An 83-year-old woman came to our institute with abdominal pain, tomographic evidence of multiple pancreatic cystic lesions, and abnormal thickening of the pyloroduodenal region. Magnetic resonance imaging showed multiple cysts in the pancreatic body, isthmus, head, and uncinate process, some with clear communication with the pancreatic duct (● Fig. 1), and a parietal thickening of the duodenum with contrast enhancement, in very close proximity to a cystic lesion in the pancreatic isthmus (● Fig. 2a). Endoscopic ultrasound (EUS) showed a multiloculated, septated cystic lesion in the pancreatic isthmus, with a hypoechoic pericystic lesion invading the duodenal bulb (● Fig. 2b). Gastroscopy confirmed a villous lesion in the duodenal bulb, involving the infero-posterior wall (● Fig. 2c). EUS showed that the intraduodenal lesion was heterogeneous with a solid-cystic aspect (● Fig. 2d). Biopsy of the duodenal lesion confirmed duodenal wall invasion by a malignant intraductal papillary mucinous neoplasm (IPMN) (● Fig. 2e, f). EUS fine-needle aspiration cytology of a pancreatic cyst in the uncinate process showed atypical cells with severe dysplasia, strongly suggestive of malignant IPMN (● Fig. 3), as previously reported [1]. This report highlights a case of a degenerating multifocal IPMN invading the duodenal bulb. Benign and malignant IPMNs can extend to surrounding organs (duodenum, common bile duct, stomach) [2–5]. In benign lesions, mucus produced by tumor cells is compressed and exerts pressure, inducing ischemia, atrophy, and disappearance of the gastroduodenal or bile duct wall, with final penetration by the IPMN; these lesions are called the “automatic type.” On the other hand, malignant lesions can directly infiltrate surrounding organs and are called the “invasive type” [2–3]. The intraluminal lesion of an invading IPMN can assume, at EUS, the same solid-cystic pattern as the malignant pancreatic lesion. It is also worth bearing in mind that there can be synchronous multifocal degeneration of IPMNs, as evidenced in our case by the presence of a malignant pancreatic isthmus IPMN invading the duodenal bulb, and a cytology-proven malignant IPMN in the uncinate process. Both these aspects should be taken into consideration for diagnostic purposes and therapeutic strategies.

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References