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IMPACT OF LIPIDIC DISBALANCE ON FORMATION AND PROGRESS OF ARTERIAL HYPERTENSION IN PATIENTS OF A CARDIOLOGICAL PROFILE

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Actuality. Hyperlipidemia (HL) and arterial hypertension (AH) have common pathological mechanisms, enhance the progress of each other, promote development of cardiovascular complications.

Goal. To investigate impacts of various HL on uprising and progression of AH.

Materials and techniques. During the period of 3 years we analyzed interactive peculiar properties of HL and development of AH in cardiological patients, except for acute cardiac events. 112 patients (58 men and 54 women; an average age was 52.7 ± 3.8 years) with HL, and without AH were examined on the stage of inclusion into the investigation. We studied particularities of lipidogram (total cholesterol (TC), lipoproteins of low (LDL), very low (VLDL) and high (HDL) density, triglycerides (TG)), 24-hour monitoring of ABP (24-HABPM) which was conducted every 6 months during the period of 3 years.

Findings of the investigation. Out of the risk factors in the examined patients, except HL, we registered also such ones as: smoking (78 %), abdominal adiposity (80 %), ischemic heart disease (81 %). Depending on the time of AH development we could distinguish the 4 groups as follows: the 1st group (22 patients, 19.6 %) in which AH was developing during the period of 1 year; the 2nd group – in 2 years (31 patient, 27.7 %); the 3rd group – in 3 years (43 patients, 38.4 %), the 4th group – during the period of 3 years AH was not developing (16 patients, 14.3 %). The most frequent cause of AH uprising was raising of TG (the 1st group – 1.98 ± 0.08 mmol/l (67 % of patients); the 2nd group – 1.92 ± 0.09 mmol/l (62 % of patients); the 3rd group – 1.88 ± 0.09 mmol/l (54 % of patients)). For progression of AH an associated raising of TG and LDL was important (correspondingly: the 1st group (65 % of patients) – 1.96 ± 0.08 mmol/l; 4.52 ± 0.09 mmol/l; the 2nd group (57 % of patients) – 1.91 ± 0.08 mmol/l; 4.24 ± 0.10 mmol/l; the 3rd group (48 % of patients) – 1.88 ± 0.07 mmol/l; 4.13 ± 0.08 mmol/l). In the 4th group of patients we fixed an isolated raising of TC (6.82 ± 0.08 mmol/l – 67 % of patients); raising of TC and VLDL (19 % of patients – 6.74 ± 0.09 mmol/l; 0.68 ± 0.06 mmol/l correspondingly); raising of LDL and VLDL (14 % of patients – 4.09 ± 0.0 mmol/l; 0.54 ± 0.08 mmol/l).

Conclusions. HL is one of the risk factors of AH development. For appearance of AH raising of TG has the biggest importance, and for further progression of AH – a combined raising of TG and LDL. An early detection and treatment of HL with mode of life modification, even in the absence of AH, can lessen a distant cardiovascular risk.