OBJECTIVE RESEARCH

Retrospective evaluation of venous phase contrast-enhanced computed tomography images in patients who developed pancreatic adenocarcinomas after treatment for nonpancreatic primary cancer

### INTRODUCTION

Pancreatic adenocarcinoma is one of the most lethal and difficult to treat malignant tumors.\(^1,2\) Surgical resection is the only curative treatment option; however, most patients have unresectable disease at the time of diagnosis.\(^3\) Furthermore, the survival rate of patients with pancreatic adenocarcinoma is better if small tumors are detected at an early stage.\(^4\) Therefore, early detection and precise diagnosis are very important for the management and prognosis of these patients. Multiphasic contrast-enhanced CT imaging is now considered the primary imaging modality for detection of pancreatic adenocarcinoma.\(^5\) Lu et al\(^6\) have stated that pancreatic phase images obtained beginning 40 sec after administration of contrast material show maximal tumor-to-pancreas contrast. Pancreatic adenocarcinomas usually appear as hypoattenuated lesions compared with...
the normal parenchyma in the pancreatic phase, whereas during the portal venous phase, these tumors are difficult to discern because of reduced contrast between the tumor and pancreatic parenchyma. However, patients with non-pancreatic cancers are often followed up with monophasic contrast-enhanced CT in venous phase timing performed after 70–100 sec from the initiation of intravenous contrast injection.6 Hence, small pancreatic adenocarcinoma could be missed in follow-up patients with other non-pancreatic cancer underwent surgery. Recently, some investigators have reported development of a subsequent pancreatic cancer in patients with non-pancreatic malignancies.2–11 The overall reported incidence of pancreatic cancer associated with other organ malignancies was 1.2–20%.12 Therefore, identification of the venous phase CT findings in early pancreatic adenocarcinomas is important for early diagnosis in patients who have previously been treated for non-pancreatic cancers. In this study, we retrospectively evaluated venous phase CT images that had been acquired during follow-up after treatment of previous nonpancreatic cancers with the aim of identifying incidental findings that suggest early pancreatic adenocarcinoma in such follow-up images.

METHODS AND MATERIALS

Patient selection
This retrospective study was approved by the ethics committee of our institution (Oita university hospital) and the requirement for informed consent was waived. The pathology and radiology databases and electric medical records of our institution were reviewed to identify patients with surgically proven pancreatic adenocarcinomas that had developed during follow-up after treatment of non-pancreatic cancer. Between April 2005 and April 2020, 147 consecutive patients with surgically proven pancreatic adenocarcinomas were identified.

Patients who met the following criteria were included in the study cohort: (a) surgically resected and pathologically diagnosed pancreatic adenocarcinoma; (b) had a previous non-pancreatic malignancy; (c) had undergone at least two venous phase contrast-enhanced CT examination; and (d) had no other pancreatic diseases.

The final study group comprised six patients (one male and five females; median age 69 years; age range, 61–86 years, median body weight 44.5 kg; body weight range, 30–48 kg). These patients’ previous nonpancreatic cancers were as follows: asynchronous triple cancer (colorectal, ureteral, and uterine cancers) (n = 1), colorectal cancer (n = 1), breast cancer (n = 1), gastric cancer (n = 1), malignant melanoma of left lower limb (n = 1) and hepatocellular carcinoma (n = 1). The hepatocellular carcinoma patient had undergone triple-phase contrast-enhanced CT and only the portal venous phase scan had been evaluated. The study cohort comprised only patients who undergone resection of a pancreatic adenocarcinoma that had been diagnosed definitively by pathological examination of the resected specimen.

CT technique
Abdominal CT examinations were performed using a 64-section multidetector CT (MDCT) scanner (Aquilion CX TSX-101A/NA; Toshiba Medical Systems, Tokyo, Japan) or a 320-section MDCT scanner (Aquilion ONE TSX-301A/2A; Toshiba Medical Systems). The scanning parameters used for MDCT were as follows: 120 kVp, 200–400 mAs, rotation time of 0.5 s, pitch of 0.98 (64 detectors) and 0.6 (320 detectors), and section thickness of 1 mm with a 1 mm reconstruction interval. CT images were retrieved through a Picture Archiving and Communication System.

Venous phase contrast-enhanced images on follow-up for non-pancreatic cancer were obtained at 100 sec after starting the contrast-material infusion. A total of 100 ml of contrast-medium (Iopamiron 300; Bayer Schering Pharma, Berlin, Germany) was infused with a power injector at a rate of 3 ml s⁻¹.

Triple-phase contrast-enhanced CT was performed for pancreatic adenocarcinoma staging. For triple-phase contrast-enhanced CT imaging, a total of 100 ml of contrast medium (Iopamiron 370; Bayer Schering Pharma, Berlin, Germany) was infused at a rate of 3 ml s⁻¹ by means of a power injector. After unenhanced images had been acquired, all patients underwent pancreatic, portal venous, and equilibrium phase imaging. Each scan delay was determined using the automatic bolus-tracking method. The average scan delays from the injection of contrast material to the start of pancreatic, portal venous, and equilibrium phase imaging were 44, 75, and 158 s, respectively. The data sets obtained were sent to a computer workstation (Aquarius NetStation v. 1.2; TeraRecon, San Mateo, CA).

Reconstructed axial and multiplanar reconstruction images with 1 mm intervals were obtained using a computer workstation (Aquarius NetStation v. 1.2, Terarecon).

Imaging interpretation
Two radiologists (R.S. and R.T., with 14 and 15 years of experience in abdominal imaging, respectively) reviewed all venous phase contrast-enhanced CT images obtained during follow-up after treatment of nonpancreatic cancer in all six patients. They evaluated the presence or absence of the following findings, which are all reportedly suggestive of small pancreatic adenocarcinomas: (1) pancreatic duct interruption and dilatation; (2) pancreatic parenchymal atrophy; (3) focal hypoattenuated areas; (4) pancreatic cystic lesions; and (5) enhanced rim.13–18 Ductal dilatation was defined as over 3 mm dilation of a main pancreatic duct or increase of more than 1 mm in main pancreatic duct diameter compared with latest surveillance CT. Pancreatic parenchymal atrophy was defined as atrophy of the parenchyma distal to a focal lesion or disproportional atrophy in the absence of any focal pancreatic lesions. Focal hypoattenuated areas were defined as localized areas of hypoattenuation in comparison with the normal pancreatic parenchyma. Time intervals between initial follow-up CT with findings suggestive of small pancreatic adenocarcinomas and latest pre-operative CT and doubling times were calculated from data obtained from hypoattenuated areas in CT images. In all of six cases, imaging findings suggestive of pancreatic adenocarcinoma were recognized on follow-up CTs, prompting triple-phase contrast-enhanced CT for staging of that pancreatic adenocarcinoma. Reconstructed axial and
multiplanar reconstruction images from the staging CT were used to assess the pancreatic adenocarcinomas. Previous studies reported that triple phase (pancreatic, portal venous, and equilibrium phase) contrast-enhanced examination was useful for differentiating pancreatic adenocarcinoma from other pancreatic diseases, especially mass-forming pancreatitis and that biphasic (pancreatic and portal venous phase) contrast-enhanced examination was useful for staging of pancreatic adenocarcinoma. Therefore, triple-phase contrast-enhanced CT was performed for diagnosis and staging of pancreatic adenocarcinoma.

**Histopathological analysis**

The post-operative pathology records of all six patients were retrospectively reviewed by a pathologist (T.D. with 31 years of experience). All pancreatic adenocarcinomas were proven by pathologic examination of surgically resected specimens. Tumor diameter, invasion into the anterior and posterior peripancreatic tissues, portal vein, and superior mesenteric vein, and lymph node metastases were also assessed in all patients.

**RESULTS**

Regarding initial CT findings suggestive of pancreatic adenocarcinoma (Table 1), small (≤10 mm) focal hypoattenuated areas were identified in three of six patients (50%) (Figure 1). Rim enhancement was observed in two of these three patients. Pancreatic duct interruption and dilatation (Figure 2), pancreatic parenchymal atrophy (Figure 2), and a cystic lesion (Figure 3) were identified in two (33%), one (17%) and one (17%) patient, respectively. The cystic lesion was pathologically confirmed to be a retention cyst. Time intervals between initial follow-up CT examination with findings suggestive of pancreatic adenocarcinoma and latest pre-operative CT examination were 6–19 months (median, 14.5 months) and follow-up CT examinations were performed 2–4 times during those intervals (median, 2.5 times). Doubling times as calculated from CT image data were 46–407 days (median, 106 days) in patients with focal hypoattenuated areas. The pancreatic adenocarcinomas in resected specimens were of diameter 12–55 mm (median, 18 mm). Local tumor invasion was detected pathologically in all six patients, comprising anterior tissue invasion in three patients, retroperitoneal tissue invasion in five, neural plexus invasion in one, and duodenal invasion in one. Lymph node metastases were detected in three patients (50%); however, no distant metastases were identified in any of the patients. No focal lesions were identified on venous contrast-enhanced CT images obtained 6 months before pancreatic surgery in the patient with the largest diameter lesion (55 mm).

**DISCUSSION**

In this study, we retrospectively evaluated venous phase contrast-enhanced CT images of six patients with pancreatic adenocarcinomas that had developed during follow-up after treatment for non-pancreatic cancer. We found that several CT findings known to constitute possible early evidence of pancreatic adenocarcinoma (e.g. small focal hypo-attenuated areas with or without rim enhancement, pancreatic duct interruption and dilatation, pancreatic parenchymal atrophy, cystic pancreatic lesions) could have been detected on venous phase contrast-enhanced CT images.
scans performed 6–19 months (median, 14.5 months) before the latest pre-operative CT. In three patients with small hypoattenuated areas on their initial CT images, tumor volume doubling times were 46–407 days (median 106 days).

Pancreatic cancer is associated with certain primary non-pancreatic cancers. Amin et al reported an increased risk of pancreatic cancer after gastric, colorectal, biliary and uterine cancers in patients aged over 65 years. It is expected that the incidence of development of pancreatic cancer after treatment of non-pancreatic cancer will increase in parallel with improved patient outcomes after treatment of the non-pancreatic primary cancers. Our study participants had various non-pancreatic cancers (namely gastric cancer, colorectal cancer, malignant melanoma, breast cancer and hepatocellular carcinoma) and one of them had asynchronous triple cancers (colorectal, ureteral, and uterine cancers). It may be important to pay particular attention to the possibility of pancreatic adenocarcinoma during surveillance after treatment of non-pancreatic primary cancers.

Ahn et al compared the CT findings in patients with delayed diagnosis of pancreatic cancers with those of a control group with non-pancreatic disease and chronic pancreatitis and found that focal low attenuated areas are commonly present in patients with early pancreatic cancer (sensitivity 75%, specificity 84%). Vernuccio et al retrospectively investigated missed CT evidence of pancreatic cancer, and also reported that focal low attenuation was the most common findings suggestive of early pancreatic cancer in all three of their patients. These investigators assessed pancreatic phase contrast-enhanced CT images or multiphasic contrast-enhanced CT images.

Lu et al have reported the usefulness of pancreatic phase imaging for detecting pancreatic adenocarcinoma. They found...
significantly greater differences between attenuation of pancreatic adenocarcinoma and parenchyma during the pancreatic phase than the hepatic phase (67 HU ± 19 vs 39 HU ± 16). The pancreatic phase is now considered optimal for detecting pancreatic adenocarcinoma. Fletcher et al. have reported excellent sensitivities for tumor detection in the pancreatic phase (97%). Pancreatic adenocarcinomas can be clearly seen as hypoattenuated lesions compared with the normal parenchyma in the pancreatic phase, whereas identification of these tumors during the portal venous phase is not always easy. In our study participants, CT findings suggestive of early pancreatic adenocarcinoma were assessed only on venous phase contrast-enhanced CT images. Hence, focal hypoattenuated areas suggestive of pancreatic adenocarcinoma were identified on initial CT images in only three of the six patients (50%). It is possible that small areas of hypoattenuation would have been detected more frequently if pancreatic phase contrast-CT had been performed. Small pancreatic adenocarcinomas occasionally have enhanced rims in the portal venous to equilibrium phase. In our study, enhanced rims were identified in two of the three patients with hypoattenuated areas. Small focal areas of hypoattenuation with enhanced rims on venous phase contrast-enhanced CT images may be an important indicator of early pancreatic adenocarcinoma. Several investigators have reported that pancreatic duct dilatation is the earliest indicator of pancreatic cancer. Amin et al. also reported that pancreatic duct interruption and dilatation can indicate pancreatic cancer in patients without a mass; however, the sensitivity was relatively low (dilatation; 50%, interruption; 45%). Furthermore, Yamao et al. reported that the presence of partial pancreatic parenchymal atrophy and/or pancreatic duct abrupt stenosis on CT images is highly suggestive of small (≤10 mm) pancreatic adenocarcinomas. In our study, pancreatic duct dilatation without a pancreatic mass was identified on initial venous phase contrast-CT in two of the six patients (33%) and partial pancreatic parenchymal atrophy surrounding pancreatic duct interruption was identified in one patient (16%). Tada et al. reported that pancreatic cystic lesions such as intraductal papillary mucinous neoplasm and simple cysts are associated with a higher risk of pancreatic adenocarcinoma. One patient (17%) in our study developed a pancreatic adenocarcinoma after having a pathologically diagnosed retention cyst. Strong parenchymal enhancement surrounding the cyst was noted in this patient. This finding may have reflected blockage of pancreatic juice drainage caused by a small pancreatic adenocarcinoma. Pancreatic duct interruption and dilatation, partial pancreatic atrophy, and pancreatic cysts on venous phase contrast-enhanced CT images are considered to be suggestive of pancreatic adenocarcinoma.

It is difficult to assess the natural history of pancreatic adenocarcinoma. Ahn et al. evaluated 100 patients with pathologically proven pancreatic cancers who had undergone at least two CT examinations prior to the final diagnosis and reported that small pancreatic adenocarcinomas (≤20 mm) on initial CT grow slowly and take significantly longer to develop distant metastases than do larger pancreatic adenocarcinomas (20 mm<). According to these authors, the tumor volume doubling time for pancreatic adenocarcinoma ranged from 20.0 to 976.8 days (mean 132.2 days). In the present study, all three small hypoattenuated areas on initial CT that could have been suspected of being pancreatic adenocarcinomas were less than 20 mm in diameter (5–10 mm, median 8.5 mm). Tumor volume doubling time as calculated from CT data was shorter than previously reported (46–407 days, median 106 days). Tumor diameters measured on resected specimens were 12–55 mm (median 18 mm). Local tumor invasion was identified in all cases (100%). In Case 6, the patient with a cystic lesion, the tumor grew rapidly to 55 mm in diameter in the 6 months before pancreatic surgery. Hence, when findings suspicious of pancreatic adenocarcinoma (e.g. small areas of hypoattenuation, pancreatic duct dilatation and interruption, parenchymal atrophy, and pancreatic cysts) are detected during post-operative follow-up of patients with non-pancreatic primary cancers, prompt action regarding a possible pancreatic adenocarcinoma is imperative.

This study had several limitations. First, its retrospective nature may have been a source of selection bias. Second, it was a small study. Third, the reviewers were aware of the diagnosis. However, the purpose of our study was to investigate early evidence of pancreatic adenocarcinoma on venous phase contrast-enhanced CT images. Further larger prospective studies are required to confirm the indicators of pancreatic adenocarcinoma on venous contrast-enhanced CT.

In summary, small hypoattenuated areas with or without rim enhancement, pancreatic duct interruption and dilatation, focal parenchymal atrophy, and cystic lesions on venous phase contrast-enhanced CT images are suggestive of early pancreatic adenocarcinoma.

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CONTRIBUTORS

Guarantors of integrity of entire study, R.T., Y.A.; study concepts/study design or data acquisition or data analysis/interpretation, all authors; manuscript drafting or manuscript revision for important intellectual content, all authors; approval of final version of submitted manuscript, all authors; literature research, R.T., Y.Y., S.M.; clinical studies, R.T., R.S., T.D., Y.E., M.I.; and manuscript editing, R.T., Y.Y, Y.A.

COMPLIANCE WITH ETHICAL STANDARDS

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. IRB approval was obtained, and the need for informed consent was waived for this retrospective study.
REFERENCES

1. Kamisawa T, Wood LD, Itoi T, Takao K. Pancreatic cancer. Lancet 2016; 388: 73–85. doi: https://doi.org/10.1016/S0140-6736(16)00141-0

2. Xu Z, Pothula SP, Wilson JS, Apte MV. Pancreatic cancer and its stroma: a conspiracy theory. World J Gastroenterol 2014; 20: 11216–29. doi: https://doi.org/10.3748/wjg.v20.i32.11216

3. Siegel R, Ma J, Zou Z, Jemal A. Cancer statistics, 2014. CA Cancer J Clin 2014; 64: 9–29. doi: https://doi.org/10.3322/caac.21208

4. Gleason MX, Mdzinarishvili T, Are C, Sasson A, Sherram A, Shats O, et al. Prognostic estimator of survival for patients with localized and extended pancreatic ductal adenocarcinoma. Cancer Inform 2013; 12: CIDN.11496–14. doi: https://doi.org/10.4137/CIN.D11496

5. Sahani DV, Shah ZK, Catalano OA, Boland GW, Brugge WR. Radiology of pancreatic adenocarcinoma: current status of imaging. J Gastroenterol Hepatol 2007; 23: 23–33. doi: https://doi.org/10.1111/j.1440-1746.2007.05117.x

6. Lu DS, Vedantham S, Krasny RM, Kadell R, Berger WL, Reber HA. Two-Phase helical CT for pancreatic tumors: pancreatic versus hepatic phase enhancement of tumor, pancreas, and vascular structures. Radiology 1996; 199: 697–701. doi: https://doi.org/10.1148/radiology.199.3.8637990

7. McWilliams RR, Rabe KG, Olswold C. Risk of malignancy in first-degree relatives of patients with pancreatic carcinoma. Cancer 2005; 104: 388–94. doi: https://doi.org/10.1002/cncr.21166

8. McWilliams RR, Bamlet WR, Rabe KG, Olson JE, de Andrade M, Petersen GM. Association of family history of specific cancers with a younger age of onset of pancreatic adenocarcinoma. Clin Gastroenterol Hepatol 2006; 4: 1143–7. doi: https://doi.org/10.1016/j.cgh.2006.05.029

9. Brune KA, Lau B, Palmisano E, Canto M, Goggins MG, Hruban RH, et al. Importance of age of onset in pancreatic cancer kindreds. J Natl Cancer Inst 2010; 102: 119–26. doi: https://doi.org/10.1093/jnci/djp466

10. Wang L, Brune KA, Visvanathan K, Laheru D, Herman J, Wolfgang C, et al. Elevated cancer mortality in the relatives of patients with pancreatic cancer. Cancer Epidemiol Biomarkers Prev 2009; 18: 2829–34. doi: https://doi.org/10.1158/1055-9965.EPI-09-0557

11. Amin S, McBride RB, Kline JK, Mitchel EB, Lucas AL, Neugut AI, et al. Incidence of subsequent pancreatic adenocarcinoma in patients with a history of nonpancreatic primary cancers. Cancer 2012; 118: 1244–51. doi: https://doi.org/10.1002/cncr.26414

12. Eriuchi N, Aoyagi S, Hara M, Okuda K, Tamae T, Fukuda S, et al. Synchronous or metachronous double cancers of the pancreas and other organs: report on 12 cases. Surg Today 2000; 30: 718–21. doi: https://doi.org/10.1007/s005950070083

13. Ahn SS, Kim M-J, Choi J-Y, Hong H-S, Chung YE, Lim JS. Indicative findings of pancreatic cancer in prediagnostic CT. Eur Radiol 2009; 19: 2448–55. doi: https://doi.org/10.1007/s00330-009-1422-6

14. Yamao K, Takenaka M, Ishikawa R, Okamoto A, Yamazaki T, Nakai A, et al. Partial pancreatic parenchymal atrophy is a new specific finding to diagnose small pancreatic cancer (≤10 Mm) including carcinoma in situ: comparison with localized benign main pancreatic duct stenosis patients. Diagnostics 2020; 10: 445. doi: https://doi.org/10.3390/diagnostics10070445

15. Yoon SH, Lee JM, Cho JY, Lee KB, Kim JE, Moon SK, et al. Small (≤20 Mm) pancreatic adenocarcinomas: analysis of enhancement patterns and secondary signs with multiphasic multidetector CT. Radiology 2011; 259: 442–52. doi: https://doi.org/10.1148/radiol.11101183

16. Tada M, Kawabe T, Arizumi M, Togawa O, Miyazaki T, Aoyagi S, et al. Benign pancreatic cystic lesions: a prospective study in 197 patients. Jpn J Clin Oncol 2005; 35: 1203–8. doi: https://doi.org/10.1111/j.1399-0656.2005.00181.x

17. Vernuccio F, Midiri M, Lagalla R, Brancatelli G, Di Giovanni G, Brugge WR. Radiology of pancreatic adenocarcinoma. CIN.S11496–14. doi: https://doi.org/10.4137/CIN.S11496

18. Takaji R, Yamada Y, Matsumoto S, Kiyonaga M, Hongo N, Mori H, et al. Pancreatic adenocarcinoma versus chronic pancreatitis: differentiation with triple-phase helical CT. Abdom Imaging 2010; 35: 163–71. doi: https://doi.org/10.1007/s00261-009-9579-7

19. Yamada Y, Mori H, Matsumoto S, Kiyonoue H, Hori Y, Hongo N. Pancreatic adenocarcinoma and chronic pancreatitis: differentiation with triple-phase helical CT. Abdom Imaging 2010; 35: 163–71. doi: https://doi.org/10.1007/s00261-009-9579-7

20. Koeblinger C, Ba-Ssalamah A, Goetzinger P, Puchner S, Weber M, Sahora K, et al. Gadobenate dimeglumine-enhanced 3.0-T MR imaging versus multiphasic 64-detector row CT: prospective evaluation in patients suspected of having pancreatic cancer. Radiology 2011; 259: 757–66. doi: https://doi.org/10.1148/radiol.11101189

21. Flecher DG, Wiersema MJ, Farrell MA, Filder JL, Burgart LJ, Koyama T, et al. Pancreatic malignancy: value of arterial, pancreatic, and hepatic phase imaging with multi-detector row CT. Radiology 2003; 229: 81–90. doi: https://doi.org/10.1148/radiol.2291020582

22. Fletcher DG, Wiersema MJ, Farrell MA, Filder JL, Burgart LJ, Koyama T, et al. Pancreatic malignancy: value of arterial, pancreatic, and hepatic phase imaging with multi-detector row CT. Radiology 2003; 229: 81–90. doi: https://doi.org/10.1148/radiol.2291020582

23. Gangi S, Fletcher DG, Nathanson AM, Christensen JA, Harmsen WS, Crownhart BS, et al. Time interval between abnormalities seen on CT and the clinical diagnosis of pancreatic cancer: retrospective review of CT scans obtained before diagnosis. AJR Am J Roentgenol 2004; 182: 897–903. doi: https://doi.org/10.2214/ajr.182.4.1820897

24. Tanaka S, Nakaizumi A, Ioka T, Oshikawa O, Kiyozumi H, Hongo N, et al. Undetected pancreatic carcinoma: a prospective study of missed findings at computed tomography. World J Gastrointest Comput Tomogr 2016; 8: 200–8. doi: https://doi.org/10.4267/whg.2016.103.013

25. Akin M, Barisalvaci S, Hatanak SA, Goetzinger P, Puchner S, Weber M, Sahora K, et al. Gadobenate dimeglumine-enhanced 3.0-T MR imaging versus multiphasic 64-detector row CT: prospective evaluation in patients suspected of having pancreatic cancer. Radiology 2011; 259: 757–66. doi: https://doi.org/10.1148/radiol.11101189

26. Flecher DG, Wiersema MJ, Farrell MA, Filder JL, Burgart LJ, Koyama T, et al. Pancreatic malignancy: value of arterial, pancreatic, and hepatic phase imaging with multi-detector row CT. Radiology 2003; 229: 81–90. doi: https://doi.org/10.1148/radiol.2291020582

27. Gangi S, Fletcher DG, Nathanson AM, Christensen JA, Harmsen WS, Crownhart BS, et al. Time interval between abnormalities seen on CT and the clinical diagnosis of pancreatic cancer: retrospective review of CT scans obtained before diagnosis. AJR Am J Roentgenol 2004; 182: 897–903. doi: https://doi.org/10.2214/ajr.182.4.1820897

28. Han SK, Choi SJ, Kim HS. Time to progression of pancreatic cancer: evaluation with multi-detector computed tomography. J Gastrointest Cancer 2017; 48: 164–9. doi: https://doi.org/10.1007/s12029-016-9876-7