LETTERS TO THE EDITOR

Endoscopists Should Be Aware of the Occurrence of Post-ERCP Pancreatitis after Prophylactic Pancreatic Duct Stenting in Cases of IPMN without a Dilated Pancreatic Head Duct

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The Author Replies I deeply appreciate your interest in our article regarding the risk factors for post-ERCP pancreatitis in patients at high risk who have undergone prophylactic pancreatic duct stenting (1). It is my pleasure to answer your questions.

Since our study was conducted in a retrospective multicenter fashion, data collection and interpretation were extremely limited compared with those of a prospective study. Intraductal papillary mucinous neoplasm (IPMN) is not generally recognized as having a high risk of pancreatitis (2-5). In our study, prophylactic pancreatic duct stenting was performed in 32 patients with IPMN. Seven patients developed post-ERCP pancreatitis (22%). There should have been many patients with IPMN who did not undergo pancreatic duct stenting during the study period. Unfortunately, detailed data regarding patients without stenting were not available. Since the endoscopists selected the patients with IPMN at high risk of post-ERCP pancreatitis, a high frequency of this particular complication in patients with IPMN is acceptable.

The extent of a pancreateogram is important for predicting an injury of the pancreatic parenchyma. Acinarization is thought to be a risk of post-ERCP pancreatitis. As the endoscopists who participated in this study are highly experienced in ERCP, I expect that they took the extent of the pancreateogram into account and avoided excessive pancreateogram during ERCP. The goal of ERCP in cases of IPMN, is not identification of the communication of a lesion with the pancreatic duct. The existence of mucin in the pancreatic duct itself is an important finding for the diagnosis of IPMN, verifying a communication of a lesion with the pancreatic duct. Moreover, the main purpose of ERCP for IPMN is to perform pancreatic juice collection or intraductal US. Therefore, it may be inferred that the extent of the pancreateogram for IPMN is possibly the same as that for other diseases.

The diameter of the pancreatic duct and the existence of a patulous orifice of the papilla of Vater are determined not only by the viscosity of mucin but also by its amount or by the function of the sphincter of Oddi’s muscle. Mucin is usually observed even in IPMN patients without a dilated pancreatic duct. I would like to emphasize that the viscosity of such mucin is higher than that of normal pancreatic juice, which can result in the risk of stent occlusion. Although mucin in patients with a dilated pancreatic duct and a patulous orifice can also occlude a pancreatic duct stent, pancreatic juice can flow along the occluded stent. I speculate that lack of such a salvage in IPMN patients without a dilated pancreatic duct plays a significant role in the development of PEP.

Data regarding the frequency of guidewire insertion into side branches of the pancreatic duct were not available in this study. Patient selection for pancreatic duct stenting due to difficult cannulation was based on the criteria of each participating facility considering the reported conditions in the literature such as pancreatic duct guidewire placement. Despite several limitations, the findings of our study can contribute to the determination of appropriate indications for prophylactic pancreatic duct stenting during ERCP. IPMN of the pancreas without a dilated pancreatic head duct is a possible risk factor for post-ERCP pancreatitis after prophylactic pancreatic duct stenting. Further investigations regarding the indications for pancreatic duct stenting in patients with IPMN after the procedure are necessary.

The authors state that they have no Conflict of Interest (COI).  
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References