INFLUENCE OF SMOKING ON BONE TISSUE CONDITION IN PATIENTS WITH COMORBID COPD AND CHRONIC PANCREATITIS

Abstract. The World Health Organization states that Chronic Obstructive Pulmonary Disease (COPD) is the third leading cause of death worldwide. COPD is a common chronic respiratory disease that leads to progressive deterioration and exacerbation of symptoms. While it can be prevented, once diagnosed, it cannot be cured. COPD management requires attention to both respiratory and extrapulmonary manifestations, such as osteoporosis, cardiovascular pathology, and anemia. Comorbidity exacerbates the course of both diseases and accelerates disability. Objective: to examine the impact of tobacco product use on bone tissue status in patients with comorbid COPD and chronic pancreatitis. Materials and Methods: Ninety patients with COPD combined with chronic pancreatitis (45 females and 45 males) participated in the study. The average age of the patients was 56.42±4.11 years. All participants underwent a medical history and complaint assessment, clinical examination, spirometry, and radiographic densitometry. Results and Discussion: In patients with the combination of COPD and chronic pancreatitis, there was a decrease in bone mineral density to the level of stage II osteopenia. All patients showed varied changes in bone mineral density with a predominance of patients with reduced bone mass. When analyzing bone density indicators in smokers and non-smokers, a significant decrease was observed in smokers, with bone density parameters in smokers corresponding to stage III osteopenia compared to healthy individuals under 45 years of age, while non-smokers had stage II osteopenia. We found a strong inverse correlation between smoking duration and bone mineral density (r=-0.718; p<0.05). Conclusions: Smoking is a factor that influences the development of osteodysplasia in patients with concurrent COPD and chronic pancreatitis. Smoking contributes to reduced bone mineral density. Densitometry data revealed diverse changes in bone mineral density in patients with COPD combined with chronic pancreatitis, with a predominance of osteopenia.
Osteosclerosis was identified in 6% of patients, first-degree osteopenic syndrome in 17%, second-degree in 27%, third-degree in 16%, and osteoporosis in every fourth patient.

**Keywords:** smoking, influence, osteoporosis, respiratory pathology, COPD, chronic pancreatitis, adults.

**ВПЛИВ ТЮТЮНОПАЛІННЯ НА СТАН КІСТКОВОЇ ТКАНИНИ У ХВОРИХ НА ХОЗЛ ТА ХРОНІЧНИЙ ПАНКРЕАТИТ**

**Анотація.** Всесвітня організація охорони здоров’я заявляє, що хронічне обструктивне захворювання легень (ХОЗЛ) є третьою причиною смерті у світі. ХОЗЛ є поширеним хронічним захворюванням дихальної системи, що призводить до поступового погіршення та посилення симптомів. Цьому захворюванню можна запобігти проте після встановлення діагнозу його неможливо вилікувати. При менеджменті ХОЗЛ доводиться звертати увагу як на респіраторні прояви, а і позалегеневі: остеопороз, серцево-судинна патологія, анемія. Коморбідність погіршує перебіг обох захворювань та пришвидчує інвалідизацію. Мета: вивчити вплив використання тютюнових виробів в контексті стану кісткової тканини у коморбідних пацієнтів із ХОЗЛ та хронічним панкреатитом. Матеріали і методи. Участь у дійсному дослідженні взяло 90 пацієнтів із ХОЗЛ у поєднанні з хронічним панкреатитом (45 жінок та 45 чоловіків). Середній вік пацієнтів становив 56,42±4,11 року. Всім учасникам було проведено збір анамнезу та скарг, клінічний огляд, спірометрія, рентгенівська денситометрія. Результати та обговорення. У хворих із поєднанням ХОЗЛ та хронічного панкреатитом мало місце зниження МЩКТ до рівня остеопенії ІІ ступеня. У всіх хворих виявлено різноспрямовані зміни КТ, причому параметри КТ стосовно здорових людей віком до 45 років у групі некурців відповідають зниженню МЩКТ до рівня остеопенії ІІІ ступеня. Нами було виявлено сильний зворотний кореляційний зв’язок між тривалістю куріння та показником МЩКТ (т=0,718; р<0,05). Висновки. Куріння є фактором впливу на формування остеодефіциту у пацієнтів із поєднаним перебігом ХОЗЛ і хронічного панкреатиту. Куріння сприяє зниженню мінеральної щільності кісткової тканини. За даними денситометрії, у пацієнтів із ХОЗЛ у поєднанні із хронічним панкреатитом виявлено різноспрямовані зміни мінеральної
щільність кісткової тканини з переважанням остеодефіциту. Остеосклероз був виявлений у 6% пацієнтів, остеопенічний синдром I ступеня - у 17%, II ступеня - у 27%, III ступеня - у 16%, остеопороз - у кожного четвертого хворого.

Ключові слова: куріння, вплив, остеопороз, респіраторна патологія, ХОЗЛ, хронічний панкреатит, дорослі.

**Introduction.** Chronic Obstructive Pulmonary Disease (COPD) holds a prominent position among non-communicable diseases and simultaneously poses a global burden on the healthcare system due to its high prevalence (affecting one in ten adults), increasing incidence, and substantial personal, social, and economic costs [1].

Tobacco smoking (both active and passive) remains a primary risk factor for the development of COPD. Toxic particles inhaled from tobacco smoke induce inflammation in the respiratory tract, which is exacerbated in individuals with COPD. This chronic inflammation persists even after smoking cessation [2].

According to the WHO, tobacco use in the 20th century resulted in 100 million deaths. If the current trend continues, tobacco use is projected to cause up to one billion deaths in the 21st century. With over one billion smokers worldwide, tobacco consumption is steadily increasing, with the epidemic shifting to developing countries. More than 80% of smokers live in low- and middle-income countries, and half of all smokers die from tobacco-related illnesses [3].

Respiratory and non-respiratory symptoms are highly prevalent, often under-assessed, and lead to several extrapulmonary manifestations of the disease, including physical, emotional, and social implications. Key non-respiratory impairments include reduced nutritional status, skeletal muscle dysfunction, osteoporosis, cardiovascular pathology, anemia, and more [4].

Osteoporosis represents a significant extrapulmonary effect in COPD. It is characterized by low bone density and microstructural changes that increase the risk of fractures. Fractures associated with osteoporosis due to reduced physical activity and prolonged bed rest have several adverse consequences for COPD patients, including worsened lung function, reduced quality of life, increased hospitalizations, and mortality [5]. Moreover, these two conditions create a vicious cycle and impose a substantial burden on these patients. Osteoporosis often remains asymptomatic in COPD patients and frequently goes undiagnosed until fractures occur. Therefore, screening for osteoporosis in COPD patients is necessary, with particular attention to early identification of those at high risk for developing osteoporosis [6].

Chronic pancreatitis is also a common issue. Researchers are increasingly focusing on the study of comorbidity between gastrointestinal diseases and COPD. When combined with COPD, chronic pancreatitis may be caused by chronic infection, sensitization, changes in blood gas composition, and microcirculation disturbances. Digestive system disorders negatively affect the course of COPD, yet there are still knowledge gaps in this area [7,8,9,10].
**Objective:** to investigate the impact of tobacco product use on the bone tissue status in comorbid patients with COPD and chronic pancreatitis.

**Materials and Methods.** A total of 90 patients with COPD in combination with chronic pancreatitis participated in the current study, including 45 females and 45 males. The patients' ages ranged from 35 to 86 years, with an average age of 56.42±4.11 years. Among these patients, 32 (36%) were diagnosed with concurrent mild COPD (stage I), while 58 (64%) had moderate COPD (stage II). The diagnoses of COPD and chronic pancreatitis were confirmed based on established criteria, utilizing data from patient histories, clinical symptoms, laboratory data, and results of instrumental examinations. The duration of COPD ranged from 1 to 25 years, with the combined duration of the diseases ranging from 1 to 10 years. Among the participants, 20 (22%) were active smokers (exclusively males). Smoking duration ranged from 10 to 30 years, with an average of 22.75±1.2 years. Nine patients had a smoking history of 10-20 pack-years, while 11 respondents had over 20 pack-years.

The degree of nicotine dependence was determined using the Fagerström Test with the following scoring: 0-3 points for low dependence, 4-5 points for moderate dependence, and 6-10 points for high dependence. Nicotine dependence was also assessed using the Smoking Index, with an index exceeding 200 indicating high nicotine dependence:

- **Smoking Index** = (number of cigarettes smoked per day) × 12.
- The smoking history was calculated in pack-years:
  - **Total pack-years** = (number of cigarettes smoked per day × number of years) / 20.
- Patients with a pack-year index exceeding 25 were considered "heavy" smokers, while those with 10 pack-years were classified as "moderate" smokers [4].

The bone status was assessed using dual-energy X-ray absorptiometry (DXA) at the lumbar spine (L1-L4) with consideration for intervertebral clefts, including osteoarthritic changes (g/cm2) with an accuracy of 0.02 g/cm2. The analyzed parameters included bone mineral density (BMD) in the L1-L4 lumbar region, expressed as grams per square centimeter (g/cm2). The relative indicators, T-score (peak bone mass) compared to healthy individuals aged 20-45 years in standard deviation (SD) units, and Z-score, analogous to T-score but concerning people in the same age group, were also measured. The results were presented as percentages of BMD in comparison to healthy young individuals and individuals within the same age group. Osteopenia, osteoporosis, or osteosclerosis were determined based on the T-score. The data were evaluated following WHO recommendations (Geneva, 1994). The levels of osteopenia were assessed using the method of Rozhynska L.Ya. Statistical analysis was performed using standard statistical software on a personal computer.

Results and Discussion. The densitometric indicators of the study participants are presented in Table 1.
Table 1

<table>
<thead>
<tr>
<th>Indicator</th>
<th>CP and COPD (n = 90)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMD (Bone Mineral Density), g/cm²</td>
<td>0,961 ± 0,017 *</td>
</tr>
<tr>
<td>T-score, CU</td>
<td>-1,74 ± 0,14 *</td>
</tr>
<tr>
<td>T-score, %</td>
<td>82,36 ± 1,33 *</td>
</tr>
<tr>
<td>Z-score, CU</td>
<td>-1,204 ± 0,11</td>
</tr>
<tr>
<td>Z-score, %</td>
<td>85,04 ± 0,87 *</td>
</tr>
</tbody>
</table>

* statistically significant results

According to our data, it was determined that the identified DXA parameters in patients with the combination of COPD and CP correspond to a decrease in BMD to the level of stage II osteopenia, considering healthy individuals under the age of 45.

All patients exhibited diverse changes in BMD, with a predominance of patients having decreased bone mass. During the analysis of the DXA indicators in smokers and non-smokers, a likely decrease in these indicators was observed in smokers (Table 2). Specifically, the DXA parameters in smokers, concerning healthy individuals under the age of 45, correspond to a decrease in BMD to the level of stage III osteopenia, while in non-smokers, it corresponds to stage II osteopenia. This provided grounds to consider smoking as one of the factors influencing the development of osteodystrophy in COPD in combination with CP. A strong inverse correlation was found between smoking duration and BMD (r=-0.718; p<0.05).

Table 2

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Non-smokers (n = 67)</th>
<th>Smokers (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMD (Bone Mineral Density), g/cm²</td>
<td>0,975 ± 0,021</td>
<td>0,908 ± 0,027 *</td>
</tr>
<tr>
<td>T-score, CU</td>
<td>—1,66 ± 0,17</td>
<td>—2,01 ± 0,13 *</td>
</tr>
<tr>
<td>T-score, %</td>
<td>83,09 ± 1,62</td>
<td>79,80 ± 1,85</td>
</tr>
<tr>
<td>Z-score, CU</td>
<td>—1,13 ± 0,14</td>
<td>—1,46 ± 0,09 *</td>
</tr>
<tr>
<td>Z-score, %</td>
<td>88,13 ± 1,17</td>
<td>83,22 ± 1,49 *</td>
</tr>
</tbody>
</table>

* statistically significant results

The obtained results emphasize the importance of prevention and control of smoking in patients with COPD and CP to preserve the health of the bone system.
Taking this factor into account is of great significance in clinical practice and aids in the planning of treatment and rehabilitation for patients with these conditions.

**Conclusions:**

1. Smoking is a factor that influences the development of osteodystrophy in patients with the concurrent course of COPD and CP. Smoking contributes to a decrease in bone mineral density.

2. According to densitometry data, patients with COPD in combination with chronic pancreatitis exhibit diverse changes in bone mineral density, with a predominance of osteodystrophy. Osteosclerosis was identified in 6% of patients, osteopenic syndrome of stage I in 17%, stage II in 27%, stage III in 16%, and osteoporosis in every fourth patient.

**References:**


