Pitfall in follow-up imaging of pancreatic neuroendocrine tumor by somatostatin receptor PET

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Abstract

56-year old woman was operated of a pancreatic NET in May 2011. Abdominal pain had led to imaging and consecutively the finding of cholecystolithiasis and the tumor. The gall bladder, left hemi-pancreas, regional lymph nodes and the (unintentional injured) spleen were resected. At routine control examination in October 2012 CT presented three contract enhancing intra-abdominal lesions with a diameter of 2–3.5 cm. Consecutively ^{68}Ga-DOTA-NOC PET-CT showed high tracer uptake (SUV 10-12) at these lesions. Therefore a relapse of the neuroendocrine tumor was suspected. After reoperation in December 2012 histology did not reveal any sign of neuroendocrine tumor but identified spleen tissue most probably caused by splenosis accidentally seeded at the first operation.
Physiologically the spleen is highly avid at ^{68}Ga-DOTATOC PET, but splenosis presents with less standard uptake value. In our case the described lesions presented with an SUV quite comparable to that of neuroendocrine tumor tissue.

CASE

A 56-year old woman was operated of a pancreatic NET GI T2 N0 (Ki67 2%) in May 2011. Abdominal pain had led to imaging and consecutively the finding of cholecystolithiasis and the tumor. The gall bladder, left hemi-pancreas, regional lymph nodes and the (unintentional injured) spleen were resected. At routine control examination in October 2012 CT presented three contract enhancing intra-abdominal lesions with a diameter of 2–3.5 cm. Consecutively ^{68}Ga-DOTA-NOC PET-CT showed high tracer uptake (SUV 10-12) at these lesions. Therefore a relapse of the neuroendocrine tumor was suspected. After reoperation in December 2012 histology did not reveal any sign of neuroendocrine tumor but identified spleen tissue most probably caused by splenosis accidentally seeded at the first operation.

DISCUSSION

Tumors originating from neuroendocrine cells are increasingly identified, especially among gastro-entero-pancreatic neoplasms (Giandomenico *et al.* 2013). According to the grading differentiated tumor cells conserve neuroendocrine characteristics as somatostatin receptor expression. Therefore functional nuclear medicine imaging has a role in staging and restaging of NET (Hicks 2010; Cwikla

CASE REPORT

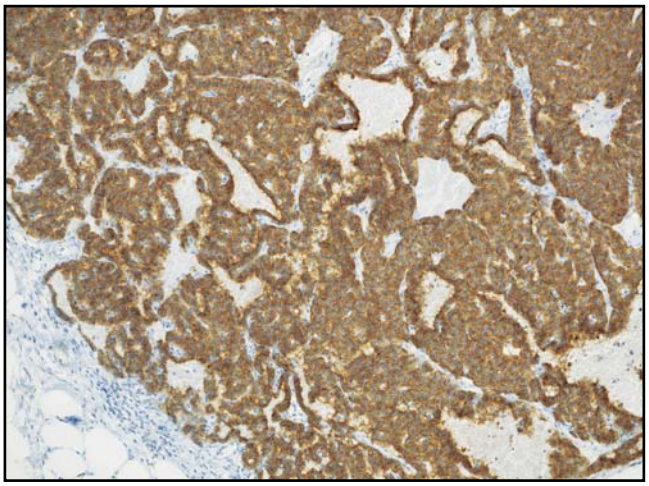

Fig. 1. In this specimen immunhistochemistry shows dense positive immunoreactivity for synapotphysin in the pancreatic NET.

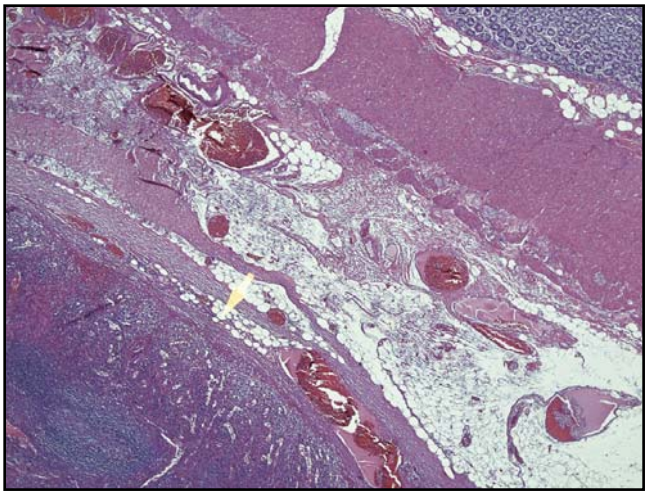

Fig. 2. Spenosis adjacent to the colon – corresponding to one of the suspicious lesions observed by ^{68}Ga somatostatin receptor PET/CT.

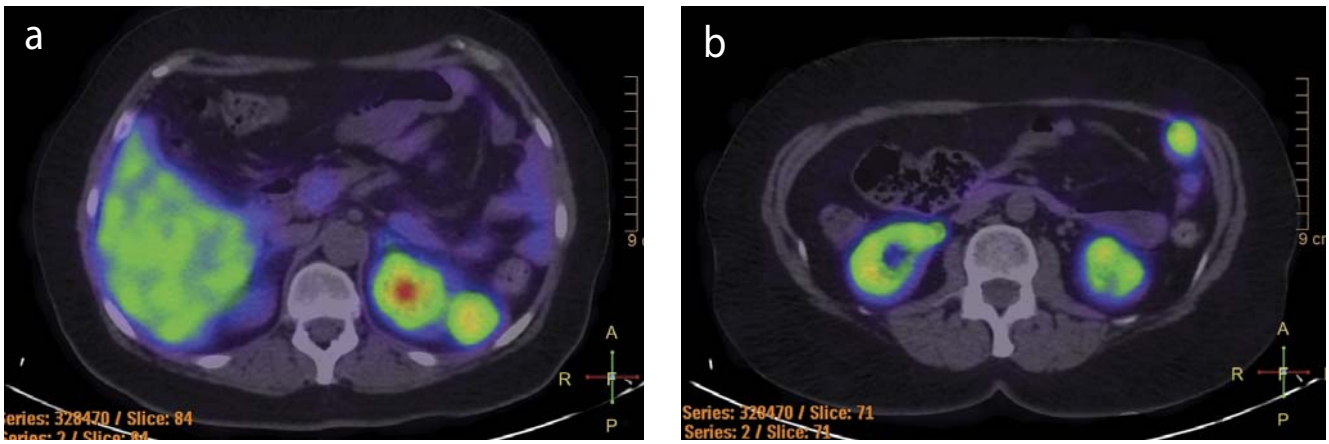

Fig. 3. ^{68}Ga-DOTA-NOC PET/CT presented three abdominal lesions (a, b, c) with high somatostatin-receptor density and therefore suspicious of NET metastases. As tissue of the spleen presents high somatostatin receptor density, this pitfall arose from splenosis which was unknown at this point of time.

et al. 2007). As inflammatory processes due to activated lymphocytes with positive somatostatin receptor staining reveal tracer accumulation in somatostatin receptor scintigraphy, pitfalls in cancer imaging by e.g. octreoscan are possible. Physiologically the spleen is highly avid at ^{68}Ga-DOTATOC PET, but splenosis presents with less standard uptake value (Kulkarni *et al.* 2013). Anyhow, in our case – after splenectomy without the possibility to compare – the described lesions presented with an SUV quite comparable to that of neuroendocrine tumor tissue.

REFERENCES

1 Giandomenico V, Modlin I, Pontén F, Nilsson M, Landegren U, Bergquist J et al (2013). Improving the Diagnosis and Management of Neuroendocrine Tumors: Utilizing New Advances in Biomarker and Molecular Imaging Science. Neuroendocrinology [Epub ahead of print].

2 Hicks RJ (2010). Use of molecular targeted agents for the diagnosis, staging and therapy of neuroendocrine malignancy. Cancer Imaging. **10**: S83–91.

3 Cwikla JB, Nasierowska-Guttmejer A, Jeziorski KG, Cichocki A, Zgliczynski W, Stepień K, et al (2007) Diagnostic imaging approach to gastro-entero-pancreatic carcinomas of neuroendocrine origin – single NET center experience in Poland. Neuro Endocrinol Lett. **28:** 789–800.

4 Kulkarni HR, Prasad V, Kaemmerer D, Hommann M, Baum RP (2013) High uptake of (68)Ga-DOTATOC in spleen as compared to splenosis: measurement by PET/CT. Recent Results Cancer Res. **194**: 373–378.