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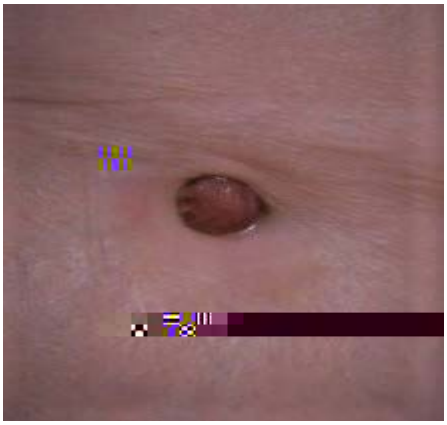

Figure 1:

in ammation, such as in pancreatitis and squamous cell carcinoma, these cells can be seen [4].

e origin of pancreatic SCC remains uncertain, but there are however some hypotheses [2, 4, 5].

- A primitive cell capable of di erentiating both into squamous or glandular carcinoma and which undergoes a malignant modi cation.
- A squamous metaplasia of the ductal epithelium undergoing a malignant transformation following repeated episodes of pancreatitis [6].
- A variant of adenosquamous carcinoma in which the glandular component of cancer has disappeared while the squamous component has persisted [6].
- A pre-existing adenocarcinoma undergoes a squamous modi cation
- An atypical squamous cell that proliferates without undergoing any malignant modi cation.
- Squamous metaplasia of the pancreatic ducts in the presence of chronic in ammation, at the origin of squamous di erentiation [7].

Current clinical data are limited to isolated cases like ours, or too small series. e English literature, for example, found only 61 cases of pure pancreatic SCC (meaning without glandular component) until 2004.

Due to its rarity, little work has been able to identify whether it was a distinct pathological entity, an under-sampled primary adenosquamous carcinoma, or metastasis from an occult squamous cell carcinoma [3]. is last case being the most frequently encountered, a paraclinical morphological and metabolic assessment makes it possible to eliminate a primary occult localization with squamous di erentiation having metastasized to the pancreas.

Unlike the mutations of pancreatic ADK that are commonly known, data on SCC-speci c mutations is limited. We note, for example, the hypothesis of a BRCA-2 exon 15 germline mutation in a patient with locally advanced SCC of the pancreas reported by Schultheis et al. [1] and in whom a BRCA-2 germline mutation was detected, given the history of ovarian and breast cancer in his parents in the rst degree.

Besides this case, a family history of pancreatic cancer has rarely been reported. e clinical presentation of pancreatic SCC is no di erent from other pancreatic cancers. e average age of discovery is 61.9 years with a male predominance. Commonly reported symptoms include anorexia, weight loss, and abdominal pain with back radiation [8]. Depending on the anatomical site of the tumor, obstructive jaundice or pyloric stenosis may also be observed, but much more rarely [9].

Some clinical presentations are more unusual, sometimes with fever, fatigue, epigastric mass, hyperglycemia, or diabetes. Exceptionally, pancreatic SCC causes digestive hemorrhage (hematemesis or melena) responsible for an anemic syndrome, due to the contiguous invasion of the stomach and/or duodenum [10].

No tumor marker is speci c for its diagnosis. However, tumor markers (CA-19.9 and ACE) are elevated in metastatic disease. In a few trials, the SCC antigen has been used to monitor response to treatment or for detection of recurrence [11]. Certain biological reactions can be observed, without being speci c. Among them, there is a leukemoid reaction as reported by Erica et al. [9].

Pancreatic imaging, particularly computed tomography (CT), has an essential role in tumor diagnosis and staging. On imaging, pancreatic SCC has two speci c characteristics: improvement in tumor contrast and angiographic blush pattern [7].

Regarding the location of the tumor, a pooled analysis published in 2016 reported that more than 50% of cases were located in the cephalic region, 21.6% in the caudal level, and 19.5% with multifocal involvement, and 5,9% of cases at the corporeal level [8].

Metabolic imaging has a primary interest in the diagnosis of this tumor. In fact, it has a dual role: it can detect metastatic locations earlier than standard imaging, and above all, it excludes the existence of a primary site other than the pancreas. Histological diagnosis with immunostaining can be performed with an imaging-guided biopsy. Optionally, ne-needle aspiration guided by endoscopic ultrasound may be proposed. Finally, a laparoscopic biopsy remains an alternative but is very little used.

Pancreatic SCC is usually diagnosed at a locally advanced or even metastatic stage [12]. As a result, curative resection is only possible in less than a third of patients, and in this case, overall survival (OS) is improved [8]. Pancreatic SCC is more aggressive than ADK and has a poorer prognosis.

Regarding the management of this tumor, no therapeutic consensus has been established [12]. We only have more or less large series and a few cases that have proposed di erent treatment protocols in order to identify the protocol providing the best bene t in OS [12]. e results were able to rst identify the factors of poor prognosis, which are essentially age and stage of the disease. Indeed, an age over 70 years and stage IV disease is associated with a poor prognosis with a survival of 1.5 months in the absence of treatment. Surgical resection of the primary tumor is associated with longer survival in localized disease (stage I-II), (21 months for the surgery arm versus 5 months for the arm without surgery), regardless of the tumor site. Stage III presents a particularity because the surgery does not o er signi cant bene t in OS with a p=0.32.

However, postoperative chemotherapy/radiotherapy in the localized disease did not show a signi cant bene t in OS (21 vs 24 months) and this is probably because the main protocols proposed in the series are those proposed in current practice for the treatment of pancreatic ADK. Regarding locally advanced and metastatic disease, several multidrug therapy protocols in particular, based on gemcitabine or a 5-Fluoro-uracil combination and platinum salts have

been proposed [13]. But with the poor results of OS ranging from 5 to 8 months depending on the series [14].

In our case, our patient received palliative chemotherapy according to the Capecitabine-Cisplatin protocol, with a very rapid progression of the disease and a deterioration of the general condition.

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Pancreatic SCC is an extremely rare entity. Apart from a few morphological characteristics, its clinical presentation is the same as that of ADK.

Generally, the prognosis is poor when surgical excision is impossible. Currently, only stage I-II surgery appears to be an e ective weapon, especially if combined with adjuvant radio chemotherapy. However, the diagnosis is too o en made at an advanced stage, and curative treatment is rarely o ered. e possibility of a hereditary mutation could be responsible for a familial formTw 9 0e5 to cf1nETj0.46165w 1.575 -1.83 Td(GeFurerapespors chs raneed st)0eageblisa vetation c excor