



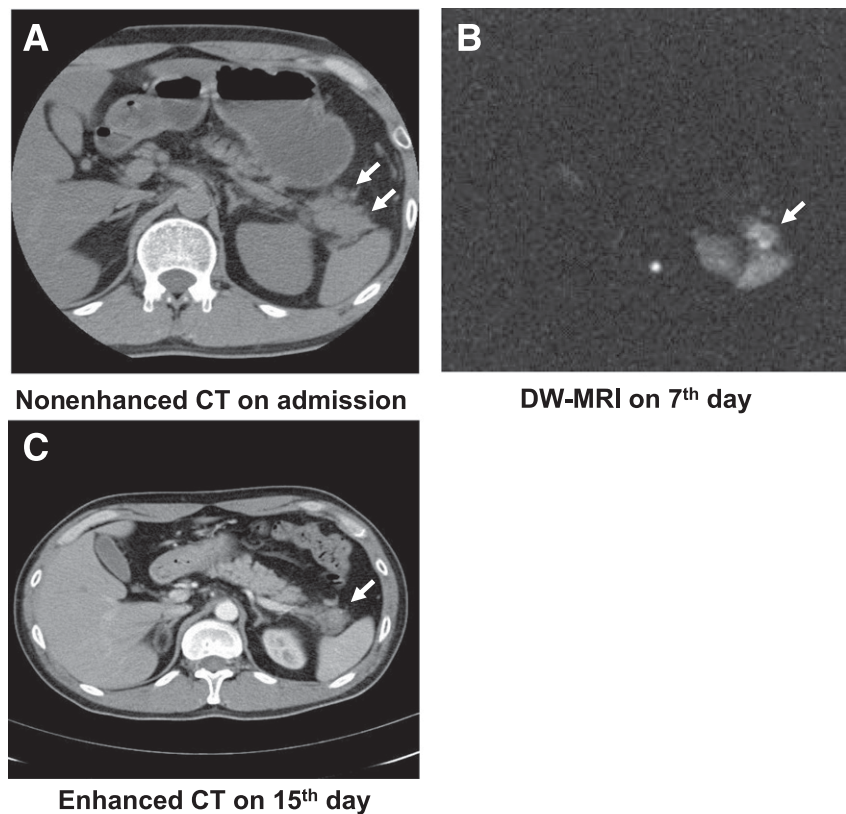
# Pancreatic Inflammation Captured by Imaging Technology at the Onset of Fulminant Type 1 Diabetes

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Type 1 diabetes mellitus is classified as autoimmune (type 1A) or idiopathic (type 1B) (1). Fulminant type 1 diabetes is classified as type 1B, characterized by acute onset of diabetic ketoacidosis soon after the development of typical diabetes symptoms, near-normal HbA<sub>1c</sub> level at onset, negative serum GAD antibody, and markedly acute progression of insulin deficiency (2). Infiltration of macrophage and T cells into the islets of Langerhans and complete destruction of pancreatic  $\beta$ -cells are characteristic. In this report, we detected pancreatic inflammation in a patient with fulminant type 1 diabetes by both dynamic computed tomography (CT) and MRI, which have been focused on as noninvasive imaging technologies for the pancreas (3).

A 36-year-old Japanese male was referred to our hospital because of diabetic ketoacidosis. He suffered from left abdominal pain, sore throat, and high fever (over 38°C) for 10 days. Polydipsia, polyuria, and nausea developed 2 days before admission. On admission, he was markedly dehydrated and hyperglycemic (49.7 mmol/L) with severe ketosis (urinary ketone 3+). Arterial pH was 7.127 and bicarbonate was 6.1 mmol/L, suggesting marked metabolic acidosis. He



**Figure 1**—A: Nonenhanced abdominal CT on admission. This image shows the swollen tail of the pancreas and fluid collection around it (white arrows). B: DW-MRI conducted on the 7th day after admission. The swelling of the pancreas remained and the extension of the inflammation to the surrounding fat tissue was observed and a high-intensity area was confirmed at the tail of the pancreas (white arrow). C: Follow-up dynamic CT conducted on the 15th day when the pancreatic inflammation seemed to be sustained. This image shows a low-density area in the tail of the pancreas (white arrow), which corresponds to the high-intensity area in DW-MRI.

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was diagnosed as having diabetic ketoacidosis and was treated by intravenous infusion of saline and insulin. On admission, HbA<sub>1c</sub> was 5.7%. Serum immunoreactive insulin (<0.5  $\mu$ U/mL) and serum (0.05 ng/mL) and urinary C-peptide response (<0.8  $\mu$ g/day) were markedly decreased, indicating severe impairment of insulin secretory capacity. GAD antibody was negative. Serum lipase, elastase 1, and amylase levels were increased: 177 IU/L (normal range 11–53 IU/L), 1,900 ng/dL (normal range 100–400 ng/dL), and 167 IU/L (normal range 33–120 IU/L), respectively. The patient's HLA genotype was DR4DQ4. We conducted abdominal CT (nonenhanced) on admission, which showed the swollen tail of the pancreas and fluid collection around it, indicating the inflammation of the pancreas (Fig. 1A). In the MRI conducted on the 7th day after admission, the swelling of the pancreas remained and a high-intensity area was confirmed at the tail of the pancreas in diffusion-weighted MRI (DW-MRI) (Fig. 1B). The follow-up dynamic CT conducted

on the 15th day, when serum amylase level remained high and pancreatic inflammation seemed to be sustained, showed a low-density area in the tail of the pancreas (Fig. 1C).

Fulminant type 1 diabetes is a disease characterized by acute onset of severe pancreatic inflammation. However, such inflammation was identified only by biopsy in only limited cases (2,4,5), and most of the reports on fulminant type 1 diabetes have shown the pancreatic inflammation only by enzymatic abnormality. In this case, we identified the pancreatic inflammation at disease onset in nonenhanced CT, and the existence of the inflammation was also proved by DW-MRI. Dynamic CT, which was conducted after the patient's condition became stable, still showed the inflammation of the tail of the pancreas. We believe that our case suggests the importance of examination of the pancreas by using a noninvasive technique such as DW-MRI and CT in cases suspected of fulminant type 1 diabetes at its onset. Further investigation will elucidate the necessity of

pancreatic imaging study for fulminant type 1 diabetes.

**Duality of Interest.** No potential conflicts of interest relevant to this article were reported.

**Author Contributions.** A.O. wrote the manuscript. H.K. and T.S. reviewed the manuscript. S.Ka., M.S., S.Ki., and A.I. contributed to the discussion. A.O. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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