establishment of a vaccination program. Further research is also needed to generate data on longterm clinical effectiveness and duration of protection, following 2 and 3-dose regimens. **Key words:** diagnosis, HPV, oncovirus, colposcopy, oncogene

### DEPARTMENT OF BIOCHEMISTRY AND CLINICAL BIOCHEMISTRY

## 260. INTOXICATION SYNDROME INDUCED BY TRAUMATIC HAEMOPERITONEUM DURING NONOPERATIVE MANAGEMENT

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**Introduction.** Trauma remains one of the medical and social problems with a major impact on the health of the population, especially affecting young people. Thus, 1.24 million people die annually only in road accidents according to the WHO data.

**Aim of the study.** To study intoxication syndrome in trauma patients with haemoperitoneum during nonoperative management (NOM) by means of evaluation of: necrotic substances (NS) and substances with average molecular weight (SAMW), advanced oxidation protein products (AOPP), advanced glycation end products (AGE) and total antioxidant activity (TAA).

**Materials and methods.** Prospective study (2011-2016) included 59 trauma patients with traumatic haemoperitoneum. Time frame of evaluation of biochemical parameters: at hospitalization, at 3-rd and at 5-7-th days. All trauma patients were divided in 2 groups considering haemoperitoneum volume at admission. Group I with haemoperitoneum volume up to 500 ml includes 38 patients (n<sup>1</sup>=38) and group II with haemoperitoneum volume more than 500 ml 21 patients (n<sup>2</sup>=21).

**Results.** Mean age of the patients was 37.6 $\pm$ 15.2 years. M/F ratio: 2.7/1. Trauma scores: ISS=22.9; RTS=7.4; TRISS=90.4%. Mean volume values of hemoperitoneum at hospitalization constitutes 299,74 $\pm$ 182,26 ml in group I and 788,1 $\pm$ 293,22 ml in group II with values ranging between 0 and 1500 ml. NS mean values in group I: 1.96 $\pm$ 0.91; 1.80 $\pm$ 0.69; 1.56 $\pm$ 0.39 c.u.; in group II: 2.74 $\pm$ 2.71; 1.89 $\pm$ 0.91; 1.55 $\pm$ 0.34 c.u. SAMW mean values in group I: 20.30 $\pm$ 8.58; 18.27 $\pm$ 6.04; 16.00 $\pm$ 3.66 c.u. (p<0,05); in group II: 25.44 $\pm$ 21.93; 18.46 $\pm$ 5.84; 15.96 $\pm$ 3.90 c.u. AOPP mean values in group I: 37.87 $\pm$ 20.43; 34.75 $\pm$ 17.89; 27.15 $\pm$ 13.28 µmol/L; in group II: 32.14 $\pm$ 18.61; 28.06 $\pm$ 17.33; 24.19 $\pm$ 19.52 µmol/L. Mean values of AGE in group I: 503.36 $\pm$ 176.30; 476.88 $\pm$ 179.10; 457.95 $\pm$ 164.69 mmol/L; in group II: 522.67 $\pm$ 170.96; 542.33 $\pm$ 186.09; 476.66 $\pm$ 155.48 mmol/L. TAA mean values in group I: 0.33 $\pm$ 0.06; 0.33 $\pm$ 0.09; 0.31 $\pm$ 0.05 mmol/L; in group II: 0.35 $\pm$ 0.07; 0.33 $\pm$ 0.05; 0.31 $\pm$ 0.06 mmol/L.

**Conclusions.** Intoxication indicators (NS, SAMW) in trauma patients with haemoperitoneum during NOM did not exceed normal range values and did not show any significant differences between group I and II. That can be appreciated as lack of intoxication syndrome in patients with traumatic haemoperitoneum during NOM. SAMW in group II showed statistically significant decrease in dynamic, but the values still not exceeded normal ones. Mean values of AOPP, AGE and TAA did not exceed the values of the normal ranges and, generally, did not show significant differences between both groups or in dynamics, suggesting that antioxidant body system is not affected during haemoperitoneum absorption process.

Key words: haemoperitoneum, nonoperative management, toxicity

## 261. NITRIC OXIDE: THE SYNTHESIS AND EFFECTS AT THE LEVEL OF RETINA

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**Introduction.** Nitric oxide (NO), the smallest signaling molecule known to be produced by three major isoforms of NO synthase: neuronal (nNOS), endothelial (eNOS) and inducible (iNOS), is receiving nowadays an increased interest considering its role in retinal function and pathology. This review had the intention to summarize some aspects of NO in the retina and suggest new ideas for future research.

**Materials and methods.** Recent retrospective studies that describe the function and implication of NO in pathogenesis of eye diseases were analyzed.

**Results.** The nNOS and eNOS are normally expressed and the NO produced in low quantities at the level of the retina is involved in neurotransmission and in the regulation of retinal arteriolar tonicity. iNOS that is found in Muller cells and in RPE it's not normally expressed and NO produced by it in large quantities is considered to generate inflammation of the retina and even retinal degeneration, that explains its implication in pathogenesis of hypertensive and diabetic retinopathy. NO has many physiological roles in the retina, one of it as a messenger of light-dark adaptation. It is also related to excitatory amino acid and free radical neuronal injury that occurs in the retina after ischemia or to the cell death found in such disorders as glaucoma. Recent studies have shown the implication of NO, in the etiology of ischemia and induced damage in the retina that can be a result of many pathologies or systemic diseases as diabetes and hypertension. Still the involvement of NO in the retinal blood flow in response to hypoxia is still controversial. Patients with hypertension, hypercholesterolemia, diabetes etc. showed an inability of the endothelium to generate adequate amounts of bioactive NO and to produce NO-mediated vasodilation.

**Conclusions.** Many studies performed on NOS in the retina, show us that the roles of different NOS isoforms may be much trickier than previously realized. NO acts as a regulator of different physiological processes. NO appears to have a neurodestructive or a neuroprotective action, or both in pathological conditions such as human neurodegenerative diseases. Future studies on the actions of NO and NOS in the retina will not only give us a better understanding of some processes, but may contribute to the development of pharmacological treatments for various neurodegenerative eye diseases.

Key words: retina, NO, ischemia

# 262. CORRELATION BETWEEN TRANS FATTY ACIDS AND CARDIOVASCULAR PATHOLOGY

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**Introduction.** Current studies reveal the harmful effect of trans fatty acids on cardiovascular health. Each additional gram of trans fatty acids increases the risk of myocardial infarction by approximately 5%. trans fatty acids, in principle isomers (18: 1) have two origins: natural trans fatty acids derived from the bio hydrogenation of ruminant unsaturated fatty acids and industrial trans fatty acids derived from the industrial hydrogenation of unsaturated vegetable oils.

**Aim of the study.** Elucidating the role of trans fatty acids in the mechanisms of production and evolution of cardiovascular diseases by comparing the two origins of trans fatty acids and other types of trans fatty acids.