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Smoking as a Factor in the Formation of Recurrent Chronic Gastroduodenal Pathology in Adolescents

Conflict of interest: nothing to declare.
Authors' contribution: Bobrova V. – concept and design of the study; Proshchenko Ju. – writing the article; Horobets A. – editing the article; Levadna L. – data collection.

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Abstract

Purpose. To establish the effect of smoking on the formation of recurrent chronic gastroduodenal pathology in adolescents.
Materials and methods. We observed 60 teenagers aged 12–17 years with verified chronic gastroduodenal pathology (chronic gastritis and chronic duodenitis, ulcer disease, duodenal ulcer). All patients underwent a study of urinary cotinine to identify active smokers. To verify chronic gastroduodenal pathology, all adolescents underwent esophagogastroduodenoscopy with targeted biopsy of the stomach and duodenum mucosa membrane. The level of acidic and neutral mucopolysaccharides, prostaglandin E was determined by immunohistochemical examination.
Results. In dominating majority of smoking teenagers we have observed expressed inflammation, atrophic and microcirculatory changes of stomach and duodenal mucosa and focal destruction of glands. Investigation has demonstrated remarkably decreased indexes of supraepithelial and epithelial mucosal protective barrier in gastroduodenal zone in smoking teens.
Conclusions. Received data indicate negative influence of nicotine on mucosal architectonics, pyloric and brunner's glands condition as well as stomach's mucosal protective barrier functioning which contribute to prolonged and relapsing course of chronic gastroduodenal pathology.
Keywords: smoking, microcirculatory disorders, inflammation, chronic gastroduodenal pathology, mucosal cytoprotective barrier

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Табакокурение как фактор формирования рецидивирующего течения хронической гастродуоденальной патологии у подростков

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Резюме

Цель. Установить влияние курения на формирование рецидивирующего течения хронической гастродуоденальной патологии у подростков.
Материалы и методы. Под наблюдением находилось 60 подростков 12–17 лет с верифицированной хронической гастродуоденальной патологией (хронический гастрит и хронический дуоденит, язвенная болезнь, язвенная болезнь двенадцатиперстной кишки). Все пациенты прошли исследование на содержание котинина в моче для выявления активных курильщиков. Для верификации хронической гастродуоденальной патологии всем подросткам была выполнена эзофагогастродуоденоскопия с прицельной биопсией слизистой оболочки желудка и двенадцатиперстной кишки. Уровень кислых и нейтральных мукополисахаридов, простагландина Е определяли иммуногистохимическим исследованием.
Результаты. У большинства обследованных курящих подростков мы наблюдали выраженную степень воспаления, атрофические и микроциркуляторные изменения слизистой оболочки желудка и двенадцатиперстной кишки, очаговую деструкцию желез. Исследование показало, что у курящих подростков отмечали выраженное снижение показателей преэпителиального и эпителиального уровня защиты слизистого барьера гастродуоденальной зоны.
Выводы. Полученные данные указывают на негативное влияние никотина на гистоархитектонику и состояние пилорических и бруннеровых желез слизистой оболочки, а также на функционирование защитного слизистого барьера желудка, способствуя длительному, рецидивирующему течению хронической гастродуоденальной патологии.
Ключевые слова: табакокурение, микроциркуляторные нарушения, воспаление, хроническая гастродуоденальная патология, цитопротективный барьер слизистой оболочки

■ INTRODUCTION

It is known that smoking causes pathology of many organs and systems of the body. The WHO report that smoking of tobacco products contributes to the pathology of the

respiratory tract, worsens the condition of the heart and blood vessels, affects the condition of the visual analyzer, is a factor in diseases of the gastrointestinal tract (GIT), specifically oral cavity and gastroduodenal pathology [3, 4, 8, 12, 15]. Smoking negatively affects not only the person who smokes directly, but also on passive smokers [9, 12,15]. It is well known that under the influence of passive smoking, inhalation of smoke irritates the mucous membrane (MM) of the upper respiratory tract, which leads to a decrease in the production of local protective factors and the forming of chronic inflammation in the upper respiratory tract. Children who are under the influence of long-term passive smoking are at risk of developing bronchial asthma and other allergic respiratory pathology. In the literature there is a lot of data on the pathogenetic mechanisms of smoking on the occurrence of diseases of the cardiovascular and respiratory systems [4, 6, 8, 9, 12, 15]. Studies about the effects of smoking on the formation and course of gastroduodenal pathology in childhood are almost absent. The etiological factors of chronic gastroduodenal pathology in addition to eating disorders, H. pylori infection, disorders of motorical and acid-forming functions of the stomach, psycho-emotional stress, also include bad habits [2, 5, 7, 10, 13, 14]. According to modern research, smoking has a negative effect on the gastrointestinal MM due to direct exposure to absorbed tar compounds during smoking. In addition, nicotine has a stimulating effect on N-cholinoreceptors, which leads to increased secretory and acid-forming functions of the stomach. Smoking of tobacco products disrupts the blood supply and microcirculation of all organs and tissues, including the gastrointestinal tract, which leads to a decrease in the synthesis of pre-epithelial, epithelial and subepithelial factors of protection of MM [4, 5, 8, 9, 12, 15].

■ PURPOSE OF THE STUDY

To establish the effect of smoking on the formation of recurrent chronic gastroduodenal pathology (CGDP) in adolescents.

■ MATERIALS AND METHODS

Under our observation there were 60 children active smokers aged from 12 to 17 years with verified CGDP (chronic gastritis and chronic duodenitis, duodenal ulcer). To objectively assess the impact of tobacco smoke the level of cotinine in the urine was used as an indicator, which is a product of nicotine metabolism and cannot enter the body in any other way. The level of cotinine in the urine was examined using an immunochromatographic method using a standard set "ImmunoChrome-Cotinine-Express" (Moscow). To verify the diagnosis all children underwent esophagogastroduodenoscopy (EGDS) and intragastric pH-metry during first 3 days after hospitalization. To detect H. pylori contamination serological and histological methods were used. H. pylori imaging was carried out by histological investigation of antral stomach's biopsy mounts colored by Romanovsky – Himze method. Coloration of tissues' slices with hematoxylin and eosin was used to evaluate histological changes of stomach and duodenum's mucosa. The received results were interpreted according to "Sydney system". In order to evaluate protective mucous barrier potency gastric mucosa's biopsy materials coloration for detection of neutral mucopolysaccharides with Schiff reagent (Vasilenko V.H., 1971) and acid mucopolysaccharides with alcian blue (pH 2,5) (Heil, 1948) were performed [1]. Prostaglandins E (PgE) were detected by immunohistochemical method with the help of mice polyclonal antibodies (Dako, Denmark).

Statistic examination of the results was made by using standard Microsoft Excel programs.

■ RESULTS

Boys aged 15–17 years (71.7±5.8%) with a disease duration of more than 5 years predominated among the examined patients. With an increase of the duration of smoking in 51.1±7.5% of respondents noted an increase in the number of exacerbations of the disease to 4–5 times a year. The burden of family history of CGDP was observed in only 25±5.6% of adolescent smokers. The data obtained indicate that smoking can be considered as a major factor in the formation and recurrent course of CGDP in this category of patients. Boys aged 15–17 years (71.7±5.8%) with a disease duration of more than 5 years predominated among the examined patients. With an increase in the duration of smoking in 51.1±7.5% of respondents noted an increase in the number of exacerbations of the disease to 4–5 times a year. The burden of family history of CGDP was observed in only 25±5.6% of adolescent smokers. The obtained data indicate that tobacco smoking can be considered as the main factor in the formation and recurrent course of CGDP in this category of patients.

At the time of admission to the hospital, all patients complained of abdominal pain, the severity and intensity of which ranged from short-term paroxysmal to mild aching. An assessment of the pain syndrome showed that the pain syndrome was not associated with food intake or defecation. The most common manifestations of dyspepsia were heartburn (53.3±6.4%) and belching (35±6.2%). Manifestations of chronic nonspecific intoxication and asteno-vegetative syndrome were present in all patients, they manifested themselves in the form of fatigue, weakness and headache.

All patients with parental consent were performed EGDS with targeted biopsy of stomach and duodenum MM for further morphological, histochemical and immunohistochemical examination. The obtained data showed that in most patients erythematous changes of stomach and duodenum MM prevailed 91.7±3.6%. Among adolescents with destructive changes in the stomach and duodenum MM, duodenal ulcer was diagnosed in 35.0±6.2%. During EGDS, attention was paid on the functional state of the cardiac and pyloric sphincter. According to the results of our study, motorical disorders of the gastrointestinal tract in the form of duodenogastric reflux and gastroesophageal reflux were noted in the majority of 70±5.9% of patients. In the study of acid-forming function of the stomach, it was found that most patients had a hypoacidity (58.3±6.5%), and hyperacidity ware diagnosed in 15±4.6% of adolescents. H.pylori infection was diagnosed in 63.6±14.5% of patients.

During the morphological study of stomach MM in most adolescents who smoke, the degree of inflammation activity was expressed against the background of microcirculatory disorders with microthrombosis, hemorrhage and dystrophic changes in the early stages of development. The relief of the MM was disturbed, there were shortening of the pits and flattening of the rollers. Superficial epithelium with areas of desquamation and foci of flattening. The MM is densely infiltrated with lymphocytes and plasma cells, single eosinophils and neutrophils with foci of fibrosis, proliferation of fibroblasts and thin collagen fibers, which have fuzzy contours and are located in both basal and superficial parts. Glands in own plate are unevenly located, with foci of destruction (fig. 1).

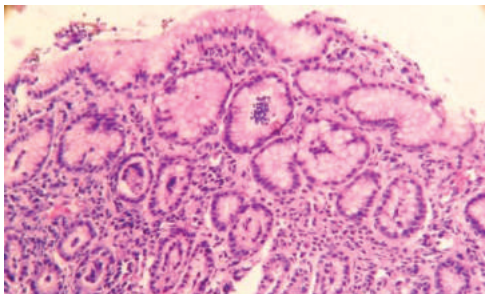


Fig. 1. Microphoto of the biopsy of the stomach MM. Antral department, pronounced uneven lympho-plasmacytic infiltration. ×200

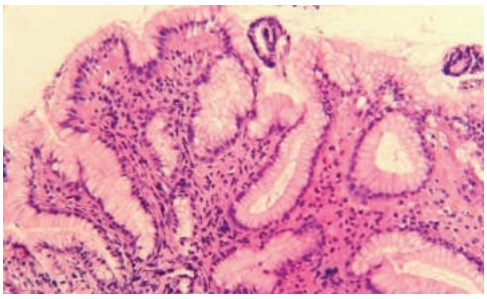


Fig. 2. Microphoto of the stomach mucosa's bioptat. Local atrophy. ×200

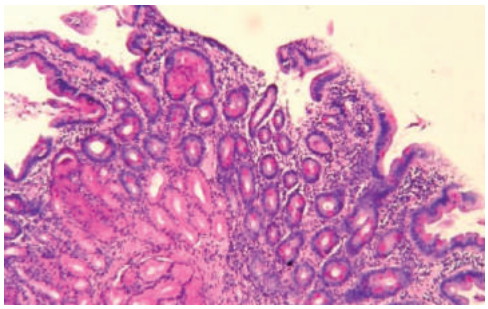


Fig. 3. Microphoto of the duodenal mucosa's bioptat. Area of brunner's glands hyperplasia. ×200

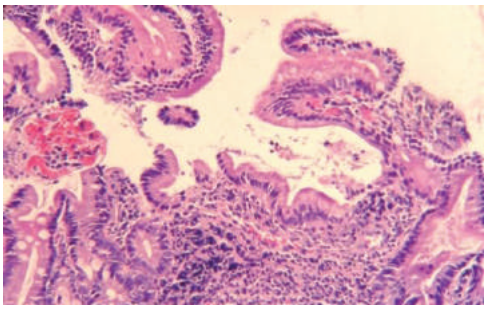
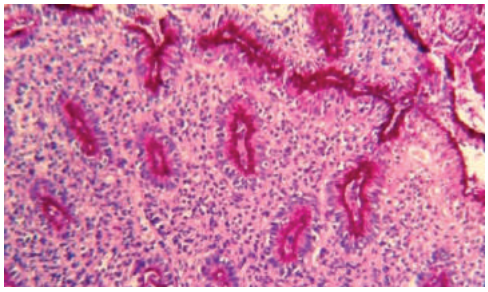
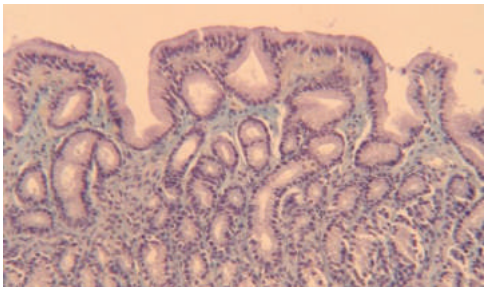


Fig. 4. Microphoto of duodenal mucosa's bioptat. Mucosal hypotrophy. ×200



a



b

Fig. 5. A – microphoto of stomach mucosa's bioptat stained with PAS reactive for neutral mucopolysaccharides, ×100, b – microphoto of stomach mucosa's bioptate stained with alcyanic blue for acidic mucopolysaccharides, ×200

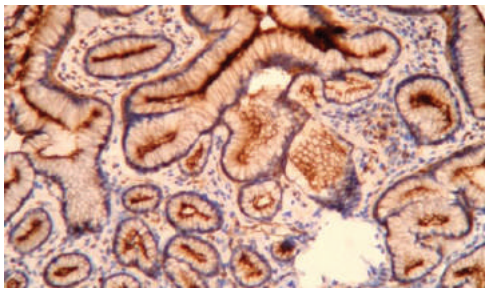


Fig. 6. Microphoto of stomach mucosa's bioptat, immunohistochemical reaction with polyclonal antibodies to PGE2

Mucosa's atrophic changes have been detected in 6.9±4.7% of teens in antral localization of pathological process. Predominantly local and rarely spread thinning of the mucosa and impairment of its relief reflected in rolls flattening and pits shortening have been found out. Areas with glands replaced by fibrous tissue have been detected with small sizes of foci across 1 – 2 rolls (fig. 2).

During morphological examination of duodenal mucosa focal impairment of mucosal relief, predominance of villi with atypical shape and superficial erosions have been marked. Local hyperplasia of brunner's glands which their replacement of basal mucosa have been found (fig. 3).

Atrophic changes of duodenal mucosa have been detected in morphological investigation in 17.2±7.0% of examined teens. In this shortening and flattening of villi and elongation of crypts have been marked. Epithelium covering mucosa's surface has been found to be remarkably flattened; those were rare and unevenly localized glanduli found in lamina propria and a part of them has been covered with a flattened epithelium and a pilled epithelium has been found in their space. Areas with glands which had been replaced by fibrous tissue were detected with small sizes of foci across 2 – 3 rolls (fig. 4).

PAS reaction intensity has been assessed in stroma, epithelial cells apical part and areas of stomach pits and glands. According to the received results of our investigation it has been estimated that neutral mucopolysaccharides have been detected in majority of teens across apical margin of epithelial cells. Glycosaminoglycans quantity has been significantly decreased and their distribution has been uneven in stomach's pits and glands. In contrast to neutral mucopolysaccharides acidic glycosaminoglycans have been marked in 27.3±13.4% of examined patients in stomach's pits which testifies to affection of the protective mucosal barrier in this category of patients as well as proves that detected acidic mucopolysaccharides which have better cytoprotective potential are a kind of compensatory reaction of the mucosa on impairment (fig. 5).

Analysis of the performed immunohistochemical investigation has demonstrated decreased level of PGE2 expression in the prevailing majority (62.1%±9.0) of examined patients (fig. 6).

■ CONCLUSIONS

Thus, in dominating majority of smoking teenagers we have observed expressed inflammation, atrophic and microcirculatory changes of stomach and duodenal mucosa and focal destruction of glands. Investigation has demonstrated remarkably decreased indexes of supraepithelial and epithelial mucosal protective barrier in gastroduodenal zone in smoking teens. Received data indicate negative influence of nicotine on mucosal architectonics, pyloric and brunner's glands condition as well as stomach's mucosal protective barrier functioning which contribute to prolonged and relapsing course of chronic gastroduodenal pathology.

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