At the cytological examination of touch smear it has been established that the healing process is accelerated on account of wound process course phologistic phase reduction. Cytologically: inflammatory-regenerative and regenerative cytogram type. It was manifested in quantity reduction of safe neutrophils up to 40-70 %, increase of tissular primitive polyphibroblasts, phibroblasts, lymphocytes up to 20-35 %, increase of macrophages number up to 5-10 %.

Bacteriological data testified to the decrease of the flora amount and the decrease of COI number by a factor of 2-3 in the wounds healed with acerbine.

In 9 persons the change of hypertrophic scars to atrophic ones was without dysfunction. These scars are easily disguised with usual dry powder. In two persons the result was poor positive, i.e. the scar is visually accessible but not skin surface overhanging, without tension function. One cryodestruction scar area elcosis case was marked. Because of allergic anamnesis the treatment with acerbine was carried out. Secondary adhesion with stellation scar formation was performed.

The given method can be recommended for linear skin scars elimination, it seems to be advisable to recommend acerbine using while cryo- and laser-burns owing to excellent cosmetic results, one of explanations of which the pH (acidity) coinciding with the pH of skin is; the method is possible to apply at keloid cicatrices.

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INFLUENCE OF ETHINYLESTRADIOL AND LEVONORGESTREL ON THROMBIN-FIBRINOGEN INTERACTION AND THROMBIN TOLERANCE (information II)

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Side effects [5, 12, 19] – hemostasis disorders and conjugated with it pathological states [1, 12, 19], are intrinsic to estrogen- and gestagen containing preparations applied in hormonal replacement therapy or contraception [3, 4]. It made clinicians pay attention to hemostasis while applying estrogen- or gestagen containing preparations [2]. Sex steroids accelerate lipid peroxidation (LPO) as well [22], that is attended by hypercoagulemia [16], and it heightened interest in investigation of LPO shifts under the influence of gestagens or estrogens in the context of hemostasis [8]. It is also important that hemorrhage affection is combined with deceleration, and thrombotism – with acceleration of thrombin-fibrinogen interaction (TFI) [5, 11], which inversely depends on thrombin tolerance (TT) and LPO activity [20].

Based on the above mentioned we experimentally studied plasmatic content of the TFI markers and TT changes at estrogen ethinyl estradiol (EE) and gestagen levonorgestrel (LNG) introduction in the context of LPO and AOP intensity in thrombocytes.

Methods: In experiments on white female rats (458 species, 170±15 g), fed with viscous consistency ration (barley and oat cereals mixture with oil), we studied the EE, LNG, prooxidant (lead acetate) and antioxidant – dimephosphon (DM), introduced with the morning portion of the ration, effects. Blood samples were taken from v. jugularis from fixed rats (narcosis – diethyl ether). The content of monomeric fibrin soluble complexes (MFSC) [18], fibrin degradation products (FDP) [15], D-dimers ("D-dimer test"-set of the firm Roche), P_3 and P_4 [10] factors, thrombin reacting fibrinogen concentration [14] and thrombin tolerance (TT) [13] were defined in the plasma. The content of diene conjugates (DC) was found out by optical density (λ - 232 nm) of heptanic phase; the content of lipid peroxides, reacting with thiobarbituric acid (TBA), was defined by fluorescence intensity (λ - 510 nm, fluorimeter "Bian130"). By the oxidation rate (OR) and induction period (IP) it was judged about the antioxidant potential (AOP) [21]. The results were evaluated by the method of variance analysis for small observational series, computing the average arithmetic (M), its average error (m), root-mean-square error (σ), confidence coefficient of Student (t) and the degree of difference possibility (p).

TFI and TT markers at EE introduction. The experiments of this family were carried out according to the scheme: the first group rats got the plain ration (control), the second one – the ration with EE (4 mcg/kg), the third – the ration with DM (1 g/kg), the fourth – the ration with DM (1 g/kg) and EE (4 mcg/kg). The samples were taken on the thirtieth day.

The EE introduction (table 1) increased the fibrinogen level and also those of FDP, FSC, D-dimers, P_3 and P_4 [10] factors, the content of DC, TBA, decreased the IP and increased the OR, i.e. accelerated the LPO rate and reduced the AOP. The DM introduction didn't influence the TFI markers, but reduced the LPO rate and increased the AOP. The DM and EE introduction eliminated the TFI shifts caused by EE. The content of DC and TBA turned out to be lower than in the control, the IP lengthened and the OR reduced. The thrombin tolerance decreased at the EE introduction and normalized at the introduction of EE+DM.

Table 1. TFI markers and thrombin tolerance in the rats fed with EE (4 mcg/kg), ДМ (1 g/kg) or EE+DM for 30 days

Factors	Experiment groups (n = 10 in a group)			
	Control (plain)	EE introduction	DM introduction	EE+DM introduction
FG, g/l	2.6±0,22	3.4±0,11*	2,4±0.22	3,3±0,24*
FDP, mg%	15,3±1,1	20,1±1,2*	14,8±0,8	16,5±1,7
MFSC, mcg/ml	22.0±0.9	26.9±0.8*	21.9±1.2	23.9±1.9+
D-D, ng/ml	0.18±0.03	0.26±0.04 *	0.17±0.03	0.17±0.04+
F. P_3,%	82,5±1,0	89,9±1,3*	79,7±1,3	84.0±1,3+
F.P_4, c	2.3±0,01	3,1±0,03*	2,2±0,04	2.4±0,01+
DC, A/mg of a lipid	0,082±0,002	0,094±0,003*	0,075±0.002*	0,078±0,002+
TBA, unit/mg of a lipid	0,21±0,005	0,32±0,002*	0,17±0,004*	0,22±0,003+
IP, min/ml	45.1±1.8	37.5±1.1*	52.7±2.1	47.5±1.3+
OR, mm3/ml/min	0.74±0.02	0.80±0.03*	0.62±0.03	0.73±0.03+
TT, %	100±4.9	61.2±3.4*	105±3.4	98.7±4.5

Symbols and notations: FG – fibrinogen, FDP – fibrin degradation products, MFSC - monomeric fibrin soluble complexes, D-D – D-dimers, F. – factor, DC - diene conjugates, TBA – products reacting with TBA, IP – induction period, A – optic density, OR – oxidation rate, DM – dimephosphon, TT – thrombin tolerance; * sign – authentic differences when compared to the first, + - to the second column.

In the second series of the experiments (the scheme is the same) the samples were taken on the 10^{th}, 20^{th} and 30^{th} day and it was found out the following: at EE introduction, especially with lead, the LPO shifts are more noticeable; at EE, lead and DM introduction there are no shifts, i.e. the lower LPO and higher AOP – the higher TT; the lower AOP – the lower TT. Probably, the TT (which characterizes animals' ability to stand hyperthrombinemia) decreases at the LPO acceleration revealed simultaneously with the growth of the TFI markers level and increases at the AOP increase, i.e. at the TFI markers level decrease.

Intensity of TFI, LPO, AOP and TT at LNG introduction. The LNG dose (6.4 mcg/kg) is higher than that of EE as well as the content of LNG in preparations for combined oral contraception. The scheme of the experiments is the same.

Signs and notations: as in table 1.

From the data of table 2 it is seen that at LNG introduction the TFI, fibrinogen, LPO, AOP and TT markers level shifts are similar, but less signified than at EE introduction. At simultaneous introduction of LNG and DM hemostatic shifts there are no changes of the LPO, AOP and TT occurred.

While studying the dynamics of the caused by LNG shifts occurrence, we carried out an experiment analogous to the one with EE. It appeared that the LPO acceleration and growth of the TFI markers level at the LNG introduction, especially with a prooxidant (lead), are more vivid; at simultaneous introduction of LNG, prooxidant and DM shifts never appeared. As in the experiments with EE the TFI markers level changes and LPO acceleration appear simultaneously, and the TT shifts are opposite on the directivity to the LPO shifts.

Table 2. TFI markers and TT in rats fed with LNG (6.4 mcg/kg), DM or LNG+DM for 30 days

Factors	Experiment groups (n = 10 in a group)			
	Control (plain), n - 9	LNG introduction, n -10	DM introduction n -10	LNG+DM introduction, n -10
FG, g/l	2.5±0,21	3.2±0,10*	2,5±0.23+	2,9±0,12*+
FDP, mg%	15,8±0,8	18,1±0,9*	14,8±0,8+	16,0±1,0+
MFSC, mcg/ml	22.3±0.8	25.1±0.7*	21.1±1.1+	23.3±.9+
D-D, ng/ml	0.17±0.02	0.21±0.02*	0.18±0.02+	0.17±0.03+
Ф. P_3,%	81,1±1,0	85,9±1,0*	79,9±1,1+	82.1±1,1+
F.P_4, c	2.4±0,02	2,8±0,02*	2,3±0,03+	2.4±0,02+
DC, A/mg of a lipid	0,085±0,003	0,093±0,002*	0,076±0.002*	0,077±0,003+
TBA, unit/ mg of a lipid	0,23±0,004	0,30±0,002*	0,18±0,003*+	0,23±0,004+
IP, min/ml	44.8±1.6	38.9±1.0*	52.1±1.8*+	47.9±1.2+
OR, m3/ml/min	0.76±0.03	0.83±0.03*	0.64±0.02*+	0.74±0.04+
TT, %	100±3.9	72.2±3.7*	104±5.4	99.5±4.5

Further it was testified that with the EE or LNG dose increase 2, 3 and 4-fold the TFI, LPO, and AOP markers level shifts surplus is not in proportion to the dose – the increase of the dose 2, 3 and 4-fold

intensifies the shifts only 1.2, 2.4 and 3.0-fold accordingly. At simultaneous EE and LNG introduction in all tested doses the TFI markers level shifts and LPO rate summarized incompletely, with the dosage increase the summation degree reduced.

Conclusions

1. At oral introduction in the dosage equivalent to antiovulatory dose for a human being EE lowers the AOP, accelerates the LPO and TFI and reduces the TT. The effects intensify with the introduction duration increase.

2. Oral introduction of LNG in equivalent doses causes less signified LPO, AOP, TFI and TT shifts of the same directivity intensifying with the introduction duration increase. **3.** At combined introduction of EE and LNG their effects on the TFI, LPO and AOP summarize only partially. **4.** An antioxidant (DM) introduction simultaneously with EE or LNG eliminates their effects on the LPO, AOP and TFI. **5.** Between the AOP of thrombocytes and TT there is a close, and between the LPO acceleration degree and TT – inverse, relationship.

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ANTIBIOGRAM ANALYSIS IN PATIENTS WITH II-III DEGREE BURNS

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Antibiotic therapy takes a leading place in complex treatment of patients with II-III degree burns S complicated with pyoinflammatory processes of soft tissues. Nowadays growth of causative agents' multiresistant strains to germicides in the given patient group is registered. That is why the disease differs from others with long-term and persistent treatment course, appreciation of treatment.

Purpose: To investigate the etiological structure of the causative agents and the spectrum of their sensibility to germicides at burn disease.

Materials and methods: In the paper the bacteB riological research analysis of the material from the burnt surfaces of 199 patients and autogenerated causative agents' sensibility to 29 germicides with the disco-diffuse method using.

Results: The contamination of the material under examination made 96,5%. The dominant activators of the burnt infection were S.aureus (50,8%) and Ps.aeruginosa (20%). From opportunistic enterobacteria Proteus (6,7%), E.coli (5,6%), Enterobacter (5,1%), Klebsiella spp. (2,6%) were plated most commonly. In 50,2% of cases combined infection forms consisting of two and more kinds, being formed on account of tolerant associations, were marked: S.aureus and Ps.aeruginosa (51,0%), S.aureus and enterobacteria of different kinds (31,0%). The antibiogram studies demonstrated nonsensibility of S.aureus to penicillins (0,9%). The most effective preparations against the given activator were: ofloxacin (45,7%),