при мощности 500-700 Вт. При оценке результатов реакции учитывается экспрессия только в инвазивном компоненте опухоли. Оценка результатов реакции проводится с помощью балльной шкалы оценки — 0, 1+, 2+, 3+.

Статистическая обработка данных производилась с использованием «Microsoft Office Excel 2010».

# Результаты исследования и их обсуждение

Было выявлено 9 (15 %) случаев HER2-позитивного РМЖ, 7 (11,7 %) случаев тройного негативного РМЖ, 28 (46,7 %) случаев люминального A РМЖ, 16 (26,7 %) случаев люминального B РМЖ.

Люминальный тип получил название от люминальных клеток, формирующих внутренний слой протоков и долек молочной железы. Люминальный А тип наблюдался в 22 (78,5 %) случаях у пациенток старше 50 лет (85 %) и в 20 (71,4 %) случаях имел строение долькового рака.

НЕR2-позитивный РМЖ чаще наблюдался в возрастном диапазоне 40–60 лет: 7 (77,8 %) случаев. У большинства пациенток отмечалась низкая дифференцировка опухоли: 8 (88,9 %) случаев.

Тройной негативный РМЖ чаще наблюдался в возрастном диапазоне 40–60 лет: 7 (85,7 %) случаев.

В 5 (8,3 %) случаях из 60 в течение 10-летнего периода наблюдения у пациенток развился рецидив РМЖ: в 2 (22,2 %) случаях из 9 в группе НЕR2-позитивного РМЖ, в двух случаях из 7 (28,5 %) в группе тройного негативного РМЖ, в 1 (9,1 %) случае из 11 в группе люминальный типа В РМЖ.

Частота метастазирования в регионарные лимфоузлы у 51 женщины с отрицательной экспрессией HER2 составила 25 (49 %), в то время как у 9 женщин с позитивным HER2-neu частота метастазирования составила 7 (77,8 %) случаев.

#### Выводы

В группах пациенток с тройным негативным РМЖ и в группе с HER2- позитивным РМЖ чаще наблюдались метастазы в регионарные лимфоузлы и чаще наблюдались случаи рецидивов РМЖ.

В проблеме злокачественных опухолей, и, в частности, РМЖ, важны как изучение этиологии и патогенеза, так и разработка новых методов лечения и реабилитации больных.

Улучшению результатов лечения способствует и разработка новых методов лучевой и химио-, гормональной терапии.

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# A CASE SERIES OF 27 PATIENTS WITH SIGNET RING CELL CARCINOMA OF THE STOMACH

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#### Introduction

Our body is made up of billions of different cell types that can only be seen under a microscope. These cells are grouped according to their function or the type of body tissue they produce. Signet cells are a type of epithelial cell called glandular

cells. Epithelial tissue is skin tissue that covers the inside and outside of the body. [5]. In fact, under the microscope, cells look like signet rings. Signet ring cell carcinoma (SRCC) is a rare form of highly malignant adenocarcinoma that produces mucin. It is a malignant epithelial tumor characterized by histological appearance of signet ring cells [4]. Primary SRCC tumors are commonly found in glandular cells of the stomach (SRCC develops in the stomach in 56 % of patients) and are less common in the breast, gallbladder, bladder, and pancreas. SRCCs do not usually form in the lungs, although some cases have been reported [2]. In colon cancers, the incidence of SRCC is <1%. Although the incidence and mortality of gastric cancer has decreased in many countries over the past 50 years, there has been an increase in the incidence of SRCC type gastric cancer [1]. Some events are inherited, and these cases are often caused by mutations in the CDH1 gene, which is the code for the important cell-cell adhesion glycoprotein E-cadherine. Somatic mutations of the APC gene have also been implicated in the development of gastric SRCC. The role of other risk factors in gastric cancer such as salty foods, smoking, and autoimmune gastritis has not been well studied in SRCC [4].

SRCC tumors grow into characteristic sheets and are diagnosed using standard imaging techniques, such as computed tomography and PET, which are less effective. Gastric cancer (GC) is responsible for 9 % of cancer deaths worldwide. Over 950,000 new cases are diagnosed each year, and about 90 % of them are in advanced stage, requiring chemotherapy [4]. In Europe there has been research based on pre- and postoperative chemotherapy treatment, using 5-fluorouracil, epirubicin, cisplatin, capecitabine, and docetaxel. Chemotherapy significantly impairs the quality of life of patients; however, the final effects are not always satisfactory. This review paper is mostly addressed to physicians who are interested in updating to the state of the art concerning different subtypes of gastric carcinoma [1]. Recent advances in the epidemiology, pathology, molecular mechanisms, and combined modality therapy (CMT) fields have shown that gastric signet ring cell carcinoma (GSRC) should be considered a distinct cancerous entity. Clinical management of this cancer is challenging, with chemoradioresistance and poor outcomes in advanced stages [3]. Pathological and molecular sets of GSRC demonstrate different features of poor cohesion and differentiation according to the WHO, Japanese Gastric Cancer Association, and Laurén classifications Figure 1 [1].

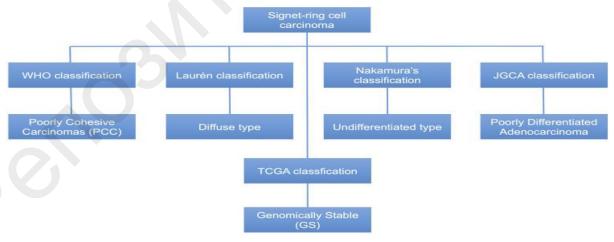


Figure 1 — The pathological classification of GSRC [2]

#### Goal

To study about the signet ring cell carcinoma. To identifying the causes, statistics and diagnostic methods from the patient's anamnesis. To know the treatment and prevention.

# Material and methods of the research

The analysis and generalization of modern medical scientific literature on this topic, also using patient's anamnesis data from pathological department form 3 Belarusian hospitals from 2020–2021.

# The results of the research and their discussion

SRCCs are named because they resemble signet rings, resulting in large vacuoles-filled with mucin that move the nucleus around the cell circumference. Stomach cancers with adenocarcinoma and some CRS (also called mixed SRCC) exhibit more aggressive behavior than pure SRCC or non-SRCC histology. The study of SRCC colon cancers compared myosin-rich SRCC tumors with myosin-poor SRCC tumors. They concluded that the latter often reveals unfavorable histological features such as lymphatic invasion, venous invasion and perineural invasion. SRCCs differ from adenocarcinomas in that they lose the ability for cell-cell interaction. The most distinct adenocarcinomas are those that mimic SRCCs and form tight junctions that typically separate MUC4, a mucin protein, and ErbB2, a carcinogen. If MUC4 and ErbB2 can communicate, they will trigger the activation loop. As a result, the ErbB2 / ErbB3 signal pathway is systematically activated, cell-cell connections are lost, and signet ring carcinomas develop. The structural activity of the ErbB2 / ErbB3 complex also promotes cell growth. The mechanism of this malignant cancer is not yet clear; however, a colon cancer cell called HCC2998 causes an increase in the production of differentiated tumors. This increase is due to the conversion of active PI3K to SRCClike cells. The type of metastasis to gastric signet cell carcinoma is different from that of gastrointestinal cancer. SRCC tumor is most often found in the peritoneum and spreads to the lymphatic infiltrates of the lungs and uterus, forming Krukenberg tumors. Cases of gastric carcinomas metastasizing to the breast and forming signetring cells have also been reported. If signal cells are found in the breast, the study suggests that the presence of gastric cancer should also be considered (Table 1).

Table 1 — Data of patients with morphologically diagnosed SRCC

No	Age	Sex	Clinical diagnosis
1	56	M	Infiltrative gastric cancer
2	67	F	Advanced stomach cancer
3	55	F	Gastropathy. HP+
4	60	M	Stomach polypoid tumor
5	74	M	Stomach cancer
6	59	F	Stomach polypoid tumor
7	80	M	Advanced stomach cancer. Gastric ulcer, Crohn disease
8	60	M	Ulcer, malignancy
9	47	F	Gastric malignancy
10	50	F	Ulcer of antral region. Malignization
11	83	F	Disease of the ascending colon.
12	48	M	C-r of the antrum of the stomach. Chr. ulcer of the prepyloric region
13	48	F	Gastropathy. HP
14	58	M	Advanced stomach cancer. Type 3 (infiltrative ulcer)
15	63	M	Gastric ulcer complicated by malignancy
16	66	M	Gastric ulcer, Cancer
17	60	M	Submucosal tumor of the antrum of the stomach
18	54	M	Erythematous gastropathy. Advanced stomach cancer
19	91	F	Stomach cancer
20	64	M	Cancer of the cardiac esophagus
21	61	F	Stomach cancer
22	55	M	Stomach cancer
23	82	F	Stomach cancer
24	77	M	Cardio esophageal cancer
25	67	F	Stomach cancer
26	2	F	C-r of the body of the stomach, infiltrative-ulcerative form
27	56	F	Stomach cancer

According to the data shows Stomach cancer (29.63 %), Infiltrative gastric cancer (11.11 %), Cardio esophageal cancer (7.04 %), advanced stomach cancer (14.81 %), tumor form (11.11 %), Gastric ulcer (25.93 %) and Crohn disease (3.7 %).

#### Conclusion

Significant progress has been made in understanding the pathogenesis and the molecular biology of GSRC and in optimizing the available treatment options and modalities. However, improving outcomes for patients with GSRC remains a significant challenge. GSRC has several features, such as chemoresistance and peritoneal metastasis, which suggest poor response to anti-cancer drug-based therapies. This article has reviewed how improving the understanding of the pathological and molecular subgroups may facilitate the selection of patients that may benefit from CMT, including surgery, chemoradiation, immunotherapy, and HIPEC. Due to the absence of specific and effective molecular targets, challenges remain in the treatment strategy of GSRC. Thus, further studies should focus on the pathogenesis and molecular biology of GSRC.

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# MORPHOLOGICAL AND DIAGNOSTIC OVERVIEW OF PULMONARY ALVEOLAR MICROLITHIASIS AND EVALUATING A SUPPORTING THERAPHY TOWARDS A RARE LUNG PATHOLOGY

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# Introduction

Interstitial lung diseases are lung disorders that affects the interstitium and the air sacs and eventually causing fibrosis of the lungs. Pulmonary Alveolar Microlithiasis (PAM) is one of rarest among the interstitial lung diseases and is associated with accumulation of diffuse microscopic calculi called microliths within the alveolar spaces that progresses with interstitial fibrosis. This disease has been distributed with less than 800 cases in all the continents of the globe and specifically with most cases of this disease been recorded in Turkey and Italy. PAM occurs in both sexes without any specific race preference. It occurs at any age, but higher incidences are between 20-50 years of age. However, according to worldwide reports we have cases recorded with neonates and also cases with elderly people. PAM is mostly detected incidentally on chest radiographs while we perform radiographic imaging's for other pathologies. The causes of PAM are still idiopathic as the mechanism inducing microliths formation is still unknown despite, it has a proven genetic theory that expresses it's a rare inherited disease in an autosomal recessive manner with identification of this pathology in children whose parents were close relatives. A mutation in the SLC34A2 gene that encodes a type IIb sodium-phosphate co-