High \(^{18}\)F-FDG uptake in sporadic paraganglioma of the retroperitoneum may be related to intra-tumor haemorrhage and macrophages

Hayato Kaida\(^1\) MD, Seiji Kurata\(^1\) MD, Akihiko Kawahara\(^2\) PhD, Yuji Hiromatsu\(^1\) MD, Masayoshi Kage\(^2\) MD, Masatoshi Ishibashi MD

1. Division of Nuclear Medicine, PET Center, and Department of Radiology, Kurume University, School of Medicine, 67 Asahi-Machi, Kurume, Fukuoka, 830-0011, Japan. E-mail: hayato@med.kurume-u.ac.jp
2. Department of Diagnostic Pathology, Kurume University Hospital, 3. Division of Endocrinology and Department of Internal Medicine, Kurume University School of Medicine, Kurume City, Fukuoka, 830-0011, Japan.


A 70 years old asymptomatic patient underwent abdominal computed tomography (CT) to follow-up after an abdominal aortic aneurysm operation, and a smooth, round tumor was detected at the interaortocaval region of the retroperitoneum. Fluorine-18-FDG-PET/CT showed a high accumulation of \(^{18}\)F-FDG in this tumor with SUVmax 30.5 in the early and 47.5 in the delayed phase. Iodine-123-MIBG scintigraphy was performed because of increased levels of serum 5 HIAA, VMA and HVA, and \(^{123}\)I-MIBG uptake was observed. The histopathology showed a paraganglioma (PGL). Immunohistochemical staining for glucose transporter-1 (GLUT-1), GLUT-3, vascular endothelial growth factor (VEGF) and hypoxia-inducible factor 1α (HIF-1α) was negative in the tumor cells. However, the red blood cells involved in the extensive intra-tumor hemorrhage showed a high expression of GLUT-1, and there were some macrophages with CD 68 expression in the tumor nest. To our knowledge, the pathological molecular mechanism of \(^{18}\)F-FDG uptake in PGL has not yet been clarified. In conclusion, high \(^{18}\)F-FDG uptake in sporadic PGL cells may be related to extensive intra-tumor hemorrhage and macrophages. Figures 1-6 refer to 4 modalities, pathology etc. as above.

Figure 1. CECT contrast shows the tumor.

Figure 2. MRI shows a low signal intensity tumor on T1WI with central high intensity.

Figure 3. High \(^{18}\)F-FDG uptake in the tumor.

Figure 4. \(^{123}\)I-MIBG uptake in the tumor.

Figure 5. Histology.

Figure 6. Representative GLUT-1 (A), GLUT-3 (B), VEGF, (C), HIF-1α (D) and CD68 (E) Immunostaining.