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A case of fulminant type 1 diabetes mellitus in a patient with type 2 diabetes mellitus

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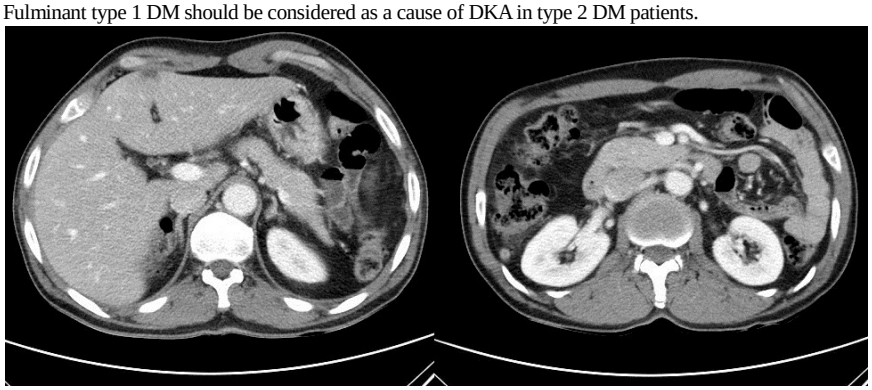
Fulminant type 1 diabetes mellitus (DM) is a rare subtype of type 1 DM characterized by sudden onset of hyperglycemia, ketoacidosis at diagnosis and decresed C-peptide secretion. After first reported by Imagawa et al. in 2000, several fulminant type 1 DM cases had been reported in South Korea. But, there were no reports of fulminant type 1 DM case in patients with type 2 DM previously well controlled with anti-hyperglycemic medications. Here, we report a rare case of fulminant type 1 DM in a patient with type 2 DM. A 70-year-old male visited emergency department of Hanil General Hospital in October 2017 and complained of abdominal discomfort, nausea, polydipsia and polyuria for 3 days. He had already been diagnosed with type 2 DM at local medical center 10 years ago. His plasma glucose level had been well controlled with sitagliptin 50mg/day and metformin 850mg/day. At emergency room, he was diagnosed with diabetic ketoacidosis (DKA) as a result of plasma glucose 404 mg/dL, arterial blood pH 7.17, anion gap 26 and blood ketone 2.2 mmol/L. There were some abnormal results of pancreatic enzymes but acute pancreatitis ruled out with no remarkable findings at contrast-enhanced abdomen computed tomography (CT) scan(figure1) and no abdominal pain or tenderness at physical examinations. Also, cancer were ruled out with abdomen CT scan. Additional laboratory tests revealed that his HbA1c level was 8.0%, fasting/postprandial C-peptide levels were very low (0.01/0.01 ng/mL) and GAD antibody was 1.39 U/ml. After recovered from DKA, he needed multiple dose insulin injection therapy (insulin glargine 12 unit, insulin glulisine 8-8-8 unit) to control his glucose level. Six months later, we observed that his C-peptide levels remained low at 0.01 ng/mL, suggesting complete destruction of pancreatic β-cells. In this case, although he had already diagnosed with type 2 DM, his laboratory test were fulfilled with the criteria of fulminant type 1 DM proposed by Imagawa et al. Fulminant type 1 DM should be considered as a cause of DKA in type 2 DM patients.



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Cilostazol induces regression of carotid plaque in patients with high cardiovascular risk

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Background/Aims: Cilostazol is reported to induce the regression of atherosclerotic changes. However, its effects on carotid plaque area are still not well-known. The study aim was to investigate effects of cilostazol versus aspirin on carotid plaque area and carotid artery intima thickness (IMT) in patients with high cardiovascular risk factors. **Methods:** To compare prevention by cilostazol and aspirin of progression of carotid atherosclerosis, we conducted a prospective, multicenter, randomized, open, blinded end point study in patients with high cardiovascular risk factors. A total of 250 patients were allocated to either an aspirin-treated (100 mg/d) group or cilostazol-treated (200 mg/d) group. The changes in total plaque area (TPA) and IMT of the carotid artery during a 1-year observation period were examined as the primary end point. **Results:** The regression in maximum right IMT, maximum left IMT, and mean common carotid artery IMT was significantly greater with cilostazol compared with aspirin(-0.07±0.18 versus 0.01±0.18 mm, P<0.001; -0.06±0.21 versus 0.03±0.19 mm, P<0.001; -0.07±0.17 versus 0.02 ± 0.15 mm, P<0.001). The regression in right TPA, left TPA, and TPA on common carotid artery was significantly greater with cilostazol compared with aspirin(-0.18±0.35 versus 0.02±0.43 mm2, P<0.001; -0.14±0.36 versus 0.04±0.31 mm2, P<0.001; -0.32±0.58 versus 0.03± 0.62 mm2, P<0.001). **Conclusions:** Cilostazol potently inhibited progression of carotid plaque area and IMT compared with aspirin in patients with high cardiovascular risk factors.

Table 1. Baseline characteristics

	Pletaal (n=128)	ASA (n=122)	P value
Age	64.8±10.1	62.7±10.5	0.119
Male	65(50.8%)	62 (50.8%)	
BMI	24.9±3.3	25.4±3.2	
Systolic BP, mmHg	130.8±18.6	131.8±16.0	0.715
Diastolic BP, mmHg	74.9±11.0	77.8±10.3	0.069
Diabetes mellitus	22 (17.2%)	29 (24%)	
Ischemic heart disesae	32 (25%)	46 (38%)	
Stroke	6 (5%)	1 (0.8%)	
Smoker	10 (8%)	8 (7%)	
RAS blocker	43 (34%)	62 (50.8%)	
CCB	64 (50%)	69 (56.6%)	
Beta blocker	59 (46%)	56 (45.9%)	
Statin	105 (82%)	101 (82.8%)	
T-chol	156.8±39.5	167.4±47.1	0.093
LDL	85.0±35.2	95.8±43.2	0.060
TG	121.0±78.4	132.3±62.5	0.270
HDL	51.1±14.3	50.5±14.2	0.597
FBS	116.8±43.0	118.3±44.1	0.751
HbA1c	6.40±1.17	6.60±1.41	0.330
hsCRP	0.22±0.40	0.12±0.17	0.795
Rt ICA IMT, mm	0.69±0.16	0.64±0.12	0.006
Rt CCA IMT, mm	0.72±0.18	0.73±0.21	0.918
Lt CCA IMT, mm	0.74±0.20	0.75±0.21	0.582
Mean IMT, mm	0.73±0.16	0.74±0.18	0.934
Rt TPA	0.29±0.41	0.29±0.40	0.448
Lt TPA	0.24±0.35	0.21±0.29	0.666
TPA	0.53±0.66	0.50±0.62	0.696

Table 2. Follow-up parameters after 1-year-treatment

	Pletaal (n=128)	ASA (n=122)	P value
T-chol	163.6±40.3	168.1±37.8	0.322
LDL	91.2±38.0	91.2±37.4	0.958
TG	121.8±80.0	165.0±95.7	0.019
HDL	53.3±15.9	48.4±10.7	0.003
FBS	120.0±45.0	117.0±47.5	0.751
HbA1c	6.24±1.0	6.86±1.5	0.012
hsCRP	0.22±0.40	0.12±0.17	0.099
Rt ICA IMT, mm	0.64±0.13	0.69±0.15	<0.001
Rt CCA IMT, mm	0.65±0.14	0.74±0.18	<0.001
Lt CCA IMT, mm	0.67±0.14	0.78±0.19	<0.001
Mean IMT, mm	0.66±0.12	0.76±0.16	<0.001
Rt TPA	0.11±0.18	0.31±0.40	<0.001
Lt TPA	0.11±0.18	0.24±0.32	<0.001
TPA	0.22±0.33	0.54±0.63	<0.001
ΔRt ICA IMT, mm	-0.05±0.16	0.05±0.15	<0.001
ΔRt CCA IMT, mm	-0.07±0.18	0.01±0.18	<0.001
ΔLt CCA IMT, mm	-0.06±0.21	0.03±0.19	<0.001
ΔMean IMT, mm	-0.07±0.17	0.02±0.15	<0.001
ΔRt TPA	-0.18±0.35	0.02±0.43	<0.001
ΔLt TPA	-0.14±0.36	0.04±0.31	<0.001
ΔTPA	-0.32±0.58	0.03±0.62	<0.001