



Management of Mixed-Type Intraductal Papillary Mucinous Neoplasm

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Keywords

- Intraductal papillary mucinous neoplasm
- Main-duct-involved IPMN
- Mixed-type IPMN
- Pancreatic mucinous cystic lesion
- Pancreatic cyst

Key points

- Radiologic diagnosis of mixed-type IPMN is inaccurate overestimating main pancreatic duct involvement in 20% of patients.
- Radiologic diagnosis of branch-duct IPMN is inaccurate underestimating main pancreatic duct involvement in up to 30% of patients.
- Multiple pathologic definitions of mixed-type IPMN exist.
- Classification of mixed-type IPMN as a subcategory of main-duct IPMN may be an oversimplification.
- Mixed type-IPMN are a heterogeneous group of lesions with various phenotypes and thus their management should be tailored according to the patient and biology of the lesion.

Disclosure Statement: Dr C.M. Schmidt is founder of B9, Inc. B9 seeks to identify diagnostic biomarkers for benign conditions. He also consults for Redpath Integrated Pathology, Asuragen, and Mauna Kea Technologies, all of which have technologies that aid in the management of patients with pancreatic cysts. Dr A.M. Roch has nothing to disclose.

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INTRODUCTION

In 1982, Ohashi and colleagues [1] described four cases of a rare pancreatic entity they called “mucin-producing cancer.” It affected the main pancreatic duct and produced excessive quantities of mucus, which filled and distended the pancreatic ductal system. This type of pancreatic tumor, later in 1996 named intraductal papillary mucinous neoplasm (IPMN) [2], is believed to account today for up to 70% of all cystic neoplasms of the pancreas. Reasons for the IPMN epidemic are unknown, but likely result from increased awareness, recognition, and detection with improved imaging resolution. IPMN are classified into branch-duct (BD-IPMN), main-duct (MD-IPMN), and mixed (MT-IPMN) types according to the radiographic topography of ductal involvement [3,4]. Patients who undergo surgical resection are definitively classified according to the pathologic topography of ductal involvement. It is well established that all IPMN types harbor a real but variable risk of malignant transformation following a stepwise progression pattern similar to that of adenomatous polyps in colonic cancer [5,6]. Over the last 30 years, more than 2000 articles on IPMN have been indexed in the PubMed/Medline database. Few papers, however, specifically focus on MT-IPMN, which account for 28% to 41% of all IPMN in surgical series [7–9]. Historically, the management of MT-IPMN has been lumped into the management of MD-IPMN because of the presence of main pancreatic duct involvement and a presumed similar risk of malignant progression [10,11]. MT-IPMN is unable to be accurately established preoperatively in up to 30% of cases when compared with the gold standard, surgical pathology. Even in pathologically proven MT-IPMN, main-duct involvement represents a spectrum from minimal to extensive, which positively correlates with the incidence of higher grades of IPMN dysplasia. Thus, the classification and optimal management of these lesions is evolving [12].

DEFINITION

MT-IPMN is characterized by IPMN involvement of both the main pancreatic duct and the branch ducts of the pancreas. Criteria used to differentiate MT-IPMN from isolated MD-IPMN and BD-IPMN are not uniform in the literature. Most studies use the results of imaging studies alone [10,11]. Following surgery, patients are definitively classified as MT-IPMN only if there is pathologic involvement of both main and branch ducts.

Radiographic

MT-IPMN is diagnosed with a reasonable degree of certainty based on cross-sectional (multidetector computed tomography [CT], magnetic resonance cholangiopancreatography [MRCP]) and/or endoscopic (endoscopic ultrasound, endoscopic retrograde cholangiopancreatography, pancreatic ductoscopy) imaging. It is radiographically defined, according to the 2012 revised International Consensus Guidelines, as the association of segmental or diffuse main pancreatic duct dilation of greater than or equal to 5 mm (without any non-IPMN cause of obstruction) and single/cluster or multiple pancreatic “cysts” greater

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