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Portal vein stent insertion for symptomatic malignant portal vein stenosis

내과, 가천대학교 길병원

*남태현, 박인근, 김정호

Background/Aims: Malignant tumors around portal vein can cause perihepatic portal vein (PV) stenosis. Severe malignant PV stenosis lead to PV hypertension and related complications. Such complications deteriorate quality of life and disturb active anticancer treatment. PV stent insertion has been used in benign or malignant PV stenosis since 1990s, and it can be useful for reducing portal hypertension related symptoms. This study was to estimate efficacy and safety of PV stent insertion for symptomatic malignant PV stenosis patient. **Methods:** We found 12 consecutive patients who had symptomatic malignant PV stenosis and underwent PV stent insertion from January 2016 to April 2018. PV stents were inserted percutaneous transhepatic approach. PV pressure gradient across stenosis site was measured before and after procedure. We retrospectively reviewed medical record to obtain procedure related complications, relief of malignant PV stenosis related symptoms, and survival after PV stent insertion. **Results:** Median age was 65 (range 32-76), and 50% was male. There were 6 pancreatic cancer, 3 bile duct cancer, and 3 other cancers (1 ampulla of Vater cancer, 1 duodenal cancer, and 1 stomach cancer, respectively). PV hypertension related symptoms were ascites (5 patients, 42%), diarrhea (3, 25%), varix formation (2, 17%), hepatic encephalopathy (1, 8%), chyloperitoneum (1, 8%), and portal vein thrombosis proximal to stenotic site (1, 8%). PV stent insertion and complete expansion of stent was succeeded in 11 out of 12 patients (92%), and PV stenosis related symptoms were restored in most patients (92%, 11/12). There was significant decline in median portal venous pressure gradient. Median pressure gradient before and after stenting were 14.4 mmHg (range 9-28) and 2.5 mmHg (1-23). Post procedural complications included transient pain (6 patients, 50%), hemothorax (1, 8%), in-stent thrombosis (1, 8%), intrahepatic PV thrombosis (1, 8%). With a median follow-up of 24 weeks (95% CI,0-48.1), median survival after stenting was 17 weeks (95% CI, 15.2-18.8) **Conclusions:** Portal vein stent placement for symptomatic malignant portal vein stenosis is a safe and effective.

Table1. Clinical manifestation and findings in 12 patiens

Characteristics	N (%)
Age, median (range), year	65 (32-76)
Sex	
Male	6 (50%)
Female	6 (50%)
Primary tumor site	
Pancreatic cancer	6 (50%)
Bile duct cancer	3 (25%)
Ampullar of vater cancer	1 (8%)
Duodenal cancer	1 (8%)
Stomach cancer	1 (8%)
Disease status	
Locally advanced	1 (8%)
Recurrent	9 (75%)
Initially metastatic	2 (17%)
Clinical symptom	
Ascites	5 (42%)
Diarrhea (portal hypertensive enterocolopathy)	3 (25%)
Varix	2 (17%)
Hepatic encephalopathy	1 (8%)
Chyloperitoneum	1 (8%)
Portal vein thrombosis proximal to stenosis	1 (8%)
Failure of complete expansion of stent	1 (8%)
Resolution of symptom	11 (92%)
Complication	
Pain	6 (50%)
Hemothorax	1 (8%)
In-stent thrombosis	1 (8%)
Intrahepatic PV thrombosis	1 (8%)
Pressure gradient, before stenting, median (range), mmHg	14.5 (9-28)
Pressure gradient, after stenting, median (range), mmHg	2.5 (1-23)

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CASE REPORT: Cardiac tamponade as the initial presentation of parapharyngeal space tumor

강동경희대학교병원 내과학교실

*전홍기, 김정아, 장혜정, 어완규

Introduction: Malignant pericardial effusion rarely progresses to cardiac tamponade. Also, cardiac tamponade occurring as the first presentation of malignancy appears to be uncommon. When occurs, it is mainly from the lung, breast and the neoplasms of the lymphoreticular system. Here we report an extremely rare case of cardiac tamponade that is caused by parapharyngeal space(PPS) tumor. **Case:** A 59-year-old man was admitted to our hospital with 5 days of dyspnea. He had medical history of hypertension and 30 pack-year history of smoking. On examination, Jugular vein distension, cardiomegaly on chest x-ray and electrical alternans in ECG were observed. Echocardiography revealed a large amount of pericardial effusion and diastolic collapse of right atrium and right ventricle, therefore, emergent pericardiocentesis was performed. After 1.4L of bloody pericardial effusion was drained, dyspnea and tamponade features were improved. Several tests were performed for differential diagnosis of pericardial effusion. Tumor markers CEA, CA19-9 and CA125 were elevated and adenocarcinoma cells were observed on pericardial fluid cytology. Further investigations to identify the primary origin of malignancy were done: chest-abdomen-pelvis CT, gastroscopy and bronchoscopy. Chest CT revealed enlargement of both axillary lymph nodes, however, no other specific findings were found on abdomen-pelvis CT, gastroscopy or bronchoscopy. For these reasons, we additionally performed whole body PET/CT and neck CT. PET/CT images revealed hypermetabolic lesion in both axillary area and hypermetabolic calcified mass in right parapharyngeal space(PPS). At the same site of PPS, 5x3x5cm sized, calcified, solid mass was also observed in neck CT images. So, biopsies were done on axillary lymph node and PPS tumor. Pathologic study of axillary lymph node confirmed metastatic carcinoma and CK7+/CK20- in immunohistochemical stain suggested the origin of salivary gland. In addition, because of the presence of malignant cells in the pathologic study of PPS tumor, we have diagnosed the case as PPS tumor with distant metastases. The patient is currently on chemotherapy with 5-FU and cisplatin.