INFLUENCE OF SYSTEMIC INFLAMMATION ON SEVERITY OF MENTAL DISORDERS SYMPTOMS IN COPD PATIENTS

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It is a well known fact that COPD is often combined with mental disorders. Unfortunately nowadays the main reason of this disorders in COPD patients is unknown. Systemic inflammation could be a cause of occurrence of mental disorders in patients with COPD.

Aim: to study the relationships between the level of systemic inflammation and severity of symptoms of anxiety and depression in COPD patients.

Materials and Methods: 37 patients with stable COPD (GOLD II-IV) were examined (male – 33 (89%), female – 4 (11%), age – 63.0 ± 0.78 yrs, FEV1 – 52.8 ± 1.24%). Methods of examination included general-clinical methods, spirometry, PHQ-9 to indentify symptoms of depression, State-Trait Anxiety Inventory (STAI) to indentify symptoms of anxiety, level of C-reactive protein (CRP), statistical methods.

Results: The lack of correlation between severity of symptoms of depression due to PHQ-9 and level of CRP in COPD patients was determined (p=0.192).

At the same time the correlation between severity of symptoms of state and trait anxiety due to STAI and level of CRP in this patients was found (p=0.033, p=0.039 respectively).

Conclusions: severity of symptoms of anxiety in COPD patients may be dependent on level of systemic inflammation.

DYNAMICS OF TRANSFORMING GROWTH FACTOR BETA 1 (TGF-B1) LEVEL IN COPD PATIENTS UNDER COMBINED TREATMENT WITH INHALATION CORTICOSTEROIDS AND LONG-ACTING BETA2-AGONISTS

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It is known, that corticosteroids therapy reduces exacerbation rates in patients with more severe disease and decrease the level of systemic inflammation. But it is unknown about its influence on profibrotic cytokine TGF-β1. Aim: to determine the dynamics of serum level of TGF-β1 under combined treatment with inhalation corticosteroids and long-acting beta agonists.

Materials and Methods: we studied 30 stable COPD patients (age – 62.4 ± 1.4 years, male – 27 (90%)), FEV1 was 45.7 [34.8–56.5]% pred. All patients were smokers or ex-smokers, index “pack/year” was 40.0 [25.0–45.0]). The control group consists of 10 healthy subjects. Measurements included clinical status, spirometry. Serum TGF-β1 level was measured twice – at first visit and after three month therapy.

Results Serum TGF-β1 levels were elevated in COPD patients as compared with healthy controls (p = 0.000). After three month therapy with inhalation corticosteroids and long-acting beta2 agonists TGF-β1 level was increased, almost twice (p = 0.003) (table 1).
Table 1 Dynamics of TGF-β₁ level

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Visit 1</th>
<th>Visit 2</th>
<th>Control</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>TGF-β₁ (pg/ml)</td>
<td>3664.8 [3293.7–5199.3]</td>
<td>6250.0 [4717.6–14581.9]</td>
<td>2620.5 [2267.7–2976.2]</td>
<td>p₁v₂v=0.003</td>
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<td></td>
<td></td>
<td>p₁v₁=0.000</td>
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<td>p₂v₁=0.001</td>
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Conclusion: Under the influence of basic therapy, which includes inhalation corticosteroids, TGF-β₁ levels were significantly increased. So, serum TGF-β₁ level may be used as an additional criterion for the disease severity, but not as an indicator of treatment effectiveness.

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THE ROLE OF PROTEIN C IN HOSPITALIZED PATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA

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There are limited data on the role of protein C in patients with community-acquired pneumonia (CAP). The aim was to estimate the role of serum level protein C (PC) on different phases of treatment in patients with CAP and to assess its value in lung thrombosis prediction.

Materials and Methods. The main group consisted of 75 patients with moderate to severe CAP. Depending on the severity, all the patients of the main group were divided into 2 subgroups: subgroup 1 – 41 patients with moderate CAP, subgroup 2 – 34 patients with severe CAP. Control group consisted of 16 healthy persons.

General clinical analyses, determination of PC were performed at admission before starting of antibacterial treatment (visit 1) and after clinical stability on 7th–10th day after hospitalization (visit 2). Statistics included ROC-analysis.

Results. Levels of PC in patients with moderate to severe CAP, Me [25-75%]

<table>
<thead>
<tr>
<th></th>
<th>Main group</th>
<th>Control group</th>
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<tbody>
<tr>
<td></td>
<td>subgroup 1</td>
<td>subgroup 2</td>
</tr>
<tr>
<td>Visit 1</td>
<td>80.0 [68.0–95.0] #^</td>
<td>70.0 [63.0–95.0] #^</td>
</tr>
<tr>
<td>Visit 2</td>
<td>101.5 [88.5–115.0]^</td>
<td>106.5 [80.0–135.0]^</td>
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<tr>
<td></td>
<td>95.0 [83.0–105.0]</td>
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</table>

Notes: * – p < 0.05 between subgroups on Mann-Witney;
# – p < 0.05 with control group on Mann-Whitney;
^ – p < 0.05 between visits on Wilcoxon.

For establishing the diagnostic value of PC we allocated 11 patients with lung vessel thrombosis (in 6 patients – on chest CT and in 5 patients – on autopsy).