Intra-arterial Vasopressin Injection for the Treatment of Massive Bleeding From the External Carotid Artery After Craniofacial Trauma

—Technical Note—

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Abstract

Vasopressin (0.8–1.0 IU), diluted with saline (10 IU vasopressin per 100 ml saline), was selectively injected into the external carotid artery (ECA) to control massive hemorrhage caused by inaccessible serious craniofacial injuries in two patients. This method produced remarkable angiographic vasoconstriction at the involved ECA branches and resulted in immediate hemostasis. Intra-arterial vasopressin injection is a useful option for the treatment of intractable traumatic hemorrhage from inaccessible ECA branches.

Key words: vasopressin, external carotid artery, craniofacial trauma, vasoconstrictor

Introduction

Craniofacial injury sometimes causes serious bleeding. In particular, hemorrhage due to skull base fracture and/or damage to the proximal segments of the main external carotid artery (ECA) branches might be fatal and should be controlled as quickly as possible. However, the complex anatomy hinders direct access to the damaged sites of the ECA branches and/or skull base. Occlusion of the artery proximal to the bleeding points by surgical ligation or selective embolization can stop bleeding from the skull base and/or main ECA branches.11,12) However, the surgical ligation method is rather time consuming because of the damaged neck and may be ineffective in skull base and extensive nasopharyngeal injuries because of well-developed anastomoses between the bilateral ECAs. Embolization is another option to control bleeding from the skull base and maxillofacial region, but requires embolization of multiple sites of the ECA branches.3,6,9] Embolization using conventional materials may result in ischemic tissue necrosis due to permanent occlusion of the multiple sites of the ECA branches in the injured region.4,7] Therefore, a reversible vasoconstrictive material is the ideal embolization material for hemostasis of bleeding from the ECA branches.

Vasopressin is the most essential regulator of body fluid osmolality and has a potent vasopressor effect by acting directly on the smooth muscle of the vessels.8] Vasopressin also promotes the release of coagulation factors from the vascular endothelium and increases platelet aggregation, so may be involved in hemostasis.9] Gastrointestinal hemorrhage has been successfully treated by selective intra-arterial vasopressin injection.1,5]

Here we report the use of intra-arterial vasopressin injection into the ECA to treat inaccessible massive hemorrhage caused by maxillofacial or skull base injury.

Materials and Methods

Vasopressin (0.8–1.0 IU), diluted with saline (10 IU vasopressin per 100 ml saline), was injected into the ECA at approximately 3 ml/min by selective cannulation with the Seldinger method or direct surgical cannulation. The concentration of vasopressin was determined by the Bende and Flisberg method.2] The Seldinger method was used in a patient who could correctly respond to our verbal questions. The direct surgical method was selected in a patient with
disturbed consciousness. Carotid angiography was performed through the cannulations to detect the bleeding sites and abnormal connections between the ECA and intracranial arteries before vasopressin injection. Angiography was repeated after vasopressin injection to evaluate the vasoconstrictive and hemostatic effects on the involved ECA. Neurological findings, systemic arterial blood pressure and urine volume were strictly checked during these procedures.

Informed consent concerning the vasopressin injection method was obtained from the patient and/or family before the treatment.

Case 1: A 22-year-old woman was struck on the face in a traffic accident. She was immediately transferred to our hospital. On admission she was alert and had no neurological deficit. However, she had massive nasal bleeding which made breathing difficult. Her respiration rate was 30 times/min, blood pressure was 110/70 mmHg, heart rate was 105 beats/min, and the bilateral carotid pulses were palpable. Her face showed severe swelling with lacerated wounds extending from the left lip to cheek. Subcutaneous hematoma was observed in her neck. Intraoral examination revealed a lacerating wound of the hard palate, hematoma in the left pharynx, and many broken teeth. She was intubated to improve the respiratory problems.

Computed tomography (CT) showed no intracranial lesion without serious maxillary bone fractures (Fig. 1). Debridement and closure of the lacerated wounds, and fixation of the fractured lower jaw bone were performed, but the arterial bleeding from the nasal cavity could not be controlled. Angiography was performed using the Seldinger method to identify the bleeding site. Left carotid angiography showed extravasation of the contrast material from the left maxillary artery (Fig. 2A). The catheter was selectively introduced into the left ECA. Intra-arterial injection of vasopressin (1.0 IU) drastically decreased the bleeding from the nasal and oral cavities. Repeated right carotid angiography showed persistent extravasation of contrast material from the right ECA (Fig. 2C). Vasopressin was injected into the right ECA through the selectively introduced catheter. Carotid angiography then showed severe narrowing of the ECA and no extravasation of the contrast material (Fig. 2B, D).

Vasopressin injections into the bilateral ECAs immediately succeeded in complete hemostasis. Direct verbal contact and neurological testing detected no cerebral ischemic insults such as contralateral sensory motor disturbance, visual disturbance, or speech disturbance. Her vital signs and urine volume remained normal. Her blood pressure was slightly elevated from 115/70 mmHg to
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Case 2: A 60-year-old woman fell from a chair and hit her occipital region. She had undergone liver transplantation for primary biliary liver cirrhosis, followed by immunosuppressive treatment. She was alert for 30 minutes after the head injury. CT revealed right occipital epidural hematoma and occipital bone fracture. She fell into a coma with anisocoria and right pupil dilatation at 40 minutes after the head injury. Repeat CT demonstrated significantly increased epidural hematoma extending to the supra- and infra-tentorium (Fig. 3A, B). Laboratory examinations found aspartate transaminase 73 IU/l, alanine transaminase 87 IU/l, gamma glutamyl transpeptidase 170 IU/l, total bilirubin 2.4 mg/dl, prothrombin time 20.8 sec, activated partial thromboplastin time 48.3 sec, thrombo test 37.9%, and fibrinogen 120 mg/dl.

Surgery was immediately performed to remove the hematoma and to achieve complete hemostasis. No massive bleeding sites were identified but blood was oozing from the skull base. This bleeding was intraoperatively controlled by packing the epidural space with dural tenting. Postoperatively she showed improvement of motor disturbance, but did not respond to verbal orders. Postoperative CT showed complete removal of the hematoma (Fig. 3C). However, epidural drainage showed progressive increase in blood loss amounting to 900 ml at 12 hours after the operation. Her family rejected a second open surgery, so vasopressin injection into the ECA was performed. The right ECA was exposed, and ligated at the distal site of the carotid bifurcation under local anesthesia. The ECA was cannulated with a 4 Fr polyethylene tube. Vasopressin (0.8 IU) was injected into the ECA, which resulted in complete hemostasis within a few minutes with prominent narrowing of the branches of ECA (Fig. 3D). The cannulation was maintained for 36 hours, but rebleeding did not occur. No complication caused by vasopressin injection was observed. Her condition gradually improved, and she could follow simple orders within 4 hours.

Discussion

The present cases suggest that vasopressin injection into the involved ECA is effective to stop serious bleeding from the branches of ECA due to craniofacial trauma.

Both the Seldinger method and direct surgical cannulation were used for intra-arterial vasopressin injection. The procedure was selected depending on consciousness level of the patient. If the Seldinger method is used for injecting vasopressin into the ECA, vasopressin might accidentally flow into the internal carotid artery, resulting in the risk of constriction of the cerebral vessels and subsequent cerebral ischemia. Therefore, the Seldinger method was selected only if the patient could correctly respond to orders. The direct cannulation method was selected if the patient could not respond to simple orders. The proximal site of ECA was ligated to introduce vasopressin only into the ECA territory with the direct cannulation method.

Excessive bleeding caused by epistaxis or posttonsillectomy has been successfully treated by continuous infusion of vasopressin in physiological saline for 1–3 days. Excessive bleeding caused by epistaxis or posttonsillectomy has been successfully treated by continuous infusion of vasopressin in physiological saline for 1–3 days.
idly, within 15 minutes, in eight patients, without serious side effects. However, some side effects such as abdominal pain, antidiuretic effect, and pleural effusion were observed. Vasopressin also produces simultaneous decreases in the coronary blood flow, which suggests that this treatment is contraindicated in patients with serious angina pectoris and recent myocardial infarction.

The arterial systems show different responses to different humoral and neural stimuli. Different angiographic changes occurred in the gastrointestinal and hepatic arterial systems after administration of vasopressin. Although the ECA showed excellent vasoconstriction caused by vasopressin, the response was transient. Therefore, additional mechanisms may be involved in the drastic hemostatic reactions observed subsequent to intraarterial vasopressin injection in the present patients with head and neck injuries. Vasopressin induces a marked rise in autologous factor VIII, which is hemostatically effective in adequate plasma concentrations in patients with moderate and mild hemophilia and von Willebrand’s disease. However, intra-arterial vasopressin injection provides a useful option for the treatment of intractable hemorrhage in patients with emergent head and neck injury.

References


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Commentary on this paper may appear in the next issue.