




Pyogenic psoas abscess on the dorsal side, and bacterial meningitis and spinal epidural abscess on the ventral side, both of which were induced by spontaneous discitis in a patient with diabetes mellitus: A case report

Megumi Horiya¹, Takatoshi Anno^{1*} , Mayuko Kawada¹, Haruki Yamada¹, Kaiou Takahashi¹, Haruka Takenouchi¹, Hideyuki Iwamoto¹, Fumiko Kawasaki¹, Katsumi Kurokawa², Hideaki Kaneto³ , Kohei Kaku¹ , Koichi Tomoda¹

¹Department of General Internal Medicine 1, Kawasaki Medical School, Okayama, Japan, ²Department of Neurology, Kawasaki Medical School, Okayama, Japan, and ³Department of Diabetes, Endocrinology and Metabolism, Kawasaki Medical School, Kurashiki, Japan

Keywords

Bacterial meningitis, Pyogenic psoas abscess, Spontaneous discitis

*Correspondence

Takatoshi Anno
Tel.: +81-86-225-2111
Fax: +81-86-232-8343
E-mail address:
anno-t@umin.ac.jp

J Diabetes Investig 2021; 12: 1301–1305

doi: 10.1111/jdi.13461

ABSTRACT

Type 2 diabetes mellitus patients are immunocompromised, particularly under poorly controlled conditions, and thereby they could develop rare inflammatory diseases, such as spontaneous discitis, pyogenic psoas abscess, spinal epidural abscess and bacterial meningitis. Herein we report a pyogenic psoas abscess on the dorsal side, and bacterial meningitis and spinal epidural abscess on the ventral side, both of which were induced by spontaneous discitis in a patient with poorly controlled type 2 diabetes mellitus. This case was very rare and interesting, because we successfully treated various infections with antibiotics over a long period of time, complicated by hyperglycemic crises, although the patient suffered severe bone destruction and required rehabilitation for a long time.

INTRODUCTION

Type 2 diabetes mellitus is a disease many complications induced by hyperglycemia, inflammation and immune disorder. Therefore, patients with type 2 diabetes mellitus have an increased susceptibility to infection, especially when glycemic control is poor^{1,2}. Symptoms of spontaneous discitis are mainly fever and back pain³, and spontaneous discitis is often complicated by diabetes. Furthermore, pyogenic psoas abscess, which is caused when spontaneous discitis is spread on the dorsal side, is also often complicated by diabetes⁴. In contrast, spinal epidural abscess and bacterial meningitis are also caused when spontaneous discitis is spread on the ventral side, and reportedly it is complicated by diabetes^{5,6}.

Herein we report a complication of spontaneous discitis, pyogenic psoas abscess, spinal epidural abscess, bacterial meningitis

and sepsis, all of which were associated with poorly controlled type 2 diabetes mellitus. It is very rare to experience such a widely spreading spontaneous discitis, as observed in the present case. In addition, we successfully treated various infections with antibiotics over a long period of time and hyperglycemic crises, although the patient suffered severe bone destruction and required rehabilitation.

CASE REPORT

A 62-year-old Japanese woman was brought to the emergency room in a coma. She was diagnosed as type 2 diabetes mellitus at the age of 57 years. Her vital signs were as follows: temperature 37.1°C, blood pressure 126/94 mmHg, heart rate 117 b.p.m. and oxygen saturation 94% (room air). Table 1 shows her laboratory data in the emergency room. She suffered from hyperglycemic crises. Diabetes-associated data were as follows: plasma glucose 688 mg/dL, hemoglobin A1c 10.3%, glycoalbumin 36.1%, total ketone body 7617.4 μmol/L,

Received 29 September 2020; revised 5 November 2020; accepted 9 November 2020

Table 1 | Patient's laboratory data in the emergency room

Variable	Result	Reference range	Variable	Result	Reference range
Peripheral blood			Diabetes marker		
White blood cells (μL)	9,600	3,300–8,600	Plasma glucose (mg/dL)	688	
Neutrophil (%)	94.0	28.0–78.0	Hemoglobin A1c (%)	10.3	4.9–6.0
Red blood cells ($\times 10^4/\mu\text{L}$)	339	435–555	Glycoalbumin (%)	36.1	12.4–16.3
Hemoglobin (g/dL)	9.8	13.7–16.8	Total ketone body ($\mu\text{mol/L}$)	7,617.4	0.0–130.0
Hematocrit (%)	29.6	35.1–44.4	Acetoacetate ($\mu\text{mol/L}$)	2,236.1	0.0–55.0
Platelets ($\times 10^4/\mu\text{L}$)	17.9	15.8–34.8	β -Hydroxybuterate ($\mu\text{mol/L}$)	5381.3	0.0–85.0
Blood biochemistry			Infectious marker		
Total protein (g/dL)	6.6	6.6–8.1	CRP (mg/dL)	29.76	<0.14
Albumin (g/dL)	2.6	4.1–5.1	Procalcitonin (ng/mL)	19.48	0.00–0.05
Globulin (g/dL)	4.0	2.2–3.4	Blood gas analysis		
Total bilirubin (mg/dL)	0.6	0.4–1.5	pH	7.365	7.360–7.460
AST (U/L)	22	13–30	PCO ₂ (mmHg)	23.9	34.0–46.0
ALT (U/L)	37	10–42	PO ₂ (mmHg)	93.3	80.0–90.0
LDH (U/L)	224	124–222	HCO ₃ ⁻ (mEq/L)	13.3	24.0–32.0
ALP (U/L)	368	106–322	BE (mEq/L)	-10.4	-2.5 to 2.5
γ -GTP (U/L)	24	13–64	SO ₂ (%)	96.0	95.0–98.0
BUN (mg/dL)	70	8–20	Lactate (mEq/L)	3.40	0.63–2.44
Creatinine (mg/dL)	2.16	0.65–1.07	Urinary test		
Cholinesterase (U/L)	183	240–486	Urinary pH	5.0	5.0–7.5
Uric acid (mg/dL)	10.8	2.6–5.5	Urinary protein	1+	–
Creatine Kinase (U/L)	20	41–153	Urinary sugar	3+	–
Amylase ($\mu\text{g/dL}$)	10	42–118	Urinary ketone body	2+	–
Ammonia ($\mu\text{g/dL}$)	11	12–66	Urinary bilirubin	–	–
Total cholesterol (mg/dL)	186	142–248	Urinary blood	–	–
Sodium (mmol/L)	140	138–145	Urinary bacteria	2+	–
Potassium (mmol/L)	5.1	3.6–4.8	Examination of cerebrospinal fluid		
Chloride (mmol/L)	100	101–108	Cell counts (μL)	2,101.3	0.0–3.3
IP (mg/dL)	3.7	2.7–4.6	ML (%)	7.0	
Calcium (mg/dL)	9.6	8.8–10.1	PML (%)	93.0	
Magnesium (mg/dL)	2.0	1.9–2.6	Protein (mg/dL)	291	10–40
			Glucose (mg/dL)	276	50–75

γ -GTP, γ -glutamyltranspeptidase; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BE, base excess; BUN, blood urea nitrogen; CRP, C-reactive protein; IP, inorganic phosphorus; LDH, lactate dehydrogenase; ML, monomorphonuclear leukocytes; PML, polymorphonuclear leukocyte.

acetoacetate 2236.1 $\mu\text{mol/L}$ and β -hydroxybuterate 5281.3 $\mu\text{mol/L}$. Blood gas analysis did not show acidosis: pH 7.365, base excess -10.4 mEq/L and lactate 3.40 mEq/L. The patient's blood osmotic pressure was remarkably elevated to 347 mOsm/L. Liver function was almost within the normal range, but the markers of renal function were elevated (creatinine 2.16 mg/dL, blood urea nitrogen 70 mg/dL). In addition, inflammation markers were markedly elevated: white blood cell 9,600/ μL (neutrophil 94.0%), C-reactive protein 29.76 mg/dL and procalcitonin 19.48 ng/mL. Her cerebrospinal fluid showed the findings of meningitis: cell counts 2,101.3/ μL , monomorphonuclear leukocytes 7.0%, polymorphonuclear leukocyte 93.0%, protein 291 mg/dL and glucose 276 mg/dL. As shown in Figure 1, the patient's abdominal computed tomography (CT; Figure 1a) and fat-saturated T2-weighted magnetic resonance imaging (Figure 1b) on admission showed a left pyogenic psoas abscess and spontaneous discitis. Based on

such findings, we thought that she probably suffered from a pyogenic psoas abscess on the dorsal side and bacterial meningitis complicated by a spinal epidural abscess on the ventral side, both of which were induced by spontaneous discitis. In addition, we also diagnosed her as hyperglycemic hyperosmolar syndrome and diabetic ketosis. Thus, we treated her with whole-body management, including infection and hyperglycemia.

On admission to the intensive care unit, we treated the patient's hyperglycemic crises and various infection first. We started administering 0.9% NaCl and continuous insulin infusion. Furthermore, we carried out CT image-guided percutaneous drainage and started antibiotics therapy for various infections (3 g/day of meropenem and 350 mg/day of daptomycin). Three days after admission, the same pathogenic bacteria (methicillin-susceptible *Staphylococcus aureus*) was detected in the patient's blood, cerebrospinal fluid and puncture culture

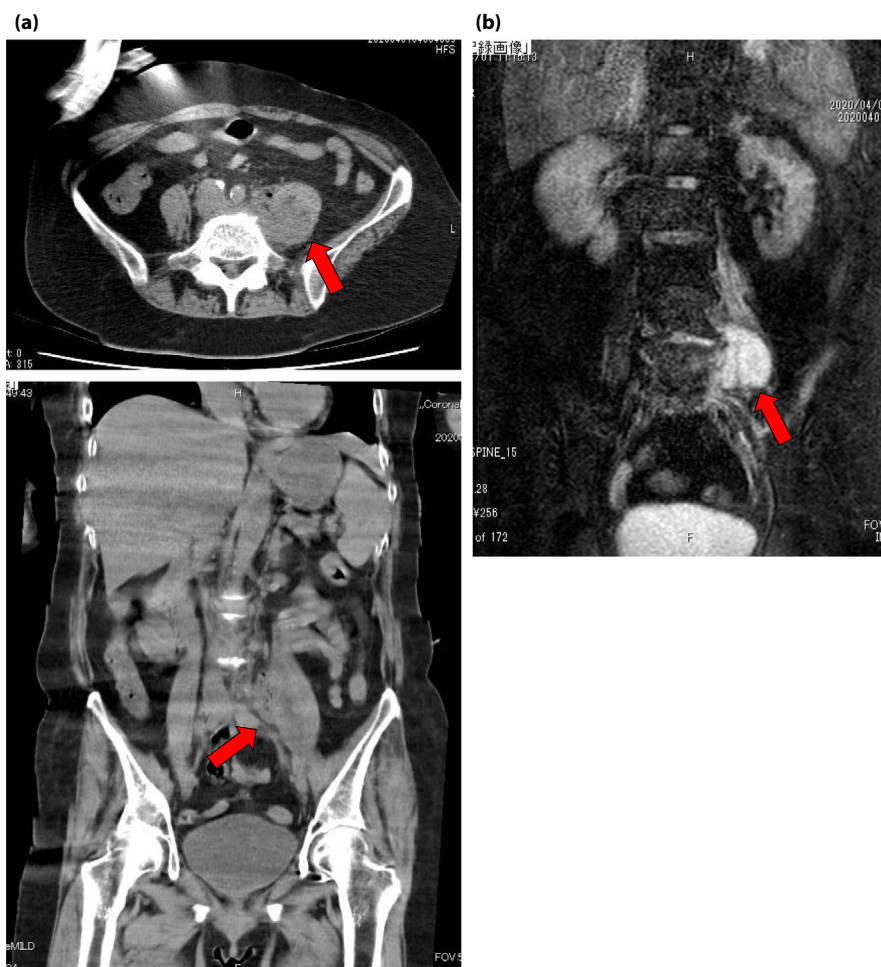


Figure 1 | (a) Abdominal computed tomography (CT) and (b) fat-saturated T2-weighted imaging of abdominal magnetic resonance imaging on admission. Abnormal computed tomography and magnetic resonance imaging showed pyogenic psoas abscess (red arrow). In addition, in the abnormal magnetic resonance imaging, the patient's intervertebral bodies at L4/L5 levels showed a high signal, which means her vertebrae at L4/L5 developed osteomyelitis and some edema.

from the pyogenic psoas abscess. We changed various antibiotics according to the sensitivity of bacteria as follows: we added 6 g/day of cefazolin on day 3, changed the antibiotics to 500 mg/day of levofloxacin on day 7, and changed to 1,200 mg/day of linezolid and 6.75 g/day of tazobactam/piperacillin on day 9. The patient's hyperglycemic crises were gradually improved. However, her infection markers still showed high levels (lowest C-reactive protein 5.46 mg/dL).

A total of 13 days after admission, she had fever and her infectious marker was elevated again (C-reactive protein 16.36 mg/dL). We carried out CT image-guided percutaneous drainage again. Then, her physical conditions became stable and infectious markers were improved, and after the second CT image-guided percutaneous drainage, we changed the antibiotics to 4 g/day of cefazolin on day 14. She was moved to the general ward from the intensive care unit on day 21. Then, we changed continuous insulin infusion to 6 units/day of insulin degludec injection, and added a total of 12 units/day of insulin aspart.

Also, we changed the antibiotics to 3 g/day of sulbactam/ampicillin on day 33, changed to 750 mg/day of amoxicillin/clavulanate on day 58, and changed to 450 mg/day of rifampicin and 800 mg/day of sulfamethoxazole and trimethoprim on day 91. Then, we changed the insulin therapy to oral diabetes drugs (30 mg/day of mitiglinide and 0.75 mg/week of dulaglutide), and the patient's hemoglobin A1c was as good as 6.4%. Finally, we successfully treated various infections and hyperglycemic crises, although the patient suffered severe bone destruction (Figure 2). She was discharged 125 days after admission.

As this is a case report, but not a clinical study, ethics approval was unnecessary. Written informed consent was obtained from the patient.

DISCUSSION

Herein, we reported a widely spreading infection from the focus of spontaneous discitis, which was spreading on the ventral side as a pyogenic psoas abscess, and was spreading on the dorsal



Figure 2 | Lumbar spine computed tomography taken 4 months after admission. There was severe bone destruction in the vertebrae at L4/L5 levels.

side as a spinal epidural abscess and bacterial meningitis in a patient with poorly controlled diabetes complicated by hyperglycemic crises. As her glycemic control was poor for a long time, we believe her spontaneous discitis and its spreading to a rare infection, at least in part, was associated with hyperglycemic conditions.

There are some reports showing that pyogenic psoas abscess is caused by immunocompromised conditions, such as diabetes, and most causative bacteria were *S. aureus*, as reported by Robert *et al.*⁷ In contrast, there are reports showing that spinal epidural abscess is often complicated by diabetes⁵ and meningitis⁶. Bacterial meningitis in adults is mainly caused by *Streptococcus pneumoniae*, meningococcus, Gram-negative bacilli and so on.

In the present patient, methicillin-susceptible *S. aureus* was detected in her blood, cerebrospinal fluid and puncture culture from a pyogenic psoas abscess. In addition, as shown on magnetic resonance imaging, her intervertebral bodies at L4/L5 levels were high signal, which means her disc at L4/L5 developed osteomyelitis and some edema. Therefore, in the present case, we believe the same methicillin-susceptible *S. aureus* was spreading from spontaneous discitis under poorly controlled diabetes. Such exacerbation of spontaneous discitis caused a pyogenic psoas abscess and spinal epidural abscess complicated by bacterial meningitis. In fact, spontaneous discitis itself is often complicated by diabetes⁸. To the best of our knowledge, it is very rare to experience such a widely spreading spontaneous discitis, as observed in this case, which was a spreading pyogenic psoas abscess on the dorsal side, and bacterial meningitis and a spinal epidural abscess on the ventral side. In addition, when a pyogenic psoas abscess is induced in a patient with poorly controlled diabetes, its prognosis is usually very poor. In the present case, however, we successfully treated

various infections with antibiotics, although it actually took a great deal of time.

Taken together, we should bear in mind that type 2 diabetes mellitus patients are immunocompromised, particularly under poorly controlled conditions, and thereby they could have rare inflammatory disease, such as spontaneous discitis, pyogenic psoas abscess, spinal epidural abscess and bacterial meningitis. In addition, the present case was very rare and interesting, because we successfully treated various infections complicated by hyperglycemic crises with antibiotics over a long period of time, although the patient suffered severe bone destruction and required rehabilitation for a long time.

ACKNOWLEDGMENTS

There was no funding for this work.

DISCLOSURE

The authors declare no conflict of interest.

REFERENCES

1. Shah BR, Hux JE. Quantifying the risk of infectious diseases for people with diabetes. *Diabetes Care* 2003; 26: 510–513.
2. Benfield T, Jensen JS, Nordestgaard BG. Influence of diabetes and hyperglycaemia on infectious disease hospitalisation and outcome. *Diabetologia* 2007; 50: 549–554.
3. Hopkinson N, Stevenson J, Benjamin S. A case ascertainment study of septic discitis: clinical, microbiological and radiological features. *QJM* 2001; 94: 465–470.
4. Kao PF, Tsui KH, Leu HS, *et al.* Diagnosis and treatment of pyogenic psoas abscess in diabetic patients: usefulness of computed tomography and gallium-67 scanning. *Urology* 2001; 57: 246–251.

5. Rigamonti D, Liem L, Sampath P, *et al.* Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. *Surg Neurol* 1999; 52: 189–196.
6. Miyazaki M, Yoshiiwa T, Kodera R, *et al.* Clinical features of cervical pyogenic spondylitis and intraspinal abscess. *J Spinal Disord Tech* 2011; 24: E57–E61.
7. Santaella RO, Fishman EK, Lipsett PA. Primary vs secondary iliopsoas abscess. Presentation, microbiology, and treatment. *Arch Surg* 1995; 130: 1309–1313.
8. Friedman JA, Maher CO, Quast LM, *et al.* Spontaneous disc space infections in adults. *Surg Neurol* 2002; 57: 81–86.