ADENOCARCINOMA OF GLANDULE BARTHOLIN FOLLOW UP FOR FIFTEEN YEARS: A CASE REPORT

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Introduction/Background Adenocarcinoma gland Bartholin’s is a very rare tumor and there is no agreement of optimal treatment for this type of carcinoma.

It accounting 2-7% of all cancers of the vulva and less than 1% of all female genital malignancies. Basic features are expanding locally, slow grow, and gives unexpected distant metastasis.

Methodology We will show our case and how we treated women with adenocarcinoma glandule Bartholin for fifteen years. 60-year old woman was admitted in our hospital because of tumor mass region glandule Bartolini on the left side and woman complained of the elevated tumor marker carcinoembryonic antigen, CEA (16, 2) detected random.

Result(s) We did local wide removal of the tumor. Histopathology confirmed that this is a Bartolini’s gland adenocarcinoma. The tumor was removed in its entirety with healthy edge. CT, MRI of the pelvis were normal and CEA dropped in 1,6. After seven years of primary treatment she had tumor mass on the same place and elevated tumor marker CEA again.

We did hemivulvectomy with lymph node dissection on the left side and radiotherapy with TD 50 Gy on the left part of vulva and in 25 session and TD 45 Gy in 22 session on region inguini. After surgery tumor marker dropped in normal range.

After fifteen years of primary treatment patient had lung metastasis and brain metastasis and she lived for three months.

Conclusion This case which we followed for fifteen years give us many questions: was the first treatment with local wide excision optimal option, what was the best way to treat this patient and how to predict the way of spread of this malignancies and can we prevent metastasis?

Abstract 439 Figure 1

VULVAR CANCER OF RAPID PROGRESSION WITH EVOLUTION TO FAILURE TREATMENT IN PATIENT CARRYING FANCONI ANEMIA

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Introduction/Background Fanconi Anemia is the most frequent cause of bone marrow failure genetically inherited. Patients may have short stature, microthymiais, skeletal deformities, spots of coffee with milk and cardiac, renal and urinary malformations. One third of the cases are asymptomatic and the presence of pancytopenia is observed as an isolated manifestation.Carriers have 50 times greater predisposition for cancer of the head and neck, esophagus, gastrointestinal tract and vulva. Regarding to head and neck and gynecological squamous cell carcinoma, this incidence is up to 500 times higher than general population, and clinical presentation are more aggressive and disseminated at the time of diagnosis.

MOLECULAR LANDSCAPE OF VULVAR SQUAMOUS CELL CARCINOMA: REVIEW OF THE LITERATURE

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Introduction/Background: Molecular landscape and carcinogenesis of vulvar squamous cell carcinoma (VSCC) have been poorly explored and lack a biomarker-driven targeted therapy. In this non-systematic review we aimed to summarize findings of studies exploring molecular landscape of VSCC.

Methodology: Key word search was conducted (PubMed, Scopus) in January 2021, using the terms (“vulvar” or “cancer” or “carcinoma”) and (“molecular” or “genomic” or “mutation”). Observational studies evaluating molecular alterations in VSCC were deemed eligible. Pre-specified data were extracted from the selected articles, including the number of samples analyzed, DNA sequencing technique, number and frequency of identified mutations, HPV prevalence and prognostic data.

Result(s): Fourteen studies published between 2005 and 2020 were identified, including a total of 747 VSCC samples. Selected studies were highly heterogeneous in terms of DNA sequencing and HPV testing strategies and included small samples. Twelve studies performed next generation sequencing (NGS), nine of whom used targeted approach, two used ples. T welve studies performed next generation sequencing and HPV testing strategies and included small sam-

Abstract 449 Table 1 Overall frequency of most studied mutations in the included studies

<table>
<thead>
<tr>
<th>Gene</th>
<th>N</th>
<th>Number of VSCC assessed</th>
<th>Overall frequency</th>
<th>Frequency range</th>
<th>Number of articles</th>
</tr>
</thead>
<tbody>
<tr>
<td>TP53</td>
<td>387</td>
<td>711</td>
<td>54,43%</td>
<td>33-79%</td>
<td>13</td>
</tr>
<tr>
<td>PIK3CA</td>
<td>112</td>
<td>711</td>
<td>15,75%</td>
<td>0-34%</td>
<td>13</td>
</tr>
<tr>
<td>HRAS</td>
<td>60</td>
<td>677</td>
<td>8,86%</td>
<td>0-28%</td>
<td>11</td>
</tr>
<tr>
<td>CDKN2A</td>
<td>156</td>
<td>610</td>
<td>25,57%</td>
<td>6-36%</td>
<td>9</td>
</tr>
<tr>
<td>PTEN</td>
<td>26</td>
<td>646</td>
<td>4,02%</td>
<td>0-6%</td>
<td>9</td>
</tr>
</tbody>
</table>

Discussion: Selected studies were highly heterogeneous in terms of DNA analysis, DNA sequencing technique, number and frequency of identified mutations, HPV prevalence and prognostic data. The most frequently studied and mutated genes were TP53 and CDKN2A, followed by PIK3CA, HRAS and PTEN (table 1). Evidence on genomic differences between HPV-associated and -independent VSCC is particularly scarce and variable between studies. Only a single, targeted NGS study showed a significant difference between HPV-associated VSCC more frequently involves PI3K/AKT/mTOR pathway, HRAS, PIK3CA, KMT2D, PTEN and FBXW7 mutations. On the other hand, HPV-independent VSCC involve alterations in TP53, CDKN2A, CCND1. The prognostic role of molecular alterations in VSCC was assessed in seven articles, with discordant results. Some articles suggest that TP53 alterations are associated to worse prognosis in patients with VSCC, particularly when combined with PIK3CA, HRAS or CDKN2A.

Conclusion: Limitations and heterogeneity of available molecular series contribute to a limited view of the molecular landscape of VSCC. Prognostic or therapeutic roles of identified mutations and pathways in VSCC remain to be elucidated. Large-scale, genome or exome-wide studies with robust HPV testing are necessary to expand the knowledge on molecular landscape in VSCC.