LETTER

IPMN and Parathyroid Adenoma: An Interesting Association

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Dear Sir:

Intraductal papillary mucinous neoplasm (IPMN) has become the second most common indication for pancreatic resection [1]. The adenoma-carcinoma sequence of IPMN is well documented. Nearly all IPMNs express DPC4 and several other molecular abnormalities believed to be responsible for the malignant transformation. K-*ras* mutations are present in nearly half of the patients. Other mutation and gene expression were identified in IPMN patients [2]. Recent study by Fritz *et al*. [3] identified several recurrent cytogenetic alterations characteristic of moderate- and high-grade IPMNs that differed from those found in ductal adenocarcinoma. The cause of IPMNs is ill defined. Familial and genetic predisposition of IPMNs is implied by reports of this disease association with inherited syndromes such as Peutz-Jeghers, familial adenomatous polyposis, and familial pancreatic cancer. Patients with IPMN are at increased risk of developing extra pancreatic malignancies, the most common of which are gastric adenocarcinoma and colorectal cancer. These malignancies may occur in a synchronous or metasynchronous sequence.

Primary hyperparathyroidism is the third most common endocrine disorder. Parathyroid adenoma is responsible for more than 80% of the cases of primary hyperparathyroidism [4] and it may be associated with multiple endocrine neoplasia (MEN) type 1 and type 2A. Approximately 25-40% of sporadic parathyroid adenomas have loss of heterozygosity at 11q13 the site of the MEN1 gene [5]. Nevertheless and despite the fact that MEN type 1 syndrome is comprised of pancreatic tumors, mainly of the endocrine variants, we could not find any report in the English literature on the association of IPMN and parathyroid adenomas. Recently we encountered two patients who suffered both pathologies. A 64-year-old male and a 65-year-old female underwent subtotal pancreatectomy for the diagnosis of IPMN. The first patient has undergone parathyroidectomy due to parathyroid adenoma 6 years prior to the current operation and right hemicolectomy for adenocarcinoma three years prior to the pancreas surgery. The second patient was also diagnosed with primary hyperparathyroidism and parathyroid adenoma but refused surgery. The association of these two pathologies is of utmost interest. There are several possible implications of such an association. The first patient did not have any genetic testing performed and the second patient had negative testing for BRCA genes. However, it might well be that one or both of them carries one of the genes described in MEN syndromes, especially type 1. Moreover as IPMN emerged as a distinct clinical entity in the past few years with rising interests it might well be that it is related to other types of neoplasia. As such there is a possibility of existence of yet undescribed neoplastic syndromes.

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