convenient and common than cutaneous vesicostomy; however, cutaneous vesicostomy has the advantages of being associated with less pain, discomfort, and bacteriuria.[5] Importantly, both these techniques require the management of a catheter to void urine, which might be problematic in a patient with cognitive decline.

The differential diagnosis of peri-urethral infection associated with diabetes consists of abscess, NSTI including Fournier's gangrene, cellulitis, candidiasis, and folliculitis.[2] Although NSTI is uncommon, its prognosis is extremely poor, and thus it is important to recognize this condition.[6] Fournier's gangrene is characterized by severe pain, skin necrosis, foul-smelling pus, subcutaneous emphysema, obstructive arterial endarteritis, and a mixed infection with aerobic and anaerobic bacteria.[7] Swelling, pain, and erythema are reported to be characteristic of NSTI;[2,8] however, painless NSTI would occur owing to diabetic neuropathy.[7] Therefore, careful observation is essential.

Elderly patients with diabetes are at a high risk of geriatric syndrome,[9] and the mild cognitive impairment associated with diabetes can lead to a decrease in instrumental activities of daily living,[10] implying that complex medical self-care, including self-catheterization, may be difficult for some individuals. Given that an increasing number of elderly patients now live alone, we clinicians should monitor our patients carefully so as not to miss either cognitive impairment or minimal signs of infection.

Disclosure statement

The authors declare no conflict of interest.

Author contributions

Y.O. and H.N. contributed to patient care and wrote the manuscript. A.M., H.K. KY.C., A.N. and H.M. contributed to patient care and the revision of the manuscript. T.A. contributed to discussions and the revision of the manuscript.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Intravenous acetaminophen-induced non-anaphylactic shock in an older patient with COVID-19

Keywords: acetaminophen, coronavirus, COVID-19, hypotension, shock.

Acetaminophen has been available as an enteral or rectal formulation for the past two decades. After that, an intravenous (i.v.) formulation was approved in 2002 in Europe, in 2010 in the USA and in 2016 in Japan. Although i.v. administration of acetaminophen is expected to have better bioavailability than rectal or oral administration, recent data are inconsistent with i.v. acetaminophen superiority for critical illness.[1,2] During the coronavirus disease 2019 (COVID-19) pandemic, acetaminophen was more frequently used for fever reduction. Although all formulations of acetaminophen are considered relatively safe, i.v. administration is associated with an increased risk of hypotension, particularly in older patients with a hemodynamically unstable status.[3]

A woman aged in her 70s was diagnosed with COVID-19 and transferred to our hospital (Oita University Hospital, Oita, Japan) because of persistent dyspnea for 3 days. She had several pre-existing cardiovascular diseases, including patent foramen ovale and

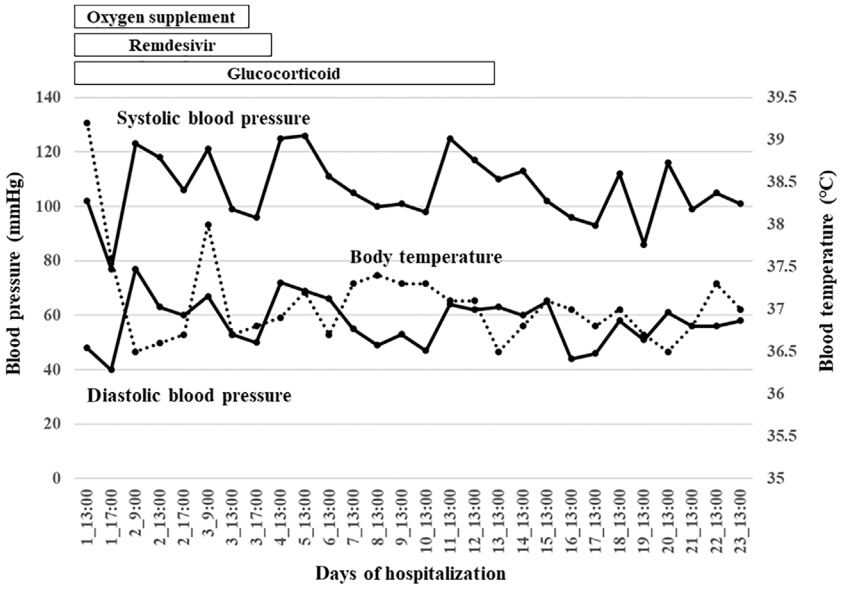

Figure 1 The record of blood pressure and body temperature of the patient during hospitalization.

ventricular septal defect, and had undergone surgery for aortic dissection 3 years earlier. Physical examination showed a body temperature of 39.2°C, an oxygen saturation (SpO_2) of 93% with supplemental oxygenation of 2 L/min, blood pressure of 102/48 mmHg, heart rate of 92 b.p.m. and impaired consciousness with the Glasgow Coma Scale of E3V4M6. Laboratory tests showed a normal leukocyte count and an elevated serum C-reactive protein level (9.77 mg/dL). Chest images showed ground-glass opacities predominantly in both lower lobes, which was consistent with COVID-19 pneumonia. Approximately 4 h after admission, the patient's consciousness level deteriorated from Glasgow Coma Scale E3V4M6 to E2V3M5. Her systolic blood pressure dropped to 70 mmHg, whereas her body temperature decreased from 39.2°C to 37.6°C (Fig. 1). However, rash and mucosal edema were not observed. We rapidly administered an i.v. infusion of 1000 mL of Ringer's acetate solution and started noradrenaline because of the lack of hemodynamic response to the infusion. Her consciousness improved a few hours later as her systolic blood pressure gradually increased.

We assessed potential causes of impaired consciousness, including cerebrovascular diseases, but no abnormal finding was observed. Considering the recovery course, the hypotension mainly was suspected to decrease the consciousness level. Blood culture was negative, and a cardiovascular specialist ruled out cardiogenic hypotension. After a detailed medical history assessment, the patient was found to have received acetaminophen (500 mg) i.v. for fever reduction 30 min before the transfer to our hospital. Furthermore, she used to take acetaminophen (250 mg) orally for chronic headaches before COVID-19 development, but she had not experienced any adverse effects. Indeed, no significant hypotension was observed when she took acetaminophen (250 mg) orally for headache 3 days after recovery from shock. As the hypotension occurred a few hours after acetaminophen administration, ruling out other potential causes, we suspected acetaminophen-induced non-anaphylactic shock. She was successfully treated and was discharged on day 25.

The patient had never been administered i.v. acetaminophen before, and hypotension was found approximately 5 h after infusion of acetaminophen. Acetaminophen-induced hypotension appears to start within 15 min, and reaches a peak approximately 60–120 min after i.v. infusion.[4] Her blood pressure was slightly low on admission, but we failed to follow up on her blood pressure until her impaired consciousness was noted by medical staff. Although viral sepsis could not be entirely ruled out as a trigger of hypotension, it was unlikely, because the severity of COVID-19 was moderate with a good clinical course.

Based on a systematic review, hypotension does not correlate with the total dose or infusion rate, i.v. infusion might be a significant risk, inducing adverse effects.[3] In a randomized control study, Kelly *et al.* showed that i.v., but not oral, administration was an independent predictor of hypotension. Patient background plays a role in predicting hemodynamic changes. Some studies found that advanced age,[5,6] lower baseline mean arterial pressure[7] and febrile illness[5] were associated with increased risk of hypotension. Furthermore, a history of cardiac surgery might contribute to acetaminophen-induced blood pressure reduction.[8] As no direct effect of severe acute respiratory syndrome coronavirus 2 on vasodilation was determined, its infection did not appear to contribute to acetaminophen-induced hypotension directly. However, the patient had several underlying cardiovascular diseases and experienced a high fever due to COVID-19, which might have been associated with an increased risk of hypotension.

Acetaminophen is commonly used as an analgesic or antipyretic agent, even for older people. However, medical workers and patients should be aware of the potential risk of acetaminophen-induced hypotension, and i.v. administration of acetaminophen needs to be avoided in high-risk patients.

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Disclosure statement

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Ethics statement

Written informed consent was obtained from the patient for the publication of this manuscript and accompanying images.

Data availability statement

The data that support the findings of this case report are available from the corresponding author, upon reasonable request.

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A senile case of heart failure associated with hypermagnesemia induced by magnesium-containing laxative agent

Keywords: constipation, heart failure, hypermagnesemia, laxative, renal insufficiency.

Dear Editor,

In the human body, magnesium (Mg) is absorbed by the small intestine and excreted from the kidneys under strict homeostatic regulation.[1,2] Long-term administration of Mg-losing diuretics and Mg-containing agents under impaired Mg homeostasis leads to hypomagnesemia and hypermagnesemia, respectively.[3] Hypermagnesemia in older adults is caused mainly by the long-term administration of Mg-containing laxatives. Yamaguchi *et al*. reported a case series of hypermagnesemia induced by Mg oxide, and speculated that hypermagnesemia occurs more frequently than previously reported.[4] The main symptoms of hypermagnesemia described in their case series are non-specific, including anorexia, weakness and drowsiness. Therefore, this pathological condition has attracted less clinical attention, but many physicians prescribe Mg-containing laxatives without monitoring serum Mg concentrations. Based on such reality, Yamaguchi *et al*. concluded that senescence and renal insufficiency are the risk factors of hypermagnesemia in patients prescribed Mg oxide.[4] However, a cardiovascular manifestation of hypermagnesemia is not described in this literature. Here, we report an older patient with hypermagnesemia leading to heart failure (HF).

A 99-year-old demented, constipated and hypertensive man was introduced to Hara Doi Hospital, Fukuoka, Japan, due to the request for hospitalization by the nursing facility because of gradual hypotension (systolic blood pressure of 70–80 mmHg) and bradycardia (heart rate of 30–40 b.p.m.). On admission, he complained of general fatigue and anorexia, but showed no abnormal physical findings. Amlodipine (5.0 mg/day) and memantine (10 mg/day) were terminated. Blood pressure and heart rate were 110–46 mmHg and 44 b.p.m., respectively. Body temperature and SpO_2 were 36.1°C and 98% at room air. Because blood chemistry showed anemia, renal dysfunction, elevated brain natriuretic peptide and hypermagnesemia, administration of Mg oxide (3000 mg/day, ×3) was terminated, sennoside was started as an alternative laxative, and drip infusion of potassium- and Mg-free solution (500 mL/day) was initiated. The main physical and laboratory findings are shown in Table 1. Chest X-ray showed cardiomegaly (cardiothoracic ratio of 62.0%), but no pulmonary congestion or effusion. Electrocardiogram on admission showed sinus bradycardia associated with second-degree sinoatrial block (longest RR interval 1.8 s, and basic HR 36 b.p.m.), left axis deviation (QRS axis of −34°) and complete right bundle branch block. However, the sinoatrial block disappeared in an electrocardiogram recorded on the next day of admission. Echocardiogram recorded on that day showed an ejection fraction of 68%. The patient was discharged after confirming the restoration of hypermagnesemia.

HF is defined as a pathological state in which cardiac output is insufficient to meet the oxygen demands and metabolic needs of vital organs. The clinical events exacerbating HF are