INTRODUCTION AND AIMS: In patients with CKD (chronic kidney disease) it is not sufficiently clarify the questions regarding the role of thrombogenesis in CVD (cardiovascular diseases) occurrence and progression of chronic kidney disease directly. It is known that the activation of intravascular thrombus formation leads to the formation and progression of a significant number of pathological processes, primarily, CVD. Platelets are important unit of thrombus formation process and plays a central role in the pathophysiology of arterial thrombosis and atherosclerotic vascular lesions. Considering stated above, it is important to study thrombosis unit of hemostasis in patients with CKD of different stages, detection mechanisms for destabilization and finding effective ways of correction, to reduce the risk of both thrombosis and atherogenesis. Aim: we evaluated intravascular platelet activation and its change against the background of L-arginine treatment in patients with stage III CKD (GFR 59-30 ml/min/1.73 m²).

METHODS: 12 pts (7 female and 6 men) with CKD stage 3 (GFR 50.4 (9.6) ± 3.1 ml/min/1.73 m²) in age from 20 to 56 years (age 54.7 (8.7) ± 2.3 years) and 3 pts (2 female and 1 man) with CKD stage 1 (GFR 112.4 (17.1) ± 5.6 ml/min/1.73 m²) - control group. At all pts the level of creatinine was determined in blood by ELISA. GFR was determined by CKD EPI. The morphological and functional status of platelets was estimated by transmission electronic microscope (PBM-100-01 "SLEMI", Ukraine). All patients received solution L-arginine 4.2% - 100 ml once a day in a complex of basic nephroprotection therapy within 2 weeks.

RESULTS: In the patients with CKD stage 3 showed a significant damage of thrombocyte hemostasis due to significant growth in the content of the activated, degranulated and aggregated forms of platelets and development of hyperactivation of platelets by hyaline type in comparison with the parameters of the control group (figure 1,2). Inclusion of L-arginine in the treatment leads to a reduction of pathomorphological changes of platelets, limitation of their hyperactivity in pts with CKD stage 3. The level of non-activated platelets increased by 41.9% (p<0.034) compared with the baseline, activated platelets decreased by 35.7% (p=0.05).

CONCLUSIONS: This study demonstrated an improvement in intravascular platelet activation in patients with Stage 3 CKD when supplementing standard L-arginine therapy. Its administration improves the functional state of platelets and can have a positive effect on endothelial function and the level of arterial thrombosis.