholinergic and adrenergic nerves. The principal cholinergic neurotransmitter is acetylcholine (ACh), which acts reducing the action potential duration. Some authors select EATv for studying EAT contribution to AF while others suggest the knowledge of the adipose tissue closer to the disorder.

Purpose: We wanted to compare the secretome between peri-atrial EAT (EATa) and peri-ventricular EAT (EATv) and its differential regulation by acetylcholine (ACh).

Material and methods: EATa and EATv from 11 patients underwent open heart surgery were splitted in 100 mg pieces and cultured. After 24 hours washing, tissue proteins were separated by 2-Dimensional electrophoresis. Secretome proteins were separated by SDS-polyacrylamide electrophoresis gel, quantified by an analysis software and identified by mass spectrometry. Muscarinic receptor type's expression was analyzed by real time polymerase chain reaction or western blot. Then ACh and acetylcholinesterase (AChE) activities were determined by colorimetric assays.

Results: Our results showed high similarities between EATa and EATv regarding to their protein and secretome profiles. Thus, 282 common proteins were identified in both tissues. EATa and EATv contained muscarinic receptor type 3 (mAChR 3), which is increased in adipogenesis-induced cells. Despite AChE activity was higher in EATa (128 [17–543 mU/mg tissue]) than in EATv (43 [8–142 mU/mg tissue]); $P < 0{\cdot}05$, both tissues modified their released protein profile after ACh treatment.

Conclusions: The similarity between the released proteins from EATa and EATv and its regulation by ACh makes them an appropriate preclinical model to clarify the interplay among EAT, AF and autonomic dysfunction.