In 2 patients the FE-1 was repeated; in 1 when treatment failed and FE-1 was still <100 ug/l (due to bile salt malabsorption), the other following treatment for coeliac disease and microscopic colitis (FE-1 147ug/l then normal). There was no difference in symptoms (steatorrhoea, diarrhoea, weight loss, abdominal pain) between the groups.

**Conclusion** This study shows that clinicians need to be aware that even in patients with FE-1 less than 100ug/l, the cause may be non-pancreatic in origin. FE-1 becomes a less reliable diagnostic tool in moderate to mild PEI parameters. FE-1 should be repeated if symptoms do not improve with pancreatic enzyme replacement. Symptoms may not be helpful in distinguishing pancreatic from non-pancreatic causes of low FE-1.

**Disclosure of Interest** None Declared

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**REFERENCES**


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**Abstract PTU-174 Table 1**

<table>
<thead>
<tr>
<th>Severe, No. (%)</th>
<th>Moderate, No. (%)</th>
<th>Mild, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High clinical likelihood supported by imaging</td>
<td>12 (52.2)</td>
<td>3 (16.7)</td>
</tr>
<tr>
<td>Clinically pancreatic cause no imaging/no evidence on imaging</td>
<td>7 (30.4)</td>
<td>4 (22.2)</td>
</tr>
<tr>
<td>Other diagnosis</td>
<td>4 (17.4)</td>
<td>6 (61.1)</td>
</tr>
</tbody>
</table>

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**Disclosure of Interest** None Declared

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**Abstract PTU-175**

**QUADRUPLE, CLINICAL, RADIOLOGICAL, CYTOTOLOGICAL AND BIOCHEMICAL ANALYSIS OF Pancreatic Cystic Lesions ARE NECESSARY PRIOR TO THERAPEUTIC PLANNING**

**Introduction**

Distinguishing benign from malignant or pre-malignant pancreatic cystic lesions is essential when formulating the surgical therapeutic strategy. Lack of a well-defined pre-operative predictability criteria makes therapeutic planning challenging.

**Aims**

To study the correlation between pre-operative morphological and biochemical features of resected pancreatic cystic lesions and predictive power of these features in relation to biological behaviour and final histology.

**Methods**

We systematically reviewed the literature to identify the relevant variables that are used to predict the nature of pancreatic cystic lesions and aid therapeutic planning; this was followed by designing a template encompassing all these variables to collate data of resected pancreatic cystic lesions from two centres. We collated clinico-pathological and biochemical data, pre-operative CT, MRI, EUS, PET CT, FNA analysis and final post-operative pathology reports.

**Results**

63 patients with pancreatic cystic lesions were identified; 3 were drained endoscopically out of which two were pseudocyst and 1 was abscess,12 underwent resection, 3 were serous, 1 mucinous, 1 IPMN, 4 ductal adenocarcinomas, 1 endocrine neoplasm, 1 pseudocyst with abscess, one patient’s final histology results was missing. Three patients had neoplastic cells on FNAC, 2 patients had FNAC results suspicious for neoplasm, 26 were reported to have benign findings at EUS and FNAC and managed conservatively. 10 had elevated intra-cystic CEA levels, 3 patients had elevated CA 19–9 levels at FNAC. 1 patient was diagnosed having VHL, 1 patient had lymphatic cyst, 1 patient was diagnosed having Giardiasis, 1 patient was stented for palliation, 7 patients were undergoing further definitive treatment, and 1 patient with IPMN had therapeutic ERCP.

**Conclusion**

Our interim results suggest that quadruple assessment including clinical, radiological (CT/PET/MRI/EUS), FNAC and biochemical analysis is necessary prior to therapeutic planning.

**Disclosure of Interest** None Declared.