We describe the treatment of two patients with hemorrhagic shock unresponsive to volume replacement and catecholamines. Both patients responded to a small-dose infusion of vasopressin, which allowed tapering off of the catecholamines. The possible role of small-dose infusions of vasopressin in fluid- and catecholamine-resistant hemorrhagic shock is discussed.

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Case 2

A 53-yr-old patient with stomach carcinoma underwent a distal gastrectomy, gastrojejunostomy, pancreatojejunostomy, and sleeve resection of the neck of the pancreas. His immediate postoperative period was uneventful. On the seventh postoperative day he developed secondary hemorrhage and had 2 L of hematemesis. He had a cardiac arrest while being endotracheally intubated. He was defibrillated, resuscitated with IV colloids, and rushed to the operating room within 30 min. There, a distal pancreateicoduodenectomy to control multiple bleeding vessels was performed and a feeding jejunostomy was inserted. Intraoperatively the patient had severe hypotension and asystolic cardiac arrest after 15 min of laparotomy. He was resuscitated with internal cardiac massage and crystalloid, blood and fresh-frozen plasma transfusions and epinephrine boluses. Bleeding was controlled 2 h after surgery started.

The patient’s total blood loss was estimated to be approximately 6 L. His CVP was 10 cm after cumulative transfusion of 13 L of crystalloids and 11 L colloids, including 5 L of blood and 1 L of fresh-frozen plasma. He continued to be severely hypotensive and dobutamine at 10 μg·kg⁻¹·min⁻¹ and norepinephrine at 0.5 μg·kg⁻¹·min⁻¹ were added. He remained hypotensive at an arterial blood pressure of 80/50 mm Hg despite these measures.

Five hours after surgery started, vasopressin was started at a dose of 0.04 U/min. Within half an hour the patient’s arterial blood pressure increased to 110/70 mm Hg. A blood gas analysis performed at this time revealed severe metabolic acidosis (pH, 7.1; base deficit, −11.4). Ventilation was continued and sodium bicarbonate was administered to correct the base deficit.

The patient was moved to the ICU. His mean arterial blood pressure remained more than 65 mm Hg. His base deficit was corrected over the next 24 h. Vasopressin was tapered off over 24 h and norepinephrine over 48 h. The patient remained hemodynamically stable and was tracheally extubated on the third day. After this, however, he developed an anastomotic leak and intraabdominal sepsis. He was defibrillated, resuscitated with IV colloids, and rushed to the operating room within 30 min. There, a distal pancreaticoduodenectomy was performed and a feeding jejunostomy was inserted. After this, however, he continued to be severely hypotensive while being endotracheally intubated. He was defibrillated, resuscitated with IV colloids, and rushed to the operating room within 30 min. There, a distal pancreateicoduodenectomy to control multiple bleeding vessels was performed and a feeding jejunostomy was inserted. Intraoperatively the patient had severe hypotension and asystolic cardiac arrest after 15 min of laparotomy. He was resuscitated with internal cardiac massage and crystalloid, blood and fresh-frozen plasma transfusions and epinephrine boluses. Bleeding was controlled 2 h after surgery started.

The patient’s total blood loss was estimated to be approximately 6 L. His CVP was 10 cm after cumulative transfusion of 13 L of crystalloids and 11 L colloids, including 5 L of blood and 1 L of fresh-frozen plasma. He continued to be severely hypotensive and dobutamine at 10 μg·kg⁻¹·min⁻¹ and norepinephrine at 0.5 μg·kg⁻¹·min⁻¹ were added. He remained hypotensive at an arterial blood pressure of 80/50 mm Hg despite these measures.

Discussion

Hypovolemic shock of marked severity and duration may progress to cardiovascular collapse that is unresponsive to volume replacement and catecholamine infusion (1). Vasopressin deficiency may contribute to the pathogenesis of irreversible shock (2). The two patients described had severe bleeding with prolonged and severe hypotension that could not be reversed despite volume replacement and catecholamine pressors.

Vasopressin has been found to be of use in the irreversible phase of hemorrhagic shock in animal studies (3,4) and in isolated case reports (5). Under normal conditions, the doses of vasopressin used have little or no pressor action (3,6), and significant increase of plasma vasopressin as a result of unregulated release of the hormone (i.e., in the syndrome of inappropriate secretion of antidiuretic hormone) does not cause hypertension (4,7). Vasopressin may be effective in resistant hemorrhagic shock resulting from inhibition of K_{ATP} channels and inhibition of nitric oxide-induced accumulation of cGMP. Replacement of depleted stores of vasopressin in the neurohypophysis may also contribute to reversal of shock (8).

Fluid-resistant hemorrhagic shock is not common, and adequate volume resuscitation is nearly always effective in resuscitation of hypovolemic patients. However, hemorrhagic shock, when advanced, can become poorly responsive to both volume and catecholamine pressors because of resistant vasodilatation and acidosis. Vasopressin may be a useful adjunct in the treatment of such cases.

References