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Case report

Anesthetic management of a patient with 10 l of blood loss during operation for a retroperitoneal mass

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Abstract Bleeding is a common problem during resection of a retroperitoneal mass. Massive bleeding may occur in case of injury of an adjacent major vessel or organ. This case report describes a successful anesthetic management of a patient with 10 l of blood loss within three hours surgery. A 44-year-old woman who underwent an operation for resection of a retroperitoneal mass, went to a hypovolemic shock, due to acute life-threatening intra-operative bleeding, and was successfully rescued with a combination of measures, including control of surgical bleeding, supportive treatment with rapid fluid infusion, massive transfusion of blood products and administration of intravenous vasoactive agents for maintaining tissue perfusion and oxygenation, utilizing intraoperative autologous blood salvaged via cell saver, as well as prevention and treatment of complications. The patient received a total of 22 units of Packet Red Blood Cells (PRBCs), 18 units of Fresh Frozen Plasma (FFP), 10 units of cryoprecipitate, 3750 ml of her own salvage blood. Postoperatively, she was transferred to the intensive care unit (ICU) with mechanical ventilator support, where she received another 5.4 units of FFP, 10 units of cryoprecipitate. The patient developed features of early acute lung injury such as fever and hypoxemia, and was managed successfully with mechanical ventilator support for a few days. At a three-month follow-up, the patient was doing very well. This paper explores the pathogenesia, implications, prevention and treatment of the transfusion-associated complications such as acidosis, hypothermia, electrolyte abnormalities, and transfusion-related acute lung injury (TRALI). Particular attention is given to the prevention of secondary coagulopathy of the patient requiring massive blood transfusion. This case study presents a good reference for similar anesthetic scenario in the future.

1. Case presentation

A 44-year-old normotensive, nondiabetic woman with a retroperitoneal mass, which was identified by Computed Tomography (CT) scan of abdomen, during her health screening two

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weeks ago, was admitted to our hospital for resection of the retroperitoneal mass.

She had a medical history of schizophrenia over 23 years, for which she was still taking an antipsychotic drug, Clozapine, 3 tablets daily, and no other past medical history of illness.

Pre-anesthetic evaluation did not reveal any abnormal findings. She had a body weight of 71 kg, height of 159 cm, heart rate (HR) of 70 beats/minute (bpm), and blood pressure (BP) of 130/80 mmHg. Her preoperative laboratory tests showed hemoglobin (Hb) of 109 g/L, hematocrit (Hct) of 31%, platelet (Plt) count of 195×10^9/L, prothrombin time (PT) of 10.9 s, international normalized ratio (INR) of 0.89 and activated partial thromboplastin time (aPTT) of 20.9 s.

On arrival in our operating room, she had BP of 140/80 mmHg with HR of 88 bpm. An 18-gauge peripheral intravenous (IV) line was placed on her right forearm and kept open with lactated Ringer's solution, and standard monitoring, including non-invasive blood pressure (BP), 3-lead ECG and Pulse oximetry (SpO_2), was given.

Surgical resection of a retroperitoneal mass was performed under general anesthesia combined with epidural anesthesia. After induction of general anesthesia, a radial arterial line was placed for continuous monitoring of arterial blood pressure (ABP) and collection of arterial blood for blood gas analysis during operation, and a central venous catheter was placed into the right internal jugular vein.

Surgery revealed that the retroperitoneal mass, located between abdominal aorta and left renal hilum, adhered to left renal hilum. In the course of the mass being separated from the left renal hilum, the patient developed significant bleeding. At the time when blood loss amounted to 500 ml, expedited infusion of lactated Ringer's solution and 6% hydroxyethyl starch were made with simultaneous applying for 3 units PRBCs from blood bank. As result of the sustained severe hemorrhage in the surgical site, the patient required massive transfusion of blood products, so as to meet the need of maintaining her circulating blood volume. But, the needed blood products were unable to be timely issued and delivered, the patient was becoming hemodynamically instable over time. When the amount of blood loss within half an hour reached to 3000 ml, her ABP decreased abruptly to 52/30 mmHg with HR of 42 bpm. She was emergently given 10 μg of epinephrine via the right internal jugular vein, followed by a continuous infusion of epinephrine for additional hemodynamic support, prompt adjustment of the dosage of epinephrine to try to maintain a mean arterial blood pressure (MAP) of 65 mmHg or above. Meanwhile, some rescue actions were taken, including the following:

- Requesting the surgeon to stop operation immediately and exerting direct pressure on the area of bleeding.
- Expedited infusion of fluids and transfusion of blood products (PRBCs and FFP).
- Established an additional wide bore venous access via left internal jugular venous.
- Pre-warming of resuscitation fluids and blood products, and using of patient warming device to prevent the patient from hypothermia.
- Emergency arterial blood gas analysis (ABG) demonstrating a PH of 7.28, ionized calcium of 0.97 mmol/L, hemoglobin of 77 g/L.
- Made decision to use intraoperative autologous blood salvage (the patient's tumor properties were not clear, the intraoperative salvaged blood possibly contained malignant cells. But in life-saving situation with the consent of the surgeon, it could be utilized to support the patient's vital signs).

After the treatments, the patient's MAP rose temporarily up to $\geqslant 65$ mmHg. As such, the surgeons could continue to perform the surgery, and look for the active bleeding spot. It was found that the source of bleeding was the lacerated left renal vein and there was unfortunately no way to repair. So, the left nephrectomy had to be performed, with the consent of the patient's family. And then, the retroperitoneal mass was removed successfully, after which the bleeding gradually reduced and eventually stopped. During this period, the operation had to be stopped several times, due to bleeding with the patient's BP declining rapidly.

Overall, total amount of blood loss of the patient within 3 h was 10.01 L. In the course of 8 h surgery, she received total 22 units of PRBCs, 18 units of FFP, 10 units of cryoprecipitate, 3750 ml of autologous salvaged blood via a cell saver, 4000 ml of crystalloids (lactated Ringer's solution and normal saline) and 2500 ml of colloid (hydroxyethyl starch), according to her physiologic parameters & laboratory testing reports. She was also given 4 g intravenous calcium gluconate, 3 g potassium chloride and some units of insulin, to correct the electrolyte abnormalities and hyperglycemia, depending on the results of repeated ABG analysis. Additionally, 4 mg intramuscular penehyclidine hydrochloride (PHC), a new anti-cholinergic drug, was given to her, for possibly improving microcirculation, depressing stress response, and so on.

After the above treatments, she gradually was hemodynamic stable. When her hemodynamic was still maintained on stable state without any vasopressor support, 10 mg of furosemide was intravenously injected. Thus, she had 2600 ml of urine output during the course of surgery. Postoperatively, she was shifted to the intensive care unit (ICU) with mechanical ventilator support, where she received another 5.4 units of FFP and 10 units of cryoprecipitate, as well as 10 g albumin to correct the hypoproteinemia, so as to increase her osmotic pressure and alleviate her systemic edema.

In ICU, guided by repeated monitoring of ABG and haematological tests, she was extubated on the third day after operation. She was shifted out of ICU to general ward, continued with supportive care. Her last report in ICU showed Hb of 97 g/L, Hct of 27%, Plt counts of 60×10^9, and PT/INR, APTT within normal limits.

Throughout the operative period and ICU stay, the laboratory examinations (see Table 1) and ABG analysis (see Table 2) were regularly monitored.

The patient had been recovering quite well. No infection of incisional wound was observed. After another 12 days in the ward she was discharged. The pathological examination of the mass revealed a spindle cell tumor, which was a benign tumor. At the three-month follow-up, she was doing all well.

2. Discussion and literature review

Hemorrhagic shock is a severe clinical syndrome, produced by rapid and massive blood loss, which may lead to hemodynamic

Table 1 Laboratory parameters.

Date/time	Hb (g/L)	Plt (10^9/L)	PT/INR (s)	aPTT (s)	Fib (g/L)	D-dimer	Creat (mmol/L)	Alb (g/L)
Preop	109	195	10.9/0.89	20.7	2.69	74	51	38.3
D1@Intraop 16:13	95	Nil	19.3/1.57	64.8	0.84	3735	Nil	Nil
21:22@ ICU	143	42	16.3/1.33	32.8	1.18	3086	61	18.3
D2@ ICU 8:40	122	50	13.1/1.07	24	2.49	673	102	25.9
D2 19:10	99	58	14.7/1.2	27.9	2.61	656	Nil	Nil
D3@ ICU 8:22	100	51	Nil	Nil	Nil	Nil	122	35.3
D4@ ICU 8:25	95	61	12.7/1.03	24.3	4.38	1547	Nil	Nil
D5@Ward	104	114	Nil	Nil	Nil	Nil	92	37.4
D8@Ward	112	202	Nil	Nil	Nil	Nil	78	35.7

Hb = hemoglobin; Plt = platelet; PT = prothrombin time; aPTT = activated partial thromboplastin time; INR = international normalized ratio; Fib = fibrinogen; Creat = creatinine; Alb = albumin.

instability, decreasing in oxygen delivery and tissue perfusion, cellular hypoxia, organ damage, and even death. One of life-saving treatments of hemorrhagic shock is massive transfusion (MT). MT is commonly defined as the transfusion of at least 10 units PRBCs within 24 h to an individual patient or the transfusion of more than one blood volume in 24 h (note that adult blood volume is approximate 70 ml/kg) [1–3]. However, it is a non-physiologic state and can be associated with many complications, such as acidosis, hypothermia, coagulopathy, electrolyte abnormalities, citrate toxicity, and transfusion-related acute lung injury (TRALI).

2.1. Complications related to massive transfusion

2.1.1. Acidosis

In patients with hemorrhagic shock, acidosis often results from tissue hypo-perfusion and hypothermia. And the acidosis is worsened by massive transfusion of acidic stored blood products, because the pH of PRBCs decreases from 7.4 to 6.9 almost immediately after storing and even falls to as low as 6.7 after 3 weeks [4].

Furthermore, acidosis may impair almost all essential parts of the coagulation process [5,6]. Platelets will change their structure and shape at pH below 7.4 [6,7]. Clotting factors are enzymes whose activity will reduce 50–90% with a decrease in pH from 7.4 to 7.0 [8–10]. Thus, thrombin generation, which is the primary engine of hemostasis, is profoundly inhibited by acidosis. Moreover, acidosis leads to increase in the degradation of fibrinogen [5,8], which further worsens the coagulopathy. Thus, in case of severe hemorrhage, buffering between physiologic pH values is recommended as the arterial pH > 7.2.

The citrate, which existed in stored blood products, will be metabolized to bicarbonate. Therefore, the correction of acidosis with sodium bicarbonate in patients requiring MT, should be used as a temporary measure, only in patients with severe metabolic acidosis and hemodynamic instability. In our case study, when the ABG analysis revealed the pH value of 7.17, 125 ml of sodium bicarbonate was infused. If needed again, should depend on the results of ABG analysis. However, once adequate tissue perfusion is restored, the citrate and lactate are converted to bicarbonate in the liver, in addition to our patient showed a good renal function with adequate urine output, and was maintained on mechanical ventilator support. So, the acidosis resolved easily (see Table 2).

2.1.2. Hypothermia

Hypothermia, defined as a situation with core body temperature < 35 °C [11], occurs frequently in patients requiring MT. There are multiple factors in trauma patients that contribute to hypothermia, including infusion of cold fluids and blood products, opening of body cavities, decreased heat production, and impaired thermoregulatory control. Infusion of cold fluids and stored blood products is a well-known cause of hypothermia. Blood products are normally stored between 1 °C to 6 °C, and thus rapid transfusion of large amount of such blood products will lead to hypothermia easily.

Hypothermia can reduce clotting factor activity. There is a 10% drop in clotting factor activity for each 1 °C drop in temperature and there is a more than 50% reduction in normal factor activity at temperature below 33 °C [7,11,12]. It can also alter platelet function (significant platelet dysfunction is observed below 34 °C [9,12]), inhibits activity of enzyme and causes induction of fibrinolysis [8,10,12]. But these effects are reversible with normalization of body temperature.

Therefore, it is important to prevent hypothermia and reduce the risk of hypothermia-induced coagulopathy. Actually, hypothermia in patients requiring MT can be avoided by the following:

- Removing wet clothing.
- Covering the patient to avoid additional heat loss.
- Increasing the room temperature.
- Surface warming the patient with heating blankets and heating lamps.
- Using of heated and humidified inspired gases for ventilators.
- Using blood and fluid warmers for all fluids administered.

Although simple, these interventions may be lifesaving, and should be implemented as early as possible in any patient with hemorrhagic shock. In our case, patient was prevented from hypothermia by pre-warming of all administered fluids and cold blood products, surface warming via patient warming devices, and increasing the operating room temperature. Because the monitor of body temperature is not yet widely available in our operating room, we monitored her body temperature only by frequently touching and feeling her limbs to ensure them keeping warm up.

Table 2 Arterial blood gas analysis in clinical course.

Date	Time	PH	PaO_2	$PaCO_2$	BE	Lac.	Glu.	Ca^{2+}	K^+	Hb	Major clinical event
D1	Intraop: 12:34	7.28	92	48	−4.1	0.9	10	0.97	3.6	77	Bloodloss 3000 ml, PRBC 8U, low-dose epinephrine
	13:14	7.23	138	44	−92	1.5	13.9	0.88	3.8	81	Use intraop blood salvage, PRBCs 4U, FFP 800 ml
	14:31	7.3	306	45	−4.3	4.1	16.9	0.85	2.8	81	Identified the lacerated left renal vein as the source of bleeding
	15:13	7.2	105	49	−8.8	5.7	19.4	0.71	3.0	98	Left nephrectomy and complete resection of the mass, PRBCs 6U,FFP 500 ml
	16:01	7.17	294	44	−12.5	6.8	22	0.74	2.8	95	Urine output 200 ml, stopping infusion epinephrine
	17:30	7.11	219	46	−14.9	9	20	0.68	2.9	95	Frusemide10 mg, PRBCs 4U, FFP 500 ml, Cryo 10U
	ICU: 21:22	7.43	93	23	−9	7.2	10.9	0.85	3.4	14	Urine output 2600 ml (intraop), FFP 540 ml, Cryo 10U
D2	ICU: 0:20	7.33	124	33	−8.5	6.6	8.3	0.91	3.9	13.3	Spontaneous breathing and ventilator support PEEP 8 cm H_2O FiO_2 40%
	6:43	7.51	76	29	−0.1	3.4	7.4	0.9	4.3	12.2	Infusion Alb 10 g, anti-infection
	13:04	7.46	91	33	−0.3	1.2	7.5	0.96	3.2	11.5	T38.3 °C
	18:35	7.44	88	34	1.02	1.6	8.2	1.02	3.3	10.4	
D3	ICU: 1:59	7.45	95	35	1.03	0.8	7.7	0.98	3.2	10.4	Spontaneous breathing and ventilator support FiO_2 40%
	6:09	7.49	105	32	0.3	0.8	7.9	1.02	3.4	10.4	T38.7 °C, anti-infection
	12:19	7.45	111	35	0.3	0.6	6.2	1.03	3.2	101	
	17:42	7.46	107	35	1.1	0.5	6.2	1.03	3.2	101	
	23:52	7.47	86	33	0.3	0.5	5.7	0.99	3.2	97	On room air
D4	ICU: 6:52	7.47	111	32	−0.4	0.5	5.6	0.99	3.2	97	T 38 °C, anti-infection, FiO_2
	12:09	7.48	111	35	2.6	0.5	5.7	1.03	2.9	11.2	Post extubation, back to ward

BE = base excess; Lac = lactic acid; Glu = glucose; FiO_2 = fraction of inspired oxygen; PEEP = positive end-expiratory pressure; Cryo = cryoprecipitate; FFP = fresh frozen plasma; PRBCs = packet red blood cells.

2.1.3. Coagulopathy

Coagulopathy related to massive transfusion is multifactorial [13], including the dilution of clotting factors, consumption of clotting factors, and hyperfibrinolysis [12,14]. Furthermore, as mentioned above, the acidosis, hypothermia and coagulopathy were known to be the lethal triad [10], which worsen hemorrhage and transfusion requirements for patients with hemorrhagic shock.

We usually began resuscitation with crystalloids and colloids, such as normal saline, lactated Ringer's solution and hydroxyethyl starch (HES). Although it may restore part of the circulating volume, the induced dilutional anemia and coagulopathy are described [9,15]. In addition to its use activating inflammatory cascades, leads to cellular swelling, acidosis, and organ dysfunction [16], which may be associated with increased mortality [17]. Thus, large-volume resuscitation with crystalloids should be avoided. However, the clinical effect of hydroxyethyl starch (HES) on coagulation remains unclear [15,18]. A study demonstrated that HES administration may promote a dose-dependent coagulopathy [19]. Therefore, if colloids are administered for resuscitation, the dosage should be within the prescribed limits.

Furthermore, massive transfusion of blood products such as RBC, FFP and PLT may also cause dilutional coagulopathy since these blood products are stored in anticoagulation solutions, leading to a decrease in concentrations of clotting factors to approximately 60% [18].

So, FFP and platelets should be given earlier to patients requiring massive transfusion. However, the optimal ratio of PRBCs, FFP and platelets is not known. Based on a massive transfusion protocol (MTP), the optimal ratio is recommended to be likely close to 1:1:1 [20,21]. Although, each unit of FFP contains 0.5 g of fibrin, hemorrhage requiring MT may still lead to consumptive and dilutional hypofibrinogenemia [22,23]. Coagulopathy worsens when serum fibrinogen level falls below 100 mg/dl. In our case, during massive transfusion, the patient's fibrinogen level in plasma was monitored and documented with 84 mg/dl, thereafter 10 units of pooled cryoprecipitate was transfused to correct the hypofibrinogenemia.

Despite enhanced understanding of the mechanisms underlying coagulopathy associated with MT, early recognition of coagulopathy remains an important clinical problem. Routine laboratory tests, such as PT/INR and aPTT have several limitations in clinical emergent situation. They are included that: (1) the time taken from blood sampling to test result is usually 45 min, (2) the coagulation tests are determined from plasma not whole blood, (3) no information regarding platelet function is provided, (4) the time for clot formation provides no information regarding clot strength of subsequent fibrinolysis. Therefore, these limitations weaken the practicality and possibly the clinical relevance of PT/INR and aPTT in the clinical emergent situation.

However, point of care monitoring devices that evaluate the viscoelastic properties of whole blood, like thromboelastography (TEG), may overcome the above limitations of routine laboratory coagulation tests in the perioperative setting. It allows whole blood to be analyzed at bedside, thus faster turnaround times. Furthermore, it may be used to quantify

fibrinogen levels, provide useful information on platelet function [9,24]. Recently, a study had showed that 82% of trauma patients requiring MT received insufficient amounts of FFP and Platelets when managed by "blind" transfusion practices [14,25]. Moreover, it has been shown that hemostatic resuscitation guided by TEG in operating room may reduce transfusion requirement and blood loss [26]. Unfortunately, it is not yet available in our operating room.

2.1.4. Electrolyte abnormality

(1) Hypocalcemia

Stored blood products are anti-coagulated with citrate, which binds ionized calcium. Each unit of PRBCs contains about 3 g of citrate. Although the healthy adult liver can clear this dose of citrate in approximately 5 min [9,12], transfusion rate higher than 1 unit every 5 min or impaired hepatic function may lead to hypocalcemia [12]. Calcium is necessary for clotting activity, myocardial contraction, and vasomotor tone. The level of arterial blood ionized calcium below 0.8–0.9 mmoL/L leads to coagulopathy and impaired cardiovascular function [6,12]. Therefore, it is critically important to frequently monitor ionized calcium concentration and keep it within the normal range, during massive transfusion. Intravenous calcium administration is the appropriate treatment of ionized hypocalcemia. In our case, patient received total 4 g of intravenous calcium gluconate, guided by a serial monitoring of the ionized calcium concentrations.

(2) Hyperkalemia and hypokalemia

Hyperkalemia may occur in patients requiring massive transfusion, due to the high extracellular potassium concentration in stored PRBCs [12]. Additionally, it also may be as result of oliguria (due to hypovolemia) and acidosis (results from tissue hypoperfusion and massive infusion of the acidic stored PRBCS). However, it did not occur in our patient.

In contrast, hypokalemia was presented in our patient. Its mechanisms are as followed:

- Dilutional hypokalemia due to massive infusion of the potassium-poor solution, such as crystalloid, colloid, FFP and platelets.
- In patients with shock, plasma concentration of epinephrine increases 50-fold and norepinephrine level increases 10-fold [27]. Moreover, the circulating catecholamine leads to movement of potassium from extracellular fluid into cells, thus lower serum potassium.
- Continuous infusion of exogenous epinephrine leads to the potassium in plasma shifting into cells.
- Restoration of the transfused red blood cell membrane ATPase pump, thus allowing potassium to re-enter the red cells.
- Alkalosis secondary to citrate metabolism to bicarbonate.

Therefore, the serum potassium concentration of patients requiring MT should be carefully monitored. A number of studies have demonstrated that hypokalemia has been seen in more than 50% of surgical patients with massive transfusion [12]. In our case, although 3 g of potassium chloride was given, hypokalemia had not been occured until the infusion of exogenous epinephrine discontinued. Hence, continuous infusion of exogenous epinephrine was thought to be one of the important causes of the hypokalemia.

2.1.5. Transfusion-related acute lung injury (TRALI)

TRALI is a serious blood transfusion complication characterized by the acute onset of respiratory distress, hypoxemia, and fever. It is defined as acute lung injury that occurs within 6 h after transfusion [12]. Although transfusion is an independent risk factor for the development of TRALI, the incidence varies with type of blood product. In particular, it is a greater risk after transfusion of FFP or platelet concentrates [27], likely due to the presence of leukocyte antibodies in transfused plasma. TRALI is a leading cause of major morbidity and mortality related to MT [12,28]. Although it is difficult for clinicians to directly prevent TRALI, clinicians need to be alerted that the adverse effect is dose related. Therefore, blood products must be transfused carefully and appropriately, and excessive transfusion should be avoided. In our case, the patient was given intraoperative cell salvage to minimize the need for allogeneic transfusion, thus reducing risks associated with allogenic blood transfusion. However, our patient developed features of early ARDS or TRALI (such as the sign of hypoxemia documented by ABG analysis, mild fever and so on) after transfusion, and was managed with mechanical ventilator support for 3 days in ICU.

2.2. Other aspects of management

2.2.1. Hyperglycemia

Hyperglycemia is especially common in patients with acute hemorrhagic shock. During early shock, endogenous catecholamine levels are significantly elevated to maintain end-organ perfusion in response to hemorrhage [29,30]. However, circulating catecholamine inhibits insulin release and activity, leading to increased plasma level of glucose. Furthermore, glucagon is also secreted in response to stress, such as hypovolemia, hypoglycemia. Thus, it has been termed *stress-induced hyperglycemia* (SIH) and defined as a transient elevation of plasma glucose level due to the stress of illness. SIH may be associated with some of the same complications as hyperglycemia in diabetes mellitus, such as poor wound healing and a higher infection rate, and higher mortality in trauma patients [31]. However, strict glycemic control and intensive insulin therapy could improve the survival rate in critically ill patients [31,32]. In our case, the patient developed a sustained hyperglycemia in the course of resuscitation. Although, when ABG analysis discovered the level of blood glucose >11.1 mmol/L, continuous infusion of some units of insulin started, the blood glucose level still transiently reached to 22 mmol/L. However, it was observed that the blood glucose level gradually decreased and returned to near normal levels, following the reduction and stopping in dosage of exogenous epinephrine infusion. Therefore, our finding suggested that exogenous epinephrine, needed for additional hemodynamic support in the course of resuscitation, is an important contributor to SIH. Epinephrine via a beta-adrenergic- receptor mechanism, causes excessive plasma glucagon elevation. Thus, the continuous infusion of epinephrine for hemodynamic support may be not appropriate for the patients with hemorrhagic shock. It may be beneficial on continuous infusion of an

alpha-adrenergic-receptor agonist, such as norepinephrine for hemodynamic support.

2.2.2. Pharmacologic therapy

Anti-cholinergic drugs have been reported in the treatment of septic shock, and found to have protective effects against septic shock. In animal experiment, anti-cholinergics may reduce bio-membrane lipid peroxide, decrease the release of cytokines and oxyradicals, stabilize bio-membrane, protect the cell structure, and thus maintain their normal functions [33].

However, traditional anti-cholinergics would increase HR, and thus resulting in the limitation of their application in the treatment of shock. Penehyclidine hydrochloride (PHC) is a new anti-cholinergic drug, which has the characteristics of selectively blocking M-cholinergic receptor subtype M_1 and M_3, with little or no effect on receptor M_2, and thus does not accelerate HR. It has been reported that PHC could significantly improve the microcirculation, shorten the time of shock resuscitation, and possibly reduce mortality in mice with septic shock [33]. More studies in animal models [33,34] showed that PHC could also attenuate renal, cerebral, gastrointestinal ischemia–reperfusion (I/R) injury, by reducing inflammatory response, oxidative stress and apoptosis.

Moreover, in recent years, several studies have demonstrated that PHC can be used in the treatment and prevention the development of ALI [35]. For the patients with ALI/ARDS, there will be appearing increase of cholinergic receptors density, hypertonia of airway and vascular smooth muscles, airway hyper-responsiveness [36,37], whereas PHC can lift airway and vascular smooth muscle spasm, inhibit respiratory gland secretion and improve cellular oxygenation at an early stage of ALI [36]. So, PHC is expected to be a potential choice for ALI management. In our case, patient was empirically given intramuscular injection of 4 mg PHC for improvement of microcirculation and prevention the development of ALI. But, at present, mechanical ventilation support is still the most effective clinical treatment of ALI/ARDS. Our patient was also supported with mechanical ventilation for several days in ICU.

2.2.3. Utilizing intraoperative blood salvage

The surgical procedure involving resection of a retroperitoneal mass for our patient should be considered for implementation of acute normovolemic hemodilution and cell salvage. But the former was due to the patient's starting hemoglobin of 10.9 g/L, later was because of the pathologic type of the retroperitoneal mass being unclear at that time. Although there was a possibility of the reinfusion of cancer cells from the surgical site, as in the unexpected life-threatening hemorrhage, the intraoperative blood salvage needed to be used following a risk–benefit analysis. In fact, it has been shown that a high percentage of patients undergoing cancer surgery have circulating tumor cells, but only 0.01% to 0.000001% of circulating tumor cells have the potential to form metastasis [38,39]. Two recently controlled studies [40] on patients undergoing radical prostatectomy were performed to evaluate the use of cell salvage, and demonstrated that massive metastasis did not occur due to the use of cell salvage. Despite this understanding, the pathologic type of the retroperitoneal mass of our patient was a benign tumor.

3. Conclusions

Acute surgical hemorrhage is a medical emergency with a high mortality if it leads to acute hypovolemic shock during surgery, hence requiring prompt and effective treatment. Besides, acute hemorrhagic shock may lead to coagulopathy, due to consumption and dilution of clotting factors, which may be worsened by hypothermia and acidosis. Previous sections have demonstrated some key points in the management of these situations, including control of bleeding, blood transfusion and supportive treatments for the maintenance of adequate tissue perfusion and oxygenation, the correction of hypothermia, anemia, acidosis and electrolyte abnormalities, and the use of appropriate blood products including FFP, platelet concentrate and cryoprecipitate to correct coagulopathy. Therefore, in patients with critical bleeding requiring massive transfusion, repeated monitoring of ABG, frequent assessment of coagulation parameters including platelets count, fibrinogen level, PT and aPTT, and continuous monitoring of temperature, are strongly required.

Furthermore, allogenic blood products are immunomodulatory, and may result in multi-organ dysfunction. Although, use of an MTP (massive transfusion protocol) with predetermined ratios of blood products (PRBCs, FFP, and platelets) may improve patient's outcomes and reduce allogenic blood components use, its implementation requires close collaboration among multidisciplinary departments, including the blood bank, the laboratory, the operating room and ICU, to ensure the timely delivery of blood products, incorporate serial monitoring of laboratory parameters, and prompt correction of patient's physiologic disturbances. In our case study, the patient required massive transfusion but we didn't follow any MTPs strictly, yet the patient recovered well. This may be due to that the facts we tried our best with the near standard blood transfusion protocol, in addition, prompt and effective administration of cell salvage reduced the need for allogeneic red cell transfusion, minimizing the transfusing-associated complications.

Conflict of interest

None.

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