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CASE REPORT

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Possible iodophor-induced systemic iodine toxicity during transurethral seminal vesiculoscopy: a case report and literature review

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Abstract

Rationale Iodophor, a disinfectant composed of iodine complexed with a surfactant, is extensively employed for preoperative skin preparation, burn wound management, and mucosal disinfection. Despite its extensive application in clinical practice, emerging clinical evidence has documented associations between iodophor irrigation and acute kidney injury in human. However, systemic iodine toxicity after the seminal vesicle was irrigated with iodophor during transurethral seminal vesiculoscopy (TUSV) has not been previously described.

Patient concerns We report the case of a 70-year-old man who developed transient hypotension, vomiting, confusion, and profound lactic acidosis after the seminal vesicles were irrigated with iodophor.

Diagnoses The patient was diagnosed with confusion and profound lactic acidosis secondary to the seminal vesicle was irrigated with iodophor.

Interventions The patient was admitted to the intensive care unit (ICU), and continuous venovenous haemodiafiltration (CVVH) was initiated 3 h after ICU admission.

Outcomes After six days, the patient's condition improved, allowing the cessation of CVVH. He was discharged from the hospital on the 22nd day after being transferred to the ICU.

Conclusion This case highlights the typical characteristics of iodophor absorption during its regular application. Here, we report the first case of systemic iodine toxicity caused by iodophor irrigation during transurethral seminal vesiculoscopy.

Keywords Iodine toxicity, Lactic acidosis, Metabolic acidosis, Transurethral seminal vesiculoscopy, Case report

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Introduction

Iodophor, a commonly used antiseptic in clinical practice, can eliminate bacteria, fungi, and certain viruses. As a result, iodophors are employed in scenarios such as preoperative cutaneous antisepsis, injection site preparation, and genitourinary tract disinfection [1–2]. Nevertheless, iodophors are linked to serious adverse events such as iodine-induced acute renal failure, iodine-induced hyperthyroidism, and systemic iodine toxicity [3–7]. Notably absent from existing literature are documented cases of iodine toxicity secondary to seminal vesicle irrigation during transurethral seminal vesiculoscopy (TUSV). We present a clinically significant case of high anion gap metabolic acidosis with lactic acidosis following intraoperative iodophor irrigation during TUSV, marking the first documented instance of this complication in urological endoscopic procedures.

Case presentation

A 70-year-old Chinese male (66 kg, 164 cm; American Society of Anesthesiologists, status II) with a 1-year history of refractory haematospermia was scheduled for transurethral seminal vesiculoscopy. Aside from eczema, his medical history was unremarkable. Chest computed tomography (CT) revealed multiple microscopic nodules in the upper lobes of the left and right lungs. Preoperative electrocardiography, echocardiography, vein ultrasound of the lower extremities, and laboratory tests revealed no obvious abnormalities.

Routine monitoring included noninvasive blood pressure, 5-lead ECG, and SpO_2 monitoring, which were initiated after the patient entered the operating room. The patient's vital signs were as follows: blood pressure (BP), 126/82 mmHg; heart rate (HR), 55–65 bpm; and SpO_2, level, 97–99% before anaesthesia. At the L3–4 intervertebral space, combined spinal–epidural anaesthesia (CSE) was performed uneventfully. Two millilitres of 0.5% bupivacaine were slowly administered in the subarachnoid space after cerebrospinal fluid was observed, and the catheter was inserted 4 cm into the epidural space. The level of blocking of the sensory system was fixed at T8. After the administration of CSE anaesthesia, the patient's vital signs were stable (BP, 130/80 mmHg; HR, 57 bpm; and SpO_2, 99%). During the procedure, the patient's vital signs were maintained at a BP of 122–130/60–75 mmHg, a HR of 57–62 bpm, and a SpO_2 of 99–100%.

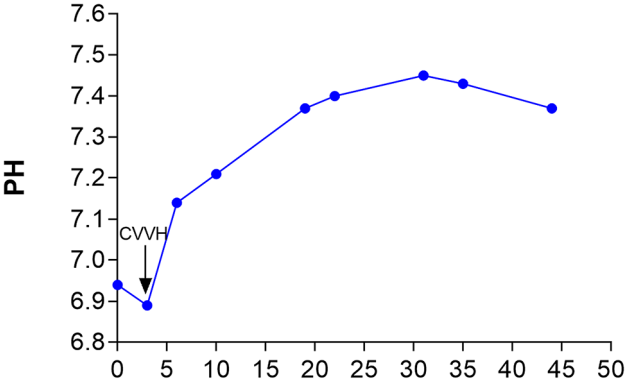

Fig. 1 Change in arterial pH over the 44 hours after the patient was transferred to the ICU. After initiating continuous venovenous hemodiafiltration (CVVH), the patient's metabolic acidosis improved rapidly. Arrow denotes the time of initiation of CVVH

The doctor identified the opening of the prostatic utricle at the verumontanum and entered the prostatic utricle through this opening. Then the seminal vesicle cavity was penetrated through the weak mucosa side channel, guided by the guidewire. The operation was uneventful until the surgeon irrigated the seminal vesicle with 200 ml of iodophor solution diluted with normal saline (1:1) within minutes. Hypotension developed within a few minutes after irrigation, and ephedrine (6 mg IV) was quickly administered to resolve it. Vomiting, lethargy, and confusion developed, and arterial blood gas (ABG) analysis was conducted immediately. ABG analysis revealed a pH of 7.40, a PCO_2 of 32 mmHg, a bicarbonate level of 19.8 mmol/L, a BE of – 4.1 mmol/L, and a lactate level of 6.1 mmol/L. Volume expansion was initiated immediately with 500 ml of normal saline solution. Twenty minutes later, the ABG analysis revealed lactic acidosis with a pH of 7.34, a PCO_2 of 33 mmHg, a bicarbonate level of 17.8 mmol/L, a BE of -7 mmol/L, and a lactate level of 8.2 mmol/L. Sodium bicarbonate (5%, 100 mL) was used to promptly address lactic acidosis. Sudden vomiting and severe agitation recurred, and the patient was quickly transferred to the ICU with subsequent monitoring and treatment.

ABG and laboratory indices were measured instantly in the ICU, and the ABG revealed a pH of 6.94 and a lactate level above the maximum measurement level (maximum 20 mmol/L), a BE of -22.79 mmol/L and an anion gap of 30.3 mmol/L. The laboratory data revealed a serum creatinine level of 113.3 μmol/L, impaired liver function (AST level, 226.8 U/L; ALT level, 118.5 U/L), and an lactate dehydrogenase (LDH) level of 633 U/L. Within hours, the patient developed extremely severe lactic acidosis and impaired liver and kidney function. Intubation and mechanical ventilation were required due to continuous deterioration. Three hours after ICU admission, continuous venovenous haemodiafiltration (CVVH) was initiated to correct severe lactic acidosis and acute renal injury. Fortunately, 44 h of CVVH therapy alleviated the acidosis, with a pH of 7.37, a $PaCO_2$ of 37 mmHg, a PaO_2 of 133 mmHg, a BE of -3.9 mmol/L, and a lactate level of 2.5 mmol/L according to ABG analysis (Figs. 1, 2 and 3). CVVH was stopped on the 6th day after admission to the ICU, and the patient was weaned from the ventilator on the 7th day. With supportive care, the patient's

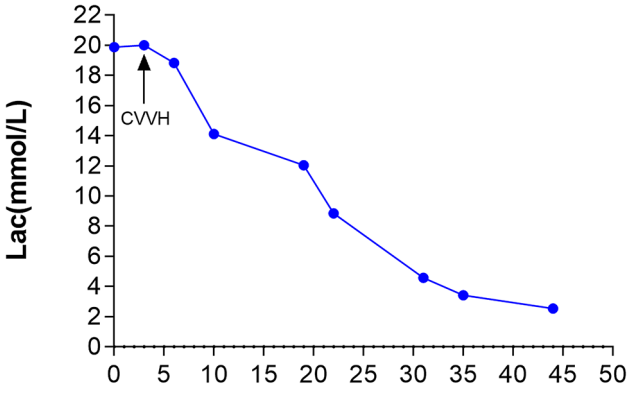

Fig. 2 Change in arterial lactate (Lac) over the 44 h after the patient was transferred to the ICU. On admission to the ICU, blood lactate was above the level of detection (maximum 20 mmol/L). After initiating continuous venovenous hemodiafiltration (CVVH), the patient's metabolic acidosis improved rapidly. Arrow denotes the time of initiation of CVVH

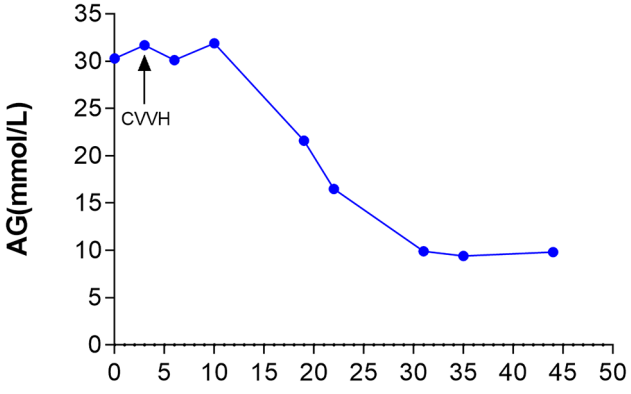

Fig. 3 Change in arterial anion gap (AG) over the 44 h after the patient was transferred to the ICU. After initiating continuous venovenous hemodiafiltration (CVVH), the patient's metabolic acidosis improved rapidly. Arrow denotes the time of initiation of CVVH

clinical condition improved, and he was discharged from the hospital on the 22nd day after admission to the ICU. Upon reviewing the intra- and perioperative courses, a possible reason for this clinical state might be iodine toxicity from abundant iodine absorption in the operative field. However, no relevant tests were performed in our hospital, and the patient's serum iodine concentration was not measured. The patient remained well after six months of follow-up.

Discussion

Seminal vesiculoscopy is the best way to examine and treat seminal vesicle gland diseases, and it is widely used in andrology because of its clear curative effect and few related surgical complications. [8–9] A typical manifestation of the seminal vesicle is honeycomb-like tissue, and there are three ways to enter the seminal vesicle. Whatever pathway to the seminal vesicles is used, the surgeon needs to examine every seminal vesicle cavity [10]. When haemorrhage is observed in the seminal vesicles, it is necessary to irrigate them out until no haemorrhage remains. Currently, irrigation solutions include antibiotics, normal saline and iodophor solutions for seminal vesiculoscopy.

Iodophor is a disinfectant solution consisting of a complex formed by iodine and a surfactant with an effective iodine content of 4.5–5.5 g/L (0.45–0.55% W/V), which is widely used for disinfect surgical sites and burn wounds. Despite the wide use of iodophors, the systemic absorption of iodine may lead to serious adverse reactions and even death. Our patient experienced transient hypotension, vomiting, confusion, and extremely severe lactic acidosis after the seminal vesicles were irrigated with iodophor. Previous reports have presented some cases of iodine toxicity resulting from iodophor irrigation [11–15] (Table 1). However, none of these cases occurred during transurethral seminal vesiculoscopy. Although there have been some reports of acidosis resulting from iodine absorption, the precise mechanism has not been clarified. A previous study proposed three mechanisms: (1) free iodine binds to bicarbonate, causing the consumption of serum bicarbonate; (2) the very different pH values of iodophor and the human body result in acidosis; and (3) acid excretion by the kidneys is impaired due to iodine toxicity [16]. Roland et al. suggested that lactic acidosis reflects a toxic effect of iodine/iodide that interferes

Table 1 Previous cases of iodine poisoning due to iodophor irrigation

No.	Author year	Ref no.	Age (yr)	Sex	Iodine source	Clinical presentation
1	Kanakiriya et al. 2003	[11]	45	male	mediastinal irrigation with povidone-iodine	Renal and hepatic dysfunction
2	Labbé et al. 2003	[12]	68	male	subcutaneous irrigation with povidone-iodine	Cardiac conduction abnormalities, lactic acidosis, acute renal failure, hypocalcemia, and hypothyroidism
3	Glick et al. 1985	[13]	3	boy	mediastinal irrigation with povidone-iodine	metabolic acidosis developed associated with alternating episodes of lethargy and agitation cardiac insufficiency
4	Ramaswamyka-nive et al. 2011	[14]	67	male	used as an irrigant during a three-hour procedure	cardiovascular collapse, metabolic acidosis, renal failure and seizure
5	Ryan et al. 1999	[15]	57	female	mediastinal irrigation with povidone-iodine	acute renal failure

in some way with adenosine triphosphate generation through oxidative metabolism [17].

Iodophors can lead to systemic iodine toxicity via absorption through any route. The extent of iodine absorption depends on the site and area where iodine is applied; mucosal absorption is high, as is absorption through wounds [18]. During seminal vesiculoscopy, it is necessary to repeatedly irrigate the inflammatory material with iodophor. The procedure requires entry of the seminal vesicles from the small prostate sac. Although there is no sinus opening for direct absorption, the angle needs to be changed during the operation, which can cause bleeding and great damage to the seminal passage. In addition, the operative space in seminal vesicles is limited and closed, and the seminal vesicle tissue is honeycomb-like with multiple irregular small spaces, so more lavage solution may be absorbed [19]. When iodophor is absorbed, it may lead to refractory high anion gap metabolic acidosis and lactic acidosis because of the high serum iodine concentration. Lactic acidosis is associated with increased anion gap metabolic acidosis and is broadly classified into types A and B. Type B lactic acidosis is further subdivided into types B1, B2, and B3, and type B2 lactic acidosis is associated with the use of drugs or toxins. [20–21] Therefore, we inferred that this patient had high anion gap metabolic acidosis resulting from type B2 lactic acidosis.

Fortunately, the patient was quickly transferred to the ICU, which ensured that he received timely and effective treatment without experiencing serious complications. On the basis of the signs and symptoms caused by iodine toxicity, the clinicians could recognize iodine toxicity even when iodine levels are not tested. The signs and symptoms reportedly caused by iodine toxicity include a transient hypotension, nausea, vomiting, confusion, and a more significant than expected metabolic acidosis, hypernatremia, hepaticdysfunction, renal failure, and thyroid abnormalities [11]. Despite the lack of specificity of each sign, their combination following an administration of iodophor should prompt the discontinuation. Kim et al. [22] suggested that increased urine iodine concentration and tan casts in the renal tubules may be helpful diagnostic characteristics of such cases, and they recommend kidney biopsy as a useful tool for diagnosing iodine toxicity. Therefore, urine iodine concentration and kidney biopsy could be used for diagnosing iodine toxicity when serum iodine concentration was not tested.

Limitations

The limitations of this study are as follows. First, no relevant tests were performed in our hospital, and the patient's serum iodine concentration was not tested. However, on the basis of the patient's clinical presentation and the timing of these symptoms, we inferred that the patient might have developed systemic iodine toxicity. In the future, a serum iodine test should be performed for patients who show unexplained metabolic abnormalities or organ dysfunction after the use of iodophors. Second, owing to a lack of experience, the diagnosis of iodine toxicity was not considered after hypotension was resolved, and the unforeseen rapid deterioration of the internal environment in the subsequent period was not anticipated. With respect to the possible serious consequences of iodophor irrigation, we believe that our case has important value in ensuring that many anaesthesiologists are vigilant about iodophor irrigation during seminal vesiculoscopy.

Conclusions

We reported the clinical course and management of a 70-year-old man who developed systemic iodine toxicity after the seminal vesicles were irrigated with iodophor during transurethral seminal vesiculoscopy. The present report illustrates that severe adverse events may be linked to the internal use of iodophor; therefore, extreme caution should be taken when iodophor solution is used to irrigate seminal vesicles since overabsorption of iodine can result in toxic serum levels and systemic iodine toxicity. In the future, the clinicians should decrease the height of irrigation and reduce the speed of irrigation, and low-pressure solution flushing should be used to remove the haematocele from the seminal vesicle during seminal vesiculoscopy. Moreover, prompt CVVH initiation in the presence of iodophor-induced lactic acidosis may enhance clearance of serum iodine concentrations, thereby potentially mitigating progressive damage and associated complications.

Abbreviations

TUSV	Transurethral seminal vesiculoscopy
ECG	Electrocardiogram
HR	Heart rate
BP	Blood pressure

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12894-025-01787-7.

Supplementary Material 1

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Author contributions

Conceptualization: Jianli Li. Data curation: Meng Zhang, Nan Zhao. Investigation: Meng Zhang, Huanhuan, Zhang. Writing-original draft: Meng Zhang.

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Data availability
All the original research data, including the results, tables, and figures, are available.

Declarations

Ethical approval and consent to participate
The study has been approved by the ethics committee of the Hebei General Hospital (ethics approval no.2024022).

Consent for publication
the patient provided written informed consent for publication of this case report.

Competing interests
The authors declare no competing interests.

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