Case Report

Midline fasciotomy for severe acute pancreatitis with abdominal compartment syndrome: Case report

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1. Introduction

The abdominal compartment syndrome (ACS) is defined as new-onset organ failure induced by sustained elevated intra-abdominal pressure (IAP). Surgical decompression to decrease IAP may be performed in addition to supportive therapy.

A 42-year-old woman with a history of type 2 diabetes, dyslipidemia, alcohol disorder (130 g of daily alcohol intake), and schizophrenia presented to the emergency department with worsening abdominal pain and anorexia for 2 days. On arrival, her Glasgow Coma Scale score was 14 (E3V5M6). Physical examination revealed tachypnea with a respiratory rate of 26 breaths/min; other vital signs were stable. She was diagnosed with severe acute pancreatitis and required massive transfusions to stabilize her hemodynamic status from the time of admission to the intensive care unit (ICU). Acute blood purification was initiated. Bilateral pleural effusions increased from the second day, and despite the evacuation of the intraluminal contents, muscle relaxation was initiated because her IAP had increased to 52 mmHg and remained the same. Therefore, midline fasciotomy was performed instead of a midline incision through the linea alba on day 4, and the patient was managed with negative pressure wound therapy thereafter. Blood purification was completed on day 15, extubation was performed on day 17, and the patient was discharged from the ICU on day 29.

Clinical discussion and conclusion: Midline fasciotomy can have a decompressive effect in patients with primary ACS. This technique may be an alternative to decompressive laparotomy because of its less invasive nature.

2. Presentation of case

A 42-year-old Asian woman, with a history of type 2 diabetes, dyslipidemia, alcohol disorder, and schizophrenia, presented to the emergency department with worsening abdominal pain and anorexia for 2 days. Her regular medications included alpha-glucosidase inhibitors, major tranquilizers, and anxiolytic agents. For the last 3 years, she had been unable to stop binge drinking and reported a daily alcohol intake of 130 g. On arrival, her Glasgow Coma Scale score was 14 (E3V5M6). Physical examination revealed tachypnea with a respiratory rate of 26 breaths/min, heart rate of 109 beats/min, and blood pressure of 155/90 mmHg, without the use of vasopressors or inotropes. She was afebrile with a body temperature of 37 °C. No crackles were heard during chest auscultation. Her abdomen was distended, but there was no tenderness. Laboratory tests revealed a white blood cell count of 14,890/µL, C-reactive protein level of 61.8 mg/dL, triglyceride level of 1019 mg/dL.
and elevated liver enzymes (alanine aminotransferase: 39 IU/L, aspartate aminotransferase: 84 IU/L). Kidney function test revealed an excessively elevated serum creatinine level of 7.69 mg/dL and a BUN level of 58.1 mg/dL. Her serum amylase, p-amylase, and lipase levels remained within their reference ranges at 124 IU/L, 22 IU/L, and 65 IU/L, respectively (Table 1).

### Table 1: Laboratory findings at the time of admission.

| Biochemistry | Complete Blood Count |
|--------------|----------------------|
| **< Total Protein** | 6.8 g/dL |
| **Albumin** | 2.8 g/dL |
| **Creatinine Kinase** | 4660 IU/L |
| **AST** | 84 IU/L |
| **ALT** | 39 IU/L |
| **LDH** | 2275 IU/L |
| **ALP** | 177 IU/L |
| **γ-GTP** | 211 IU/L |
| **Amylase** | 124 IU/L |
| **Pancreatic amylase** | 22 IU/L |
| **Lipase** | 65 IU/L |
| **Triglyceride** | 1019 mg/dL |
| **Total cholesterol** | 306 mg/dL |
| **Total bilirubin** | 1.3 mg/dL |
| **Direct bilirubin** | 0.9 mg/dL |
| **Creatinine** | 7.61 mg/dL |
| **BUN** | 58.1 mg/dL |
| **Sodium** | 118 mEq/L |
| **Potassium** | 3.1 mEq/L |
| **Chloride** | 76 mEq/L |
| **Magnesium** | 2.2 mg/dL |
| **Calcium** | 2.6 mg/dL |
| **Glucose** | 237 mg/dL |
| **HbA1c** | 5.9 % |
| **CRP** | 61.8 mg/dL |

### Abbreviations
- ACS: abdominal compartment syndrome
- CT: computed tomography
- IAH: intra-abdominal hypertension
- IAP: intra-abdominal pressure
- ICU: intensive care unit
- AST: aspartate aminotransferase
- ALT: alanine aminotransferase
- LDH: lactic acid dehydrogenase
- ALP: alkaline phosphatase
- γ-GTP: γ-glutamyl transpeptidase
- BUN: blood urea nitrogen
- HbA1c: hemoglobin A1c
- CRP: C-reactive protein
- FiO2: fraction of inspiratory oxygen
- Fibrinogen: 575 mg/dL
- PT-INR: prothrombin time-international normalized ratio
- FDP: fibrin degradation product
- F1O2: fraction of inspiratory oxygen

Contrast-enhanced computed tomography (CT) revealed enlargement of the pancreas without pancreatic necrosis and several poorly defined peripancreatic fluid collections extending inferiorly to the kidney, consistent with interstitial edematous acute pancreatitis (Fig. 1A).

She was diagnosed with severe acute pancreatitis and admitted to the intensive care unit (ICU). After ICU admission, the patient was intubated, and mechanical ventilation was started. She also needed an aggressive intravenous infusion of approximately 10 L for the first 24 h to maintain the intravascular volume and achieve a mean arterial pressure of 65 mmHg and a urine output of 0.5 mL/kg/h.

She became anuric despite the administration of a large amount of ringer bicarbonate, which suggested the development of acute kidney injury (KDIGO grade 3; *Kidney International Supplements, 2012*). She was started on renal replacement therapy, including hemodiafiltration and extracorporeal ultrafiltration (Fig. 2).

On day 2, the bilateral pleural effusion increased in size and the intra-abdominal pressure (IAP) worsened. Despite reducing the rate of intravenous fluid administration and evacuating the intraluminal contents, her IAP increased to 52 mmHg. We initiated neuromuscular blockade using rocuronium, resulting in transient improvement with relapse of IAH. On day 4, as the IAP remained high (47 mmHg), the patient became hemodynamically unstable and required vasopressors to maintain the mean blood pressure. Nonsurgical management did not seem successful; therefore, surgical management was considered. To achieve surgical decompression, we performed a midline fasciotomy. The operation was performed by the attending physician who had received at least 5 years of surgical specialty training at our center. A skin incision was made inferiorly from the xiphoid process to the umbilicus, the peritoneum was preserved by cutting both the anterior and posterior rectus sheaths, and the wound was managed with negative pressure wound therapy (Fig. 1B–E).

After the operation, her IAP decreased to 30 mmHg, with subsequent improvement in her hemodynamic status. Her renal function improved, and the urine output was sufficient; therefore, daily hemodiafiltration was completed on day 15. Consistently, her organ function showed good recovery, with continuous enteral nutrition being well tolerated. Abdominal closure was performed on the 13th day; both the fascial layer and skin incisions were closed successfully. The patient was extubated on the 17th day.

Four weeks after ICU admission, follow-up CT revealed that the pancreatitis was no longer complicated by ACS. There were no complications, such as fascial layer separation, ventral herniation, or surgical wound infection. On the 29th day, adequate oral intake was established, so the patient was discharged from the ICU. On hospitalization day 53, the patient was transferred to a community hospital.

### 3. Discussion

We report the case of a patient with severe acute pancreatitis complicated by ACS that was successfully treated with midline fasciotomy. As a decompression technique, median fasciotomy seemed effective for the primary and secondary ACS, and disadvantages of laparotomy (such as infection, bleeding, and difficulty in fluid management) were circumvented.

IAH is defined as an IAP of ≥12 mmHg, which is associated with new-onset organ failure [1]. Approximately 40% of patients with severe acute pancreatitis develop ACS (although this number differs in many reports), and the mortality rates for acute pancreatitis are reported to be 49% with ACS and 11% without ACS [3]. The major contributors to an increased IAP include pancreatic and peripancreatic edema, which are exacerbated by the intravenous fluid administered for maintaining circulation. In the present case, the patient required an excessive amount of intravenous fluid to correct the kidney dysfunction and underlying intravascular hypovolemia.

As per the 2013 WSACS guidelines, a stepwise approach to reducing
the IAP is recommended [1]. This approach includes non-operative methods, such as nasogastric decompression, use of neuromuscular blockers, and fluid removal and hemofiltration. The methods for treating IAH should be chosen according to the cause of IAH and the degree of organ dysfunction [4]. Deteriorating patients with overt ACS treated with a non-operative strategy may require surgical decompression. The standard technique for surgical decompression is midline laparotomy, wherein a long midline incision is made through the linea alba to open the abdominal cavity; this is usually followed by an open abdomen [5]. Though effective and rapid, this approach is associated with a risk of infection, bleeding, intestinal fistula, and failed fascia closure, which may in turn require complex reconstructive surgery. Furthermore, patients with an open abdomen often need careful maintenance of fluid balance and nutritional support, because they have increased levels of

![Fig. 1. Abdominal contrast-enhanced CT on admission, and schema of the technique and negative pressure wound therapy (A) Contrast-enhanced CT revealed enlargement of the pancreas without pancreatic necrosis and several poorly defined peripancreatic fluid collections. Skin findings before (B) and after (C) midline fasciotomy. (D) The schema of midline fasciotomy on abdominal wall. (E) ABTHERA ADVANCE™ Open Abdomen Dressing was used for temporary abdominal closure.](image1)

![Fig. 2. Clinical Course](image2)

IAP, intraabdominal pressure; CDP-choline, cytidine diphosphate choline; HDF, hemodiafiltration; SLEDD, sustained low-efficiency daily dialfiltration; CHDF, continuous hemodiafiltration.
insensible fluid loss and are in a hypercatabolic state [6,7].

We chose midline fasciotomy as an alternative to decompressive laparotomy. Midline fasciotomy reduces resistance to abdominal wall compliance. In this scenario, the peritoneum is dilated, and the IAP is reduced compared to when the abdominal muscles are intact. However, the decompression effect of midline fasciotomy relative to that of decompressive laparotomy is unclear and needs to be validated further. Duchesne et al. indicated that midline fasciotomy improves the IAP in patients with blunt polytrauma and secondary ACS. To our knowledge, this is the first case report on the use of midline fasciotomy as a surgical decompression strategy for primary ACS [8]. In our patient’s case, midline fasciotomy decreased the IAP and improved organ function. Volume management was not complicated, and the wound was closed in a short time without intra-abdominal infection.

4. Conclusion

Midline fasciotomy had a decompressive effect in our patient with severe acute pancreatitis complicated by ACS. This technique may be an alternative to decompressive laparotomy because of its less invasive nature.

Ethical approval

In Japan, approval from an ethics committee is not required to report these cases. This case was reported in accordance with ethical guidelines for medical and health research involving human subjects established by the Japanese government.

Source of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

Study concept and design (YK, RK, HO, and YF), Data acquisition (YK, RK, HO, YN, YF, TF, TY, SY, and SO). Data analysis (YK, RK, HO, YN, YF, TF, TY, SY, and SO). Drafting and critical revision of the manuscript (YK, FY, HO, and RK). Approval of the final manuscript (YK, RK, HO, YN, YF, TF, TY, SY, and SO).

Trial registry number

1. Name of the registry:
2. Unique Identifying number or registration ID:
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

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Consent

Written informed consent was obtained from the patient’s legal guardians for the publication of this case report and accompanying images. A copy of the consent form is available for review from the Editor-in-Chief.

Research registration

None.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author/guarantor upon reasonable request.

Provenance and peer review

Not commissioned, externally peer-reviewed.

This case was presented at a regional conference (November 21–23, 2021, Tokyo, Japan).

Declaration of competing interest

I certify that all authors have no conflicts of interest that could affect this case report.

Acknowledgements

The authors would like to thank the paramedical crew who shared the data with us and allowed us to use the data in this report. We would like to thank Editage (www.editage.com) for English language editing.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jamsu.2022.104081.

References

[1] A.W. Kirkpatrick, D.J. Roberts, J. De Waele, R. Jaeschke, M.L. Malbrain, B. De Keulenaer, M. Bjercke, A. Leppanniemi, J.C. Eijke, M. Sugrue, M. Cheatham, R. Ivatury, C.G. Ball, A. Reintam Blaser, A. Regli, Z.J. Balogh, S. D’Amours, D. Debergh, M. Kaplan, E. Kimball, C. Olivera, Pediatric guidelines sub-committee for the world society of the abdominal compartment syndrome. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the world society of the abdominal compartment syndrome, Intensive Care Med. 39 (2013) 1190–1206, https://doi.org/10.1007/s00134-013-2906-z.

[2] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, The SCARE 2020 guideline: updating consensus surgical case report (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230, https://doi.org/10.1016/j.ijsu.2020.10.034.

[3] S. van Brunschot, A.J. Schut, S.A. Bouwense, M.G. Besselink, O.J. Bakker, H. van Goor, S. Holker, H.G. Gooszen, M.A. Boermeester, H.C. van Santvoort, Dutch Pancreatitis Study Group, Abdominal compartment syndrome in acute pancreatitis: a systematic review, Pancreas 43 (2014) 665–674, https://doi.org/10.1097/MPA.0000000000000198.

[4] J.E. De Laet, M.L.N.G. Malbrain, J.J. De Waele, A clinician’s guide to management of intra-abdominal hypertension and abdominal compartment syndrome in critically ill patients, Crit. Care 24 (2020) 97, https://doi.org/10.1186/s13054-020-2782-1.

[5] De Waele, J. Jan, Pancreapedia: Exocrine Pancreas Knowledge Base, 2015. Version 1.0, https://www.pancreapedia.org/exocrine-pancreas-current-concepts-of-health-and-disease/. (Accessed 1 April 2022).

[6] F. Coccolini, W. Biffl, F. Catena, M. Ceresoli, O. Chiara, S. Cimbansass, L. Fattori, A. Leppanniemi, R. Manfredi, G. Montori, G. Pesenti, M. Sugrue, L. Ansaloni, The open abdomen, indications, management and definitive closure, World J. Emerg. Surg. 10 (2015) 32, https://doi.org/10.1186/s13017-015-0026-5.

[7] J.J. De Waele, E.A. Hoste, M.L. Malbrain, Decompressive laparotomy for abdominal compartment syndrome—a critical analysis, Crit. Care 10 (2006) R51, https://doi.org/10.1186/cc4870.

[8] J.C. Duchesne, M.P. Howell, C. Eriksen, G.M. Wahl, K.V. Rennie, P.E. Hastings, N.E. McSwain Jr., M.L. Malbrain, Linea alba fasciotomy: a novel alternative in trauma patients with secondary abdominal compartment syndrome, Am. Surg. 76 (2010) 312–316.