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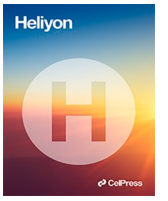

Case report

Point-of-care ultrasound in the diagnosis and treatment of Budd-Chiari syndrome: A rare case report and literature review

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ABSTRACT

Budd-Chiari syndrome (BCS) is a life-threatening disease characterized by the partial or complete obstruction of hepatic venous outflow anywhere from the liver to the heart. In China, secondary BCS is rare. We present a case of secondary BCS caused by compression of the suprahepatic inferior vena cava (IVC), mainly due to local bile accumulation in the caudate lobe of the liver. This case highlights the scarcity of secondary BCS worldwide and the importance of point-of-care ultrasound (POCUS) in the diagnosis and treatment, especially in critical and comatose patients. Prompt diagnosis and recanalization with POCUS-guided puncture and drainage help improve patient prognosis.

1. Introduction

Due to its rarity, Budd-Chiari syndrome (BCS) is an overwhelmingly misdiagnosed vascular disorder. It occurs at the levels of the accessory hepatic veins (HVs), the ostial opening of the HV into IVC, and the suprahepatic IVC (a portion of the IVC above the level of the hepatic venous confluence) [1]. Obstruction caused by deformity, tumor compression, or venous thrombosis, to varying degrees, results in congestive hepatosplenomegaly and portal hypertension syndrome [2–5]' which can be classified as primary or secondary depending on the causes [5]. A Meta-analysis revealed that the pooled annual prevalence of BCS was 11 per million and the pooled incidence of BCS was 1 per million [6]· Secondary BCS accounts for only 0.26 % of all BCS cases in China [7,8].BCS is associated with high mortality with 90 % at 3 years [9], despite the improvement in prognosis in the past decade due to early diagnosis and appropriate management [10–12].

The diagnosis of BCS is based on the clinical manifestations, laboratory studies, and radiological findings. Clinical presentations and laboratory studies of BCS are extremely heterogeneous and non-specific; therefore, owing to its rarity, the diagnosis of BCS is easy to ignore. The key feature for the diagnosis of BCS is obstruction of the hepatic venous outflow. Color Doppler ultrasonography

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(CDUS), computed tomography (CT), and magnetic resonance imaging (MRI) have proven to be efficient in the diagnosis of BCS [13]. The guidelines recommend CDUS as the first choice for examination as it is useful and convenient for depicting the position of the obstruction and guiding further treatment for BCS [14]. Point-of-care ultrasound (POCUS) can be performed by clinicians who provide care at the patient's bedside, especially for the timely diagnosis of critically ill patients. It is a crucial and valuable tool for the rapid bedside identification of potential disorders and is important for the guidance of various invasive operations.

We describe a case of secondary BCS to pancreatitis with the involvement of the IVC, which was compressed by a cystic mass in the caudate lobe of the liver, to enrich the literature on rare cases of secondary BCS, providing new ideas, especially in critical patients. We conclude that POCUS plays an important role in the rapid recognition of BCS and guiding treatment with positive outcomes.

2. Case presentation

A 34-year-old man with a 1-month history of limb paralysis and loss of consciousness was admitted to our hospital for rehabilitation. He suffered from severe hemorrhage in the right basal ganglia and brainstem while receiving surgical therapy at a local hospital. He developed secondary cerebellar infarction after surgery, mainly due to brain edema and ischemic and hypoxic injuries. The patient had a medical history of immunoglobulin A (IgA) nephropathy for 15 years, and was managed with glucocorticoids and herbs; however, he stopped all treatments himself 6 years prior. Furthermore, he had chronic kidney dysfunction and hypertension for 5 years without regular therapy and monitoring, and no history of hepatitis, jaundice, or alcohol consumption.

Upon admission, vital signs revealed a high fever with a body temperature of 39 °C and a heart rate of 134 beats per minute. On physical examination, the patient was on mechanical ventilation with a tracheotomy tube and had a Glasgow Coma Scale (GCS) score of 3 points. Abdominal distension was observed without edema of the extremities.

Laboratory examinations revealed severe anemia with a blood hemoglobin (Hb) level of 60 g/L (normal range 130~175 g/L), a total leukocyte count (WBC) of 10.91 $\times$ 10^9/L (normal range 3.50–9.50 $\times$ 10^9/L), and a high-sensitivity C-reactive protein (hs-CRP) level of 55.8 mg/L (normal range $\leq$10.0 mg/L). Liver function showed that serum albumin (ALB) was 32.6 g/L (normal range 40~55 g/L), serum glutamic-pyruvic transaminase (ALT) was 244 U/L (normal range <50U/L), total bilirubin was 15.1 μmol/L (normal range 3.4–24.0 μmol/L), direct bilirubin was 5.6 μmol/L (normal range $\leq$6.8 μmol/L), alkaline phosphatase (AKP) was 128 U/L (normal range 45~125 U/L), and serum amylase (AMY) was 126 U/L (normal range 30~110 U/L). The coagulation function was as follows: prothrombin time (PT) 13 s (normal range 9.1–12.3 s), international normalized ratio (INR) 1.13 (normal range 0.85–1.20), and activated partial thromboplastin time (APTT) 31 s (normal range 21.6–32.4 s).

A nasogastric tube was inserted for distention of the abdomen, and 700ml of blood was quickly drained. The patient was arrested during the investigation, and the Hb level decreased to 44 g/L. Fortunately, the patient recovered successfully after 15 min of resuscitation efforts, including chest compressions, aggressive intravenous fluid resuscitation, blood transfusion, and epinephrine and norepinephrine administration. Urgent bedside esophagogastroduodenoscopy (EGD) revealed a bleeding ulcer in the post-bulbar duodenum combined with many large blood clots, and endoscopic hemostasis was achieved with hemoclips (Fig. 1(A–C)). Bedside POCUS (ultrasound assessment of the abdominal viscera was performed using a 3–5 MHz convex array transducer (Mindray, M8 super)) revealed a normal liver, gall bladder, pancreas, and spleen, although the images showed interference by gas, and no obvious ascites were found.

Unfortunately, bleeding recurred the next day, and ultimately stopped after transcatheter gastroduodenal embolization. However, the blood analysis revealed elevated pancreatic enzymes (serum AMY 4786 U/L, serum lipase 4653 U/L) and hyperglycemia (270 mg/dl), and abdominal CT confirmed acute pancreatitis with a small amount of ascites after embolization on day 2. It was managed with fasting, aggressive fluid therapy, and somatostatin to inhibit the secretion of pancreatic enzymes, along with intravenous short-acting insulin to control blood sugar levels. However, the levels of total bilirubin, especially direct bilirubin, were elevated on day 6 (Table 1), and POCUS revealed a large amount of ascitic fluid. Percutaneous peritoneal drainage was performed, and the fluid tests indicated inflammatory ascites with yellow cloudy ascites, a high leukocyte count (12000/μl) with a neutrophil ratio of 83 %, and a high total level of protein (2700 mg/dl). Piperacillin with tazobactam (Tazocin, Wyeth Lederle S.R.L.) 4.5g intravenously over 2 h, four times daily was prescribed. Eventually, the pancreatic enzyme levels gradually normalized 8 days later, and ascites improved, as evidenced

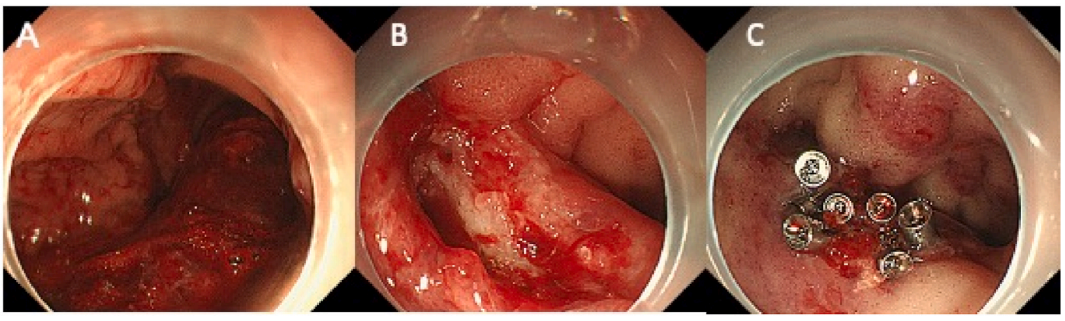

Fig. 1. Endoscopic images of ulcer and hemostasis using hemoclips in the post-bulbar duodenum.
A: many large blood clots in the duodenum; B: a large bleeding ulcer in the post-bulbar duodenum; C: bleeding stopped with hemoclips.

Table 1
Changes of laboratory findings.

Time	Day 1	Day 2	Day 6	Day 8	Day 10	Day 11	Day 14	Day 19	Day 21	Day 24
ALB (g/L)	32.6	27.1	29.3	29.2	32.6	31.9	29.9	29.5	26.5	26.5
ALT (U/L)	244	1636	1360	452	182	192	83	32	26	11
total bilirubin (μmol/L)	15.1	19.6	43.7	48.7	78.5	94.8	141.8	84.7	54.5	28.0
direct bilirubin (μmol/L)	5.6	8.9	23.1	27.5	42.2	51.4	120.2	51.7	31.5	11.1
AKP (U/L)	128	130	–	166	–	264	–	566	–	267
PT (s)	13	19.3	14.8	15.2	13.2	12.1	13.4	15.6	14.8	15.6
INR	1.13	1.84	1.41	1.45	1.26	1.15	1.28	1.49	1.41	1.49
APTT (s)	31	37.1	49	42.9	45.0	41.5	39.7	53.6	46.8	42.7
serum AMY (U/L)	126	4786	294	94	73	–	–	–	–	–
serum lipase (U/L)	–	4653	130	58	27	–	–	–	–	–

Abbreviation: ALB: serum albumin; ALT: glutamic-pyruvic transaminase; AKP: alkaline phosphatase; PT: prothrombin time; INR: international normalized ratio; APTT: activated partial thromboplastin time; AMY: amylase. —: the data was not tested on that day.

by laboratory results of ascites and abdominal POCUS.

However, on day 14, the patient developed severe bilateral pitting edema in the lower extremities, with no edema in the upper limbs. On physical examination, the liver was palpitation 1 cm below the costal margin, and the spleen was not palpable. Laboratory findings showed that the serum total bilirubin had increased to 141.8 μmol/L, and the direct bilirubin had increased to 120.2 μmol/L, with normal levels of transaminase at that time (Table 1). Serological tests for acute viral hepatitis were negative. Examinations for anti-neutrophil cytoplasmic antibodies, antinuclear antibodies, and antiphospholipid antibody levels were also negative. Bedside POCUS indicated a cystic lesion in the caudate lobe of the liver measuring 72 mm × 60 mm × 45 mm, with compression over the suprahepatic IVC and no dilatation of the HVs (Fig. 2A, Video 1). Furthermore, the compression of suprahepatic IVC was obvious and confirmed using CDUS (Fig. 2B–Video 2). Therefore, a diagnosis of secondary BCS was considered. The patient underwent an abdominal CT with contrast to confirm the diagnosis, demonstrating a cystic mass compressing the adjacent IVC (Fig. 3). POCUS-guided transhepatic puncture drainage of the cystic lesion was performed successfully, and approximately 35 ml of bile-like liquid was extracted (Fig. 4(A and B)). Recanalization of venous outflow in the IVC was observed immediately after puncture (Video 3, Video 4). The biochemical analysis of the fluid aspirated from the lesion confirmed that it was bile in origin (the levels of total bilirubin and direct bilirubin in the fluid were 503.5 μmol/L and 118.4 μmol/L, respectively). The edema of the bilateral lower extremities improved 3 days later (day 17), and a non-contrast abdominal CT scan showed no cystic lesion, or compression of the IVC on day 21 (Fig. 5). Serum bilirubin became normal 10 days after puncture (day 24).

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3. Discussion

The causes of BCS differed between Western and Eastern countries [15]. Thrombogenesis is the most common cause of BCS in Western countries; however, thrombotic risk factors are rare in China [16]. IVC obstruction and a combination of IVC and hepatic obstruction are the main types of obstructions in China [17–19]. Secondary BCS is due to an obstruction originating outside the vein, i.e. the compression or invasion by a benign mass or malignant disorder that blocks the hepatic blood outflow [2,20]. It was rare in China which only formed less than 1 % of the total cases in China [21]. We propose that early recognition, rapid diagnosis, and adequate treatment can predict a favorable prognosis, contributing to the clinical data and appealing for more attention to this disease.

The diagnosis of BCS is difficult, especially in comatose patients. A delay of 6 months from the onset of symptoms before the diagnosis of BCS was noted in most patients in a recent study [22]. The clinical manifestations and laboratory abnormalities are diverse and non-specific, and appropriate 20 % of patients are asymptomatic. In a study involving 45 patients with BCS, a combination of specific signs on radiological imaging with symptoms and laboratory examinations offered the highest predictive value, with a

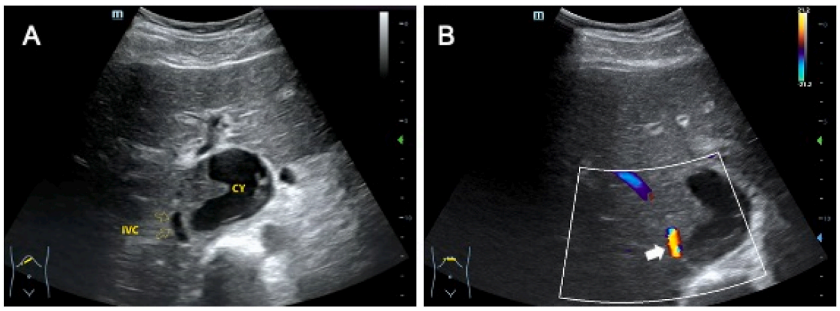

Fig. 2. The compressed IVC due to the cystic lesion in hepatic caudate lobe. A: The compressed suprahepatic IVC on B-mode ultrasound showing the narrow lumen of the IVC; B: The compressed IVC on color Doppler ultrasound showing colorful blood flow. Yellow arrow and white arrow: the compressed IVC on B-mode ultrasound and color doppler ultrasound respectively. Abbreviation: IVC: inferior vena cava; CY: cyst.

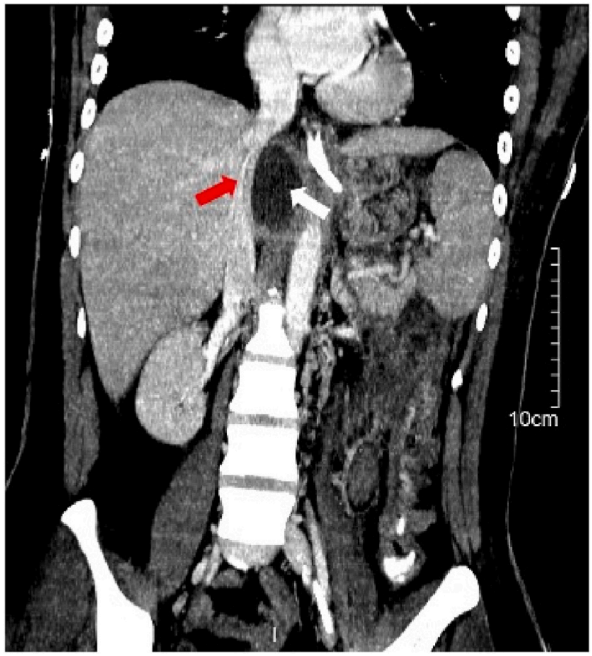

Fig. 3. Coronal abdominal CT with contrast demonstrating a cystic mass (white arrow) compressing adjacent suprahepatic IVC (red arrow). Abbreviation: IVC: inferior vena cava.

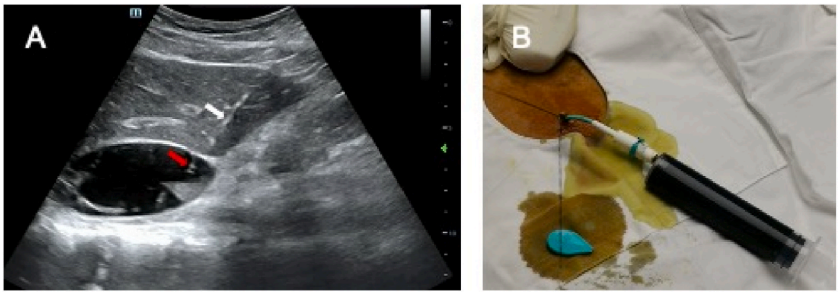

Fig. 4. Successful puncture of the cystic lesion in the caudate lobe of the liver and the fluid extracted from the lesion. A: Successful puncture of the cystic lesion; B: Fluid extracted from the lesion. White arrow and red arrow: the tube through the liver into the cystic lesion.

specificity of 100 % [23]. Consensus guidelines suggest that the diagnosis of BCS relies on a combination of clinical manifestations and imaging findings, and both the clinician and radiology team should maintain a high index of suspicion in patients with unexplained portal hypertension [8,14].

CDUS is an excellent first-line imaging modality for patients undergoing BCS. A recent meta-analysis revealed that the pooled sensitivity and specificity of CDUS for the diagnosis of BCS were 89 % and 68 %, respectively. The pooled sensitivity of Doppler was higher than that of CT, the latter was higher than that of MRI [24]. CT had a diagnostic accuracy of 89.3 % and 80.2 % in detecting BCS, and 83.4 % of cases correctly correlated with MRI according to the initial imaging analysis of BCS in the Henan province of China [19]. The combination of CDUS with CT or MRI increases the accuracy of diagnosis to 100 % [18]. The advantages of CDUS include easy availability, low cost, reproducibility, lack of radiation and multi-planar imaging, and the ability to provide functional information regarding vascular flow, stenosis, or thrombosis. Bedside CDUS is more convenient than CT or MRI, especially for critically ill patients; however, traditional bedside CDUS is ordered by a clinician, and performed by a sonographer who is not directly involved in patient care. Thus, POCUS which can be performed by clinicians, is rapidly developing and used in multiple medical specialties, including emergency room, intensive care units, trauma, cardiology, and radiology. It can help in screening and diagnosing illnesses, guiding interventions, and monitoring changes in diseases during clinical decision-making [25]. Boling et al. described the basic approaches for POCUS examination of abdominal organs, such as the biliary tract, liver and kidneys, as well as the identification and management of intra-abdominal free fluid [26]. Imaging-guided therapeutic procedures have been supported as the mainstay of clinical practice to reduce morbidity and improve safety [27]. POCUS was successfully performed for diagnosis, differential diagnosis, and treatment in the absence of self-reported symptoms.

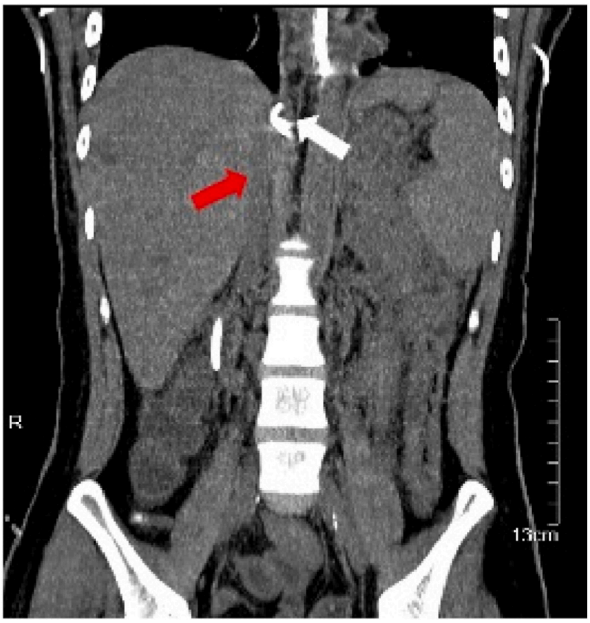

Fig. 5. Non-contrast abdominal CT scan showed no cystic lesion or compression of IVC anymore. White arrow: the tube retained in the lesion; Red arrow: the normal IVC without compression. Abbreviation: IVC: inferior vena cava.

In terms of prognosis and treatment, the survival rate of BCS is 87 % at 1 year and 82 % at 2 years, and the 5-year survival rate is >80 % owing to early recognition, prompt diagnosis, and adequate management [8,10,28]. A stepwise treatment approach for BCS has resulted in favorable long-term survival [10,28]. Secondary BCS caused by IVC compression is a potentially treatable form of BCS. Recanalization was effective in symptomatic BCS with membranous or segmental obstruction; it was also satisfactory with a low incidence of complications [11,14,29]. In this case, the edema of the bilateral legs and laboratory tests recovered successfully due to POCUS-guided recanalization of the hepatic venous outflow, which was convenient, efficient and accurate in this critical patient.

The causes of acute pancreatitis are unclear and interesting. POCUS initially excluded pancreatitis. Abdominal CT had not been performed before he visited our hospital because there were no indications. Abdominal CT was subsequently performed at our hospital because his vital signs were unstable, and serum lipase was not tested late at night. The etiology of acute pancreatitis may be the obstruction of the pancreatic duct by large blood clots in the duodenum, as demonstrated by EGD. Another possibility might be correlated with gastroduodenal embolization, the mechanism of which is likely to be ischemia, hemorrhagic shock, and cardiac arrest, which may have exacerbated this process. Gastroduodenal embolization is considered a safe and effective treatment for patients at high surgical risk for endoscopy-refractory nonvariceal upper gastrointestinal bleeding [30]. Duodenal ischemia and hepatic infarction from non-target embolization are common complications of gastroduodenal embolization [7]. Pancreatitis is a rare, but life-threatening, post gastroduodenal embolization complication. Han et al. reported a rare case of new onset pancreatitis and a pseudocyst 10 weeks after the embolization of a gastroduodenal artery aneurysm [31]. Wei Ming Chua [32] also described a rare case of acute focal pancreatitis following gastroduodenal embolization, which was highly similar to that observed in our patient. It is crucial to identify trials of disorders mainly based on clinical signs and radiological findings in comatose patients.

During treatment, the patient developed jaundice and bilateral lower-extremity edema. Liver dysfunction with consequent cholestasis is one of the many complications that occur in up to 40 % of critically ill patients [33]. In the current study, POCUS was used to diagnose and differentiate the possible causes and revealed a cystic mass with bile compressing the suprahepatic IVC. No dilation or congestion of the HVs was found on ultrasonography because the junction of the IVC and right atrium was not compressed by the cystic mass, and the disorder had an acute onset. Deep vein thrombosis and biliary duct obstruction were excluded by POCUS. POCUS-guided puncture was initiated immediately to ensure the safety and effectiveness of the procedure. The dynamic changes in the cystic lesion and compressed IVC were monitored using ultrasound, showing the recanalization of the venous outflow in the IVC. POCUS plays an important role in the diagnosis, treatment, and monitoring of secondary BCS.

4. Conclusion

BCS is largely underdiagnosed owing to its atypical and heterogeneous clinical presentation, signs, and laboratory findings. The diagnosis of BCS relies on a combination of clinical manifestations and imaging findings. Recanalization is the mainstay for the management of secondary BCS. This case suggests that clinicians should be aware of this disease in patients with acute or chronic liver dysfunction, especially when the etiology is unknown. POCUS plays an important role in the identification, diagnosis, and guidance of appropriate treatments for BCS, especially in critically ill patients.

Ethics statement

Our study was approved by the Ethics Committee at Zhejiang Provincial People's Hospital (approval number is ZJPPHEC2023O (342)) by the ethical standards of the Helsinki Declaration. The written and informed consent for publication of this case and any details was acquired from the patient's brother as a legal guardian because the patient was unconscious, and the informed consent for publication of any details was also obtained if he regained consciousness in the future. The patient's brother consented to the publishing of all images, clinical data, and other data included in the report. We strictly abided by data protection legislation and ethical standards, patient's personal information such as name, gender, and hospitalization number won't appear in terms of images, laboratory examination of our article.

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Data availability statement

ALL authors confirm that the data included in this study are available in the article.

CRediT authorship contribution statement

Lingzhi Jiang: Writing – original draft, Project administration, Conceptualization. **Ligang Wang:** Project administration, Conceptualization. **Mingshan Wang:** Methodology, Investigation, Data curation. **Yuyun Xu:** Methodology, Investigation, Data curation. **Ye Shen:** Methodology, Investigation, Data curation. **Xiangming Ye:** Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.heliyon.2024.e36192.

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