

Case Report

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Collision of Ductal Adenocarcinoma and Sarcoma Tumor of the Pancreas: A Case Report

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Abstract

We describe a clinical case of a 64-year-old woman with the diagnosis of obstructive jaundice due to a lesion located on the uncinate process of pancreas, with the final anatomy-pathological diagnosis of pancreatic carcinosarcoma, a rare subtype of pancreatic cancer with limited clinical and pathologic data reported in the literature.

Case report

A 64-year-old woman is referred from another health center referring epigastric pain and jaundice, caused by a lesion greater than 2 centimeter located on the uncinate process of pancreas, detected on an imaging test, carried out at the referral center (Figure 1). Her previous medical history included smoking habit and hypothyroidism.

The liver function test revealed a high Total Bilirubin (TB) serum level of 5.20 mg/dl (with direct bilirubin (DB) 4.10 mg/dl and indirect bilirubin (IB) 1.10 mg/dl); aspartate transaminase (AST) 49 U/L, alanine transaminase (ALT) 46 U/L; gamma-glutamyltransferase (GGT) 297 U/L; alkaline phosphatase (ALP) 501U/L and lactate dehydrogenase (LD) 226 U/L.

An Endoscopic Retrograde Cholangiopancreatography (ERCP) is performed detecting a blockage over the distal portion of the common bile duct with a biopsy sample taken, associated to an upper gastrointestinal bleeding (UGIB) as a complication of the procedure. Homeostasis is achieved with the endoscopic injection of adrenaline, requiring the patient a blood transfusion.

The pathology report informed about the presence of malignancy, probably attributable to a pancreaticobiliary adenocarcinoma.

With the diagnosis, aforementioned, a cephalic pancreaticoduodenectomy (Whipple procedure) was performed, being discharged the patient at the seventh day without surgical complications. However, the patient suffered an episode of atrial fibrillation, during the post-operative, being properly treated.

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Figure 1: Preoperative CT. 29.1x22.9 mm lesion to the head-uncinate process of the pancreas. Biliary stent.

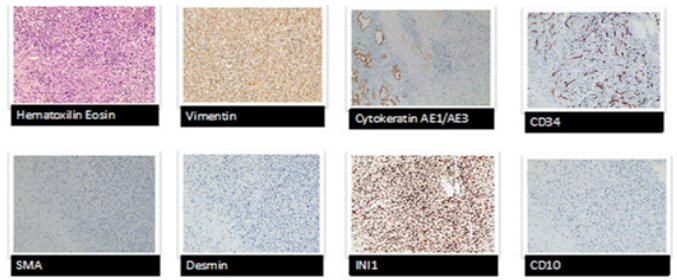


Figure 3: Sarcoma. Pathological Characteristics and immunohistochemical features.



Figure 4: CT follow-up. Pancreatogastrostomy.

Pathological examination and Immunohistochemistry (IHC) analysis

The pathological examination revealed a pancreas confined tumor with free resection margins, lymphovascular and perineural invasion, and fourteen benign lymph nodes (0/14). The final diagnosis was a pancreatic carcinosarcoma.

Microscopically the tumor was constituted by a morphological and immunophenotypical components with no similarities between them. The carcinoma component was constituted by a moderately differentiated ductal adenocarcinoma, which express cytokeratin AE1/AE3 and being negative for vimentin (Figure 2).

The sarcomatous component (Figure 3) was constituted by tumoral cells with a diffuse architectural pattern, a marked nuclear atypia and pleomorphism, the presence of multinucleated giant cells and numerous mitotic figures, with vimentin expression and being negatives for cytokeratin AE1/AE3. There is no expression for CD 34, smooth muscle actin and desmin, besides no angiomatous and muscular differentiation. There was not loss of INI1 expression (the loss of INI1 expression is present on the sarcomatous undifferentiated carcinoma). The CD 10 was negative, predicting a bad prognosis in case of been positive, according to experts.

During the oncological follow up the patient received adjuvant therapy with eight cycles of Gemcitabine, and after three years the patient is free of disease (Figure 4).

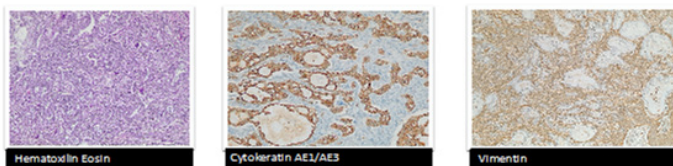


Figure 2: Adenocarcinoma. Pathological Characteristics and immunohistochemical features.

Discussion and conclusion

The collision tumors are very rare. The coincidence at the diagnosis of various tumors at a time could be frequent, being synchronous when the second primary cancer is diagnosed within 6 months of the primary cancer and metachronous when the second primary cancer is diagnosed more than 6 months after the diagnosis of the first primary cancer. Nevertheless, a collision tumor is a neoplastic lesion comprised of two or more distinct cell populations that maintain distinct borders.

To properly establish the histological diagnosis of carcinosarcoma, as the last WHO [1] classification guide of pancreatic tumors state, require a biphasic pattern, without a mix or transition area, with a sarcomatous component (including or not heterologous elements) and an epithelial morphology component, which could be an adenocarcinoma. Each component should represent the 30% of the tumor to properly consider it as a carcinosarcoma. Each component is immunophenotypically similar to its typical lineage, with a divergent expression on each component.

The sarcomatoid carcinoma should be considered on the differential diagnosis, which unlike the carcinosarcoma, is an undifferentiated carcinoma that can harbor fusiform, pleomorphic and multinucleated giant cells, resembling the sarcoma, but lacking of the typical biphasic pattern of the carcinosarcoma. Immunophenotypically, could express cytokeratin AE1/AE3 and loss of INI1 expression.

Most of these tumors could be located on the uterus, but are no often present on others organs as ovaries, breasts, lungs, pros-

tate, urinary and digestive tracts. Those tumors diagnosed in the pancreas are even more rare, and any publications regarding this topic in the literature is limited [2].

The pancreas carcinosarcoma is a rare subtype of pancreatic cancer with limited clinical and pathologic data reported in the literature with a bad prognosis [3]. It has an epithelial component which is usually an adenocarcinoma and a sarcomatous component of fusiform cells. Each one with different immunohistochemical and molecular features related to their differentiation lines [4].

The microscopic semblance is variable. The carcinoma component ranges from well to poorly differentiated ductal like adenocarcinoma. The sarcomatous component exhibits pleomorphic and spindle morphologies resembling mesenchymal tumour [5].

There have been a controversy around these tumors pathogenesis from long time. The neoplastic components can develop as two adjacent tumor lineages, so carcinoma and sarcoma collide. This could be the theory of tumoral collision. Another theory points out to an origin sharing from a pluripotent cell that enable the development of two different histological components [6]. The genetic predisposition, the advanced age, the environmental carcinogens exposure, previous radiotherapy or chemotherapy and the immunosuppression, among others, are part of the factors that increase the risk of the tumor collision [7].

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