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DIFERENCES BETWEEN HEMATOLOGY AND BIOCHEMISTRY RESULTS IN ACUTE AND CHRONIC RENAL FAILURE

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Renal failure means inability to maintain homeostasis of the organism due to kidney lesion. Patients with renal failure have reduced capacity for adaptation in stress conditions such as infection or dehydration. Renal failure is not defined by cause, degree or distribution of lesions, but can be diagnosed by laboratory results, such as haematology, biochemistry, urinalysis as well as anamnestic data, clinical examination and ultrasonography. For establishing laboratory results in patients with renal failure, total number of 19 (n=19) patients were exterminated in University veterinary hospital. The main purpose of this sudy was defining differences in acute and chronic renal failure, such as: hematology and biochemistry parameters, urinalysis and urinary sediment in dogs with acute and chronic renal failure. Dogs with renal failure were divided in two groups: first group were dogs with acute renal failure (n=9) and second were dogs with chronic renal failure. Blood samples for whole blood and serum were taken from v. cephalica antebracii externa, while urine samples were brought by the owners. Compete blood count was performed on veterinary hematology analyzer (Exigo, Sweden). Biochemistry results were performed on automated analyzer Chemwell 2910 (Awereness Tecnology, USA), according manufacture instruction (Human, Germany). Urinalysis was performed by urine strips Laboquick (Turkey). Urine sediment was obtained after centrifugation of 1500 rpm in duration of 5 minutes and sediment was stained by Diff Quick (Merck, Germany). Statistical significant differences ($p<0,001$) were noticed in RBC (10^{12}/l), (acute 6,65±1,04; chronic 3,05±0,77); HCT (%), (acute 38,66±6,01; chronic 16,83±4,06); HGB (g/dl), (16,48±2,45; chronic 7,57±1,99). There were no significant differences in the other haematology parameters. Nonregenerative, normocitic normochromic anemia was regular finging in group with chronic renal failure due to lack of erythropoietin, produced by peritubular interstitial cells of renal cortex and external medula. Other hematology parameters revealed that infective and inflamatory response are not the key factor in developing chronic renal failure. Perivascular deposition of immune complex molecules on glomerular bazal membrane and functional endotel were the main reasons for developing mesangioproliferative changes in chronic renal failure. Biochemistry results have shown that stastisticaly significant diference ($p<0,05$) appeared in following parameters: urea (mmol/L) (acute 31,12±6,77; chronic 43,19±14,40), total protein (g/L) (acute 64,58±7,28; chronic 51,63±8,26), albumins (g/L) (acute 27,89±3,41; chronic 21,81±4,76) and cholesterol (mmol/L) (acute 3,97±1,19; chronic 7,63±1,00). There were no significant differences in the other biochemistry parameters. Urinalysis revealed