

Germ cell tumor-related lymphadenopathies caused acute pancreatitis

Chieh-Sheng Lu^{1,2}, Yi-Jen Peng³, Ping-Ying Chang²

¹ Department of Internal Medicine, Kaohsiung Armed Forces General Hospital, Kaohsiung, Taiwan

² Division of Hematology/Oncology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan

³ Department of Pathology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan

A 23-year-old man presented with symptoms of epigastric pain and a 7-kg weight loss over 1 month. The abdominal pain was dull in quality and radiated to the back. Progressive yellowish skin and dark-yellow urine developed 1 week before admission. The patient denied a history of systemic disease or operation. A physical examination showed icteric sclera and local tenderness over the epigastrium without rebounding tenderness. Serum amylase levels of 716 U/l (reference range, 16–108 U/l) and lipase levels of 1144 U/l (reference range, 13–60 U/l) were detected. Initially, acute pancreatitis was considered. Later, an abdominal computed tomography (CT) revealed pancreatic swelling and paraaortic confluent masses (**FIGURE 1A**). Magnetic resonance imaging confirmed swelling of the pancreas with pancreatic duct dilatation due to mass compression (**FIGURE 1B**). The biopsy of masses revealed a poorly differentiated carcinoma. Serum free β -human chorionic gonadotropin (β -HCG) levels of 36.50 ng/ml (reference range, 0–0.1 ng/ml) and α -fetoprotein levels of 2.34 ng/ml (reference range, 0–20 ng/ml) were noted. Immunohistochemical staining of the biopsied tissue was positive for placental alkaline phosphatase (**FIGURE 1C**) and β -HCG (**FIGURE 1D**). A diagnosis of germ cell tumor-related lymphadenopathies caused acute pancreatitis was made. Therefore, the patient received treatment and his abdominal pain was relieved after the first cycle of chemotherapy. An abdominal CT showed a significant improvement 3 months later (**FIGURE 1E**).

In general, the diagnosis of acute pancreatitis requires the presence of 2 of the following 3 criteria: 1) acute onset of persistent severe epigastric pain often radiating to the back; 2) elevation in serum lipase or amylase levels to 3 times or more than the upper limit of normal; and 3) characteristic findings of acute pancreatitis on imaging.¹ Once the diagnosis of acute

pancreatitis is established, the underlying etiology should be determined. There are numerous causes of acute pancreatitis, which can be easily identified in 75% to 85% of patients. In developed countries, obstruction of the common bile duct by stones (38%) and alcohol abuse (36%) are the most frequent causes of acute pancreatitis.^{2,3} Mechanical obstruction of pancreatic duct by tumor is uncommon,⁴ and germ cell tumor-related lymphadenopathies to cause acute pancreatitis is rare. Our patient had no risk factors for acute pancreatitis, and advanced images were necessary to determine the etiology. The abdominal CT revealed paraaortic confluent masses, and we made the final diagnosis on the basis of tissue biopsy with immunohistochemical examination. We should remember about the possibility of lymphomas and germ cell tumors in patients with paraaortic confluent masses, especially in young patients. Prompt identification of the uncommon cause of acute pancreatitis is the key to successful management of the clinical symptoms, especially for the therapy-sensitive disease in young patients.⁵

REFERENCES

- 1 Banks PA, Freeman ML; Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol*. 2006; 101: 2379-2400.
- 2 Lankisch PG, Assmus C, Lehnick D, et al. Acute pancreatitis: does gender matter? *Dig Dis Sci* 2001; 46: 2470-2474.
- 3 Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: An update. *Best Pract Res Clin Gastroenterol*. 2008; 22: 45-63.
- 4 Wang GJ, Gao CF, Wei D, et al. Acute pancreatitis: etiology and common pathogenesis. *World J Gastroenterol*. 2009; 15: 1427-1430.
- 5 Shinagare AB, Jagannathan JP, Ramaiah NH, et al. Adult extragonadal germ cell tumors. *AJR Am J Roentgenol*. 2010; 195: W274-W280.

Correspondence to:

Ping-Ying Chang, MD, Division of Hematology/Oncology, Department of Internal Medicine, Tri-Service General Hospital, National Defense Medical Center, No. 325, Sec. 2, Cheng-Gong Rd., Neihu 114, Taipei, Taiwan, phone: +886 2 87923311, fax: +886 2 87927209;

e-mail: max-chang@yahoo.com.tw

Received: April 17, 2015.

Revision accepted: April 28, 2015.

Published online: April 29, 2015.

Conflict of interest: none declared.

Pol Arch Med Wewn. 2015;

125 (6): 471-472

Copyright by Medycyna Praktyczna,

Kraków 2015

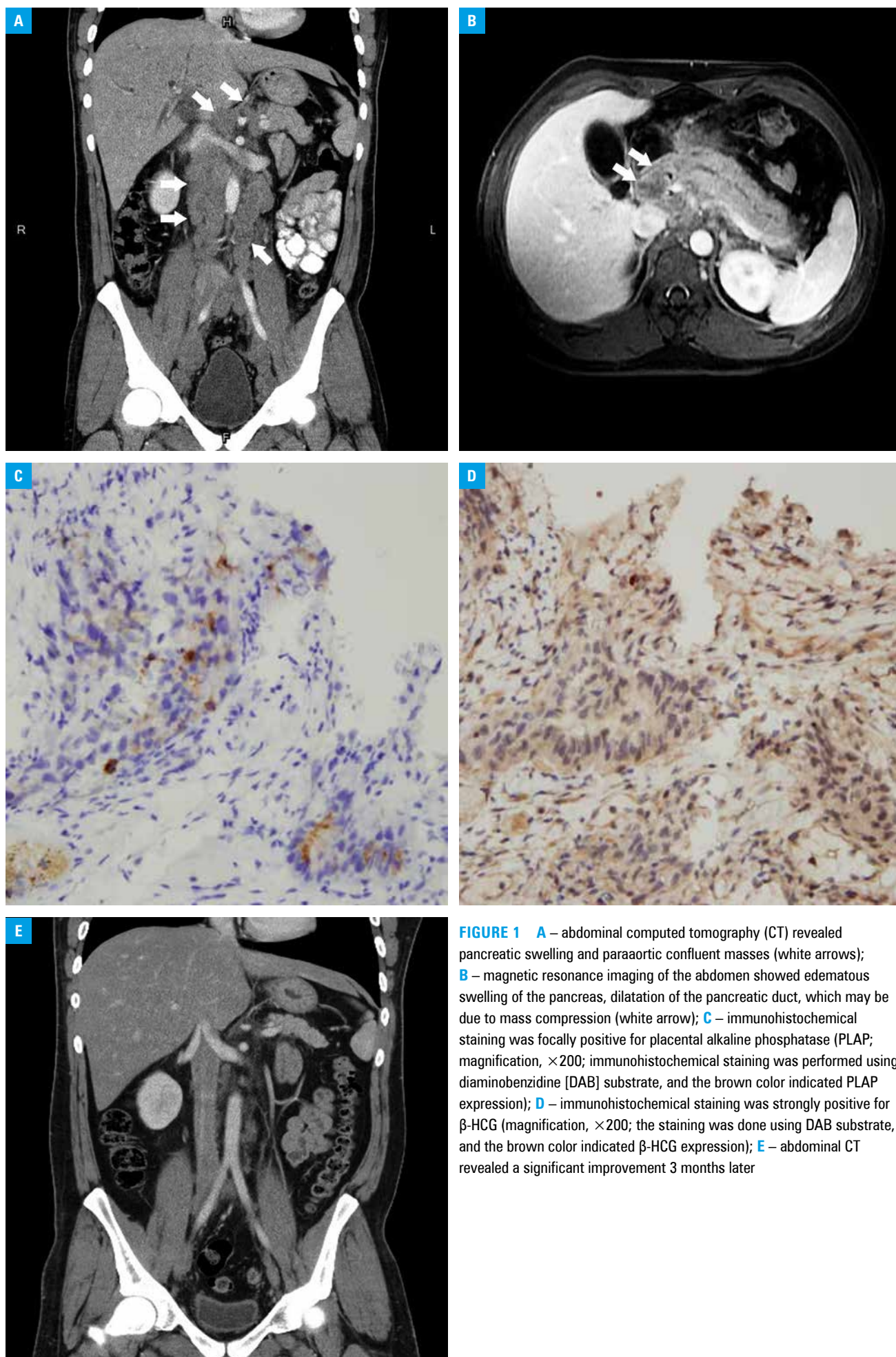


FIGURE 1 **A** – abdominal computed tomography (CT) revealed pancreatic swelling and paraaortic confluent masses (white arrows); **B** – magnetic resonance imaging of the abdomen showed edematous swelling of the pancreas, dilatation of the pancreatic duct, which may be due to mass compression (white arrow); **C** – immunohistochemical staining was focally positive for placental alkaline phosphatase (PLAP; magnification, $\times 200$; immunohistochemical staining was performed using diaminobenzidine [DAB] substrate, and the brown color indicated PLAP expression); **D** – immunohistochemical staining was strongly positive for β -HCG (magnification, $\times 200$; the staining was done using DAB substrate, and the brown color indicated β -HCG expression); **E** – abdominal CT revealed a significant improvement 3 months later