A 66-year-old man was admitted to the emergency clinic with abdominal pain, nausea, vomiting and constipation for four days. Physical examination revealed abdominal tenderness, distention. Abdominal CT showed thickening on the sigmoid colon wall suggesting a malignant tumor and a suspicious metastasis with 1 cm diameter on the segment 4 and 8 border in the liver. Sigmoid colon resection and additional nonanatomic liver resection of segment 4 were performed. Histologic examination of the colectomy specimen revealed adenocarcinoma infiltrating the serosa. The nodule contained necrotic, eosinophilic granular material with a fibrotic rim. A part of the nodule was composed of ghost hepatocyte plates with bile pigment and preserved normal reticular pattern consistent with coagulative necrosis of the liver parenchyma. An artery with narrowed lumen near the nodule was also observed. There was no thrombus within the artery.

Solitary necrotic nodule of the liver (SNNL) is a small subcapsular nodule characterized by a necrotic core surrounded by collagenized fibrous tissue. Most of the lesions are asymptomatic and they are detected by preoperative evaluation for another cause or incidentally during surgery [1]. Previous reports described this entity as a “burnt-out phase” of a variety of lesions and most of them lack specific etiology [1, 2]. The pathogenetic mechanism is still unclear. In their original study, Shepherd et al favored traumatic or infectious etiology [3]. Sunderesan et al showed the presence of the feeding vessels within the nodule suggesting a hemangiomatic origin. They described central reticulin fibers within the nodule suggesting sclerosing hemangioma [4]. There have been reports of thrombosis associated with SNNL and De Luca et al favored the ischemic hypothesis [5]. Parasitic infections including hydatid cyst are more likely to play an important role in certain cases. Another possible cause of SNNL is a metastatic tumor, as we have shown in a previous report [2].

All of the reported lesions shared nearly the same histologic features, characterized by amorphous necrotic material surrounded by fibrous capsule. The cellular origin of the necrotic material was usually not identified [2]. Necrotic hepatocytes with bile pigment and well-preserved reticulin framework were consistent with early hepatic parenchymal infarction. This histologic finding proved that some of these lesions are focal infarctions of the liver parenchyma. However, the underlying mechanism of this local infarct is not clear. Besides the metastatic disease, gastrointestinal malignant tumors seem able to affect hepatic microvasculature via the portal vein thus contributing to the development of SNNL.

References