The conduct in diagnosing the vulvar lesion is to carefully inspect the affected region and the groin nodes, assessing the size of the lesion and the regional adenopathies. The main element of the diagnosis is vulvar biopsy with morphological confirmation. Treatment is performed by partial or radical vulvectomy.

Aim of the study. Appreciation of the methods of diagnosis and treatment of benign vulvar tumors.

Materials and methods.. In the study group were included 16 patients with benign vulvar tumors diagnosed and treated in IMSP IO from the Republic of Moldova during the years 2014-2019.

Results. Total enrolled: 16 patients. Distribution by age groups: 41-50 years - 5 patients (31.25%), 51-60 years - 5 patients (31.25%), 31- 40 years - 3 patients (18.75%), 61-70 years - 3 patients (18.75%). Based on the predisposing factors in the development of benign vulvar tumors, there were 7 cases of obesity (43.75%) and the presence of HPV virus type 6 and 11 in 9 patients (56.25%). According to the location: on the right labia - 6 patients (37.5%), and on the left labia - 10 patients (62.5%). In the examination of patients by ultrasonography we obtained in 10 patients a formation less than 5cm (62.5%), in 3 patients a formation of 6-10cm (18.75%) and in 3 patients a formation greater than 11 cm (18.75%). %). Based on the histological examination there were 5 cases of vulvar papilloma (31.25%), 8 cases of vulvar fibroma (50%) and 3 cases of vulva leukoplakia (18.75%). All patients underwent surgical treatment: partial vulvectomy -15 patients (93.75%) and radical vulvectomy - one patient (6.25%).

Conclusions. 1. Obesity and the presence of HPV virus types 6 and 11 are some of the primary factors leading to the development of benign vulvar tumors 2. Histopathological examination represents the gold standard in the diagnosis of benign vulvar tumors. 3. Surgical treatment is the method of choice in the treatment of benign vulvar tumors..

Key words: HPV, vulvar tumor, diagnosis, treatment, histological examination.

97. THE FEASIBILITY OF SENTINEL LYMPH NODE (SLN) BIOPSY EXAMINATION AFTER NEOADJUVANT CHEMOTHERAPY FOR BREAST CANCER PATIENTS

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Introduction. The feasibility of sentinel lymph node biopsy examination for breast cancer patients that had no clinically detected lymph nodes and underwent neoadjuvant chemotherapy have been analyzed by injecting blue dye into the area near the tumor.

Aim of the study. Axillary status is one of the most important prognostic factors for breast cancer. Sentinel node biopsy has become a standard procedure for axillary staging in clinically node-negative patients. This technique brings out important information that helps physician in therapeutic management of these patients. Lymphadenectomy is an invasive procedure associated with higher morbidity and complications that has shown to be unnecessary in some cases of breast cancer.

Materials and methods.. Forty patients with stages 0-II breast cancer treated with neoadjuvant chemotherapy were enrolled in the study. The sentinel node biopsy was performed after blue

dye injection into the tumor. Sentinel nodes stained bright blue and were removed. The sentinel nodes have been examined under the microscope for cancer signs. Depending on the biopsy results this was followed or not by lymphadenectomy . Parameters like age, size of tumor, Nottingham grade, presence of hormonal receptors, HER 2 enriche, presence of microcalcification, necrosis and inflammatory infiltrate have been studied to predict the risk of axillary metastasis.

Results. Forty patients received SLN biopsy after neoadjuvant chemotherapy. Ten cases (25%) of these had positive sentinel lymph nodes confirmed by anatomopathological examination. Lymphadenectomy procedure have been performed and only 3 of them (30%) had metastatic lymph nodes in the rest of the axilla.

Conclusions. SLN biopsy accuracy after neoadjuvant chemotherapy is still debated in literature. Thirty of our patients were saved form an unnecessary axillary lymph node dissection by using SLN biopsy technique.

Key words: breast cancer, neoadjuvant chemotherapy , sentinel nodes

98. TYPE 3C (PANCREATOGENIC) DIABETES MELLITUS

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Introduction. Exocrine pancreatic insufficiency is frequently associated with diabetes, with high prevalence in both insulin-dependent or insulin-independent patients. Historically, diabetes due to diseases of the exocrine pancreas was described as pancreatogenic diabetes mellitus, but recent literature refers to it as type 3c diabetes as it was classified by American Diabetes Association.

Aim of the study. De-novo diabetes mellitus is an important consequence of distal pancreatectomy, ductal adenocarcinoma, chronic pancreatitis and a better understanding of the frequency and risk factors for this outcome may allow alteration of the treatment course. Our goal involves identifying causes and differences between some entities of type 3c diabetes mellitus

Materials and methods.. The following represents a summary of the relevant literature in electronic databases, with the purpose of providing more insight into the important relationships between pancreatic ductal adenocarcinoma (PDAC), distal pancreatomy and chronic pancreatitis with diabetes. Relevant literature cited in electronic databases Scopus, EMBASE, MEDLINE, Web of Science, The Nature, The Lancet.

Results. Even if in case of distal pancreatectomy etiology may be clear-absence of islets leads to lowering of the insulin, there are however some specifics: Due to an increased peripheral sensitivity to insulin and the reduced glucagon level in pancreatogenic diabetes, exogenous insulin administration frequently causes hypoglycemic attacks, characteristically called 'brittle' diabetes. On the other side low levels of pancreatic polypeptide raises blood glucose level drastically. In chronic pancreatitis (CP) inflammatory environment and increased concentration of pro-inflammatory cytokines such interleukin 1β, 1R, tumor necrosis factor (TNF) α and agents like adrenomedullin or vanin-1 within the pancreatic parenchyma mediate β-cell dysfunction before frank β-cell loss. As chronic pancreatitis progresses, the extensive