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Endovascular Treatment of Intractable Oronasal Bleeding Associated with Severe Craniofacial Injury

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Background: **Severe craniofacial injury may cause intractable oronasal bleeding, which is refractory to conventional treatments. This study will evaluate the efficacy of endovascular treatment for such oronasal bleeding.**

Methods: **Nine males between the ages of 19 and 62 years who had intractable oronasal bleeding resulting from severe craniofacial injuries received treatments of transarterial embolization using Gelfoam pledgets, polyvinyl alcohol particles, or platinum coils. We then reviewed their clinical and neuroradiologic characteristics retrospectively.**

Results: **In all but one case, angiography demonstrated bleeding points as extravasation. These bleeding points were multiple in seven cases. Except for bleeding from ethmoidal arteries, selective embolization was successful. In all cases, intractable oronasal bleeding was controlled. Patient survival was not directly related to oronasal bleeding, but rather was strongly correlated with associated brain injuries.**

Conclusion: **Endovascular treatment is an acceptable treatment for intractable oronasal bleeding associated with severe craniofacial injuries when conventional treatments have failed.**

Key Words: **Angiography, Craniofacial injury, Embolization, Epistaxis, Oronasal bleeding.**

Severe facial injury associated with complex maxillomandibular fractures may cause intractable oronasal bleeding, which is refractory to conventional treatments including nasal packing, cautery, and reduction and fixation of the fractures.[1–4] This type of severe facial injury is often associated with intracranial bleeding and brain contusions as well as life-threatening injuries elsewhere in the body. Traumatic intractable oronasal bleeding is thus a distinct clinical entity from spontaneous intractable nasal bleeding, and its treatment is extremely difficult.[2,4,5]

Since the first report by Sokoloff et al. in 1974,[6] there have been many reports of endovascular treatment for spontaneous intractable nasal bleeding,[7–19] but few reports have dealt with massive, traumatic oronasal bleeding.[2,4,20,21] With the advent of microcatheter techniques in the late 1980s, interventional neuroradiology has advanced greatly. Here we describe nine patients with traumatic intractable oronasal bleeding who were treated with embolization and discuss the usefulness of this procedure and its clinical application.

MATERIALS AND METHODS

Case summaries of the nine patients with traumatic intractable oronasal bleeding are presented in Table 1. "Intractable" means that it is impossible to achieve hemostasis using conventional methods such as nasal packing, cautery, and reduction and fixation of fractures. In all patients, bleeding was from the nasal cavity as well as the oral cavity. The patients were all males between the ages of 19 and 62 years, with a mean age of 26.9 years. Four patients had trauma resulting from falls, three from motor vehicle crashes, and two from blows received from falling objects. In seven cases, the patients' Glasgow Coma Scale scores were <7 on admission and could not be evaluated at embolization because all patients were sedated and underwent either tracheostomy or endotracheal intubation.

Four patients were in hypotensive preshock states because, in spite of blood transfusions, hemorrhaging led to loss of blood. All nine patients were treated at first with tight nasal packing, with tight oral packing when necessary. Cautery, reduction, and fixation of fractures were carried out as needed. Surgical ligation of the external carotid artery or the internal maxillary artery was not performed in any patient. The patients underwent embolization because (a) a computed tomographic (CT) scan did not disclose intracranial mass lesions that required surgical removal, (b) conventional methods failed, (c) bleeding was massive, (d) bleeding points seemed to be multiple, (e) it was impossible to define the bleeding side, or (f) some patients were at high risk for surgery under general anesthesia. In our hospital, neurosurgeons carry out diagnostic or interventional neuroangiography. At least one neurosurgeon is always in the hospital and can start diagnostic angiography within 1 hour upon request. Neurosurgeons who are responsible for embolization can also join the procedure within 1 hour. At least one critical care physician always supports the patient's general condition in the angiography suite in the treatment of intractable oronasal bleeding.

In the nine cases, we first used a 5F diagnostic Headhunter catheter to perform conventional transfemoral cerebral angiography. Then, superselective angiography was carried out using a 3F microcatheter (Tracker-18 or FasTracker-18, Target Therapeutics, Inc., Fremont, Calif) coaxially through the diagnostic catheter to disclose bleeding points. We used Gelfoam pledgets (Pharmacia & Upjohn, Inc., London, England), polyvinyl alcohol particles (150–600 μm), or platinum mi-

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TABLE 1. Case summary

Case No.	Age/Sex	GCS Score on Admission	Brain Lesion	Other Lesions	Blood Infused (mL)	Bleeding Vessel	Side	Embolization Time (minutes)	Time to Embolization	Outcome
1	22/male	4	Epidural hematoma	Lung, femur fx, tibia fx	3,520	SPA	Right	90	9 days, 16 hours	Good recovery
2	20/male	6	SAH	Finger fx	10,480	SPA, IOA, FA	Bilateral	180	17 hours	Good recovery
3	20/male	4	Subdural hematoma	Lung	3,440	SPA	Left	75	6 hours	Good recovery
4	19/male	6	SAH	Lung, pelvic fx, femur fx, tibia fx	12,520	SPA, LPA, ADTA, IAA, FA, LA	Bilateral	120	12 hours	Moderate disability
5	20/male	3	SAH, epidural hematoma	No	13,840	SPA, FA	Bilateral	143	80 minutes	Vegetative state
6	62/male	7	Anoxic brain	Pelvic fx, femur fx, Humerus fx	10,080	SPA, AAA	Bilateral	120	3 hours	Vegetative state
7	20/male	3	Cerebellar hematoma	Radius fx	12,640	STA, FA	Right	75	2.5 hours	Death
8	36/male	14	Subdural hematoma	No	11,960	GPA, TFA, AMA, EA	Bilateral	170	6 hours	Death
9	23/male	12	SAH, brain swelling	No	1,200	SPA, SPAA	Bilateral	40	5.5 hours	Death

AAA, anterior auricular artery; ADTA, anterior deep temporal artery; EA, ethomoidal artery; FA, facial artery; fx, fractures; GCS, Glasgow coma scale; GPA, greater palatine artery; IAA, inferior alveolar artery; IOA, infraorbital artery; LA, lingual artery; LPA, lesser palatine artery; SAH, subarachnoid hemorrhage; SPA, sphenopalatine artery; SPAA, superior posterior alveolar artery; STA, superficial temporal artery.

crocoils (0.018-inch in diameter with a variety of lengths and shapes) through the microcatheter to carry out embolization. Gelfoam pledgets was used only in case 1. Time from the trauma to commencement of diagnostic angiography was within 3 hours in three patients, 6 hours in three patients, 17 hours in two patients, and 9 days in one patient. In case 1, delayed oronasal bleeding occurred 9 days after surgery for epidural hematoma, and 16 hours passed between oronasal bleeding and embolization. In all cases, aggressive blood transfusion was required during embolization. Oronasal packing was usually removed several days after embolization.

RESULTS

All of the patients suffered from extensive maxillary fractures, and except for three patients, they all suffered from mandibular fractures (Figs. 1*A* and 2*A*). In the patient in case 1, angiography did not demonstrate extravasation but did show abnormal stasis of the contrast material in the sphenopalatine artery. In the other eight patients, conventional or superselective angiography showed extravasation of the contrast material from at least one artery. There were no dangerous anastomoses between the branches of the external carotid arteries and the internal carotid artery or the ophthalmic artery in any of the patients. Bleeding from the sphenopalatine artery was observed in seven patients (Figs.1*B* and 2*B*). The other bleeding arteries are listed in Table 1. We noted unilateral bleeding in three patients and bilateral bleeding in six patients. In all but two patients, the bleeding points were multiple. By embolization, hemostasis was obtained in all vessels except for the ethmoidal arteries. In all patients, we were able to control intractable oronasal bleeding.

Because of marked hypovolemia and insufficient blood transfusion, the patient in case 4 developed bradytachyarrhythmia during embolization, which became sinus tachycardia after successful embolization. In four patients, tracheostomy was performed before embolization, whereas embolization was carried out under endotracheal intubation in four patients. In case 6, when we were transferring the patient from the emergency room to the angiography suite, we had to perform resuscitation and an emergency tracheostomy when

FIG 1. Case 4, a 19-year-old man. (*A*) CT scan of the face shows multiple fractures of the maxilla (*arrows*) and right temporal bone (*arrowheads*). (*B*) Right external carotid injection (anterior-posterior view) shows extravasation of the contrast material from the lesser palatine artery (*long arrow*), the septal branch of the sphenopalatine artery (*arrowhead*), the anterior deep temporal artery (*short arrow*), and the inferior alveolar artery (*open arrow*).

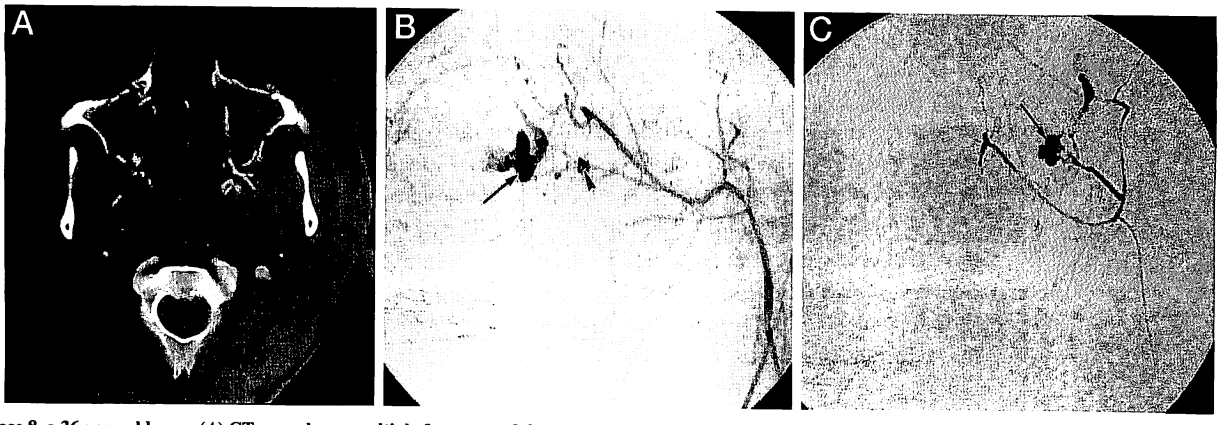

FIG 2. Case 8, a 36-year-old man. (*A*) CT scan shows multiple fractures of the maxillary bone (*arrowheads*). (*B*) Right external carotid injection (lateral view) demonstrates extravasation from the sphenopalatine artery (*arrow*) and the transverse facial artery (*arrowhead*). (*C*) Left proximal internal maxillary artery injection (lateral view) shows extravasation from the left accessory meningeal artery (*arrow*).

he suffered cardiopulmonary arrest because of clots in the endotracheal tube that blocked his airway. Although resuscitation was successful and was followed by endovascular treatment, follow-up computed tomography revealed global anoxic brain, and the patient remained in a persistent vegetative state. Other than this patient, however, no patient developed any serious systemic or neurologic complication during angiography and embolization. In case 2, the patient's blood group was Rh negative, and because it is difficult to prepare a lot of Rh negative blood, embolization was performed. Two patients underwent surgical removal of acute intracranial hematomas, which were revealed by postembolization CT scan, immediately after embolization. One patient (case 4) underwent embolization in the territory of the bilateral internal iliac arteries for retroperitoneal bleeding after embolization of oronasal bleeding. Most surviving patients required maxillofacial surgery or orthopedic surgery during their chronic stage.

The time required for diagnostic angiography and embolization of oronasal bleeding ranged from 40 to 180 minutes, with a mean time of 112 minutes. The amount of blood required for oronasal bleeding and associated injuries within several days after ictus ranged from 1,200 to 13,840 mL, with a mean quantity of 8,853 mL. It was difficult to estimate the amount of blood loss from facial injuries in each patient because of the associated trauma elsewhere in the body. Because blood loss was mostly from oronasal bleeding in five patients, however, a mean quantity of blood loss from the facial injury could be estimated at 10,024 mL. Even though blood pressure during embolization was low, major bleeding points were occluded, and blood pressure soon became normal in three patients.

Overall, the outcome was a good recovery in three patients, moderate disability in one patient, vegetative state in two patients, and death in three patients. The three patients died because of irreversible brain damage 7 to 20 days after ictus, with a mean of 13 days. The follow-up periods for surviving patients ranged from 2 to 19 months, with a mean of 8.2 months. During this period, no recurrent bleeding was observed in any patient. Patient survival was strongly related to the associated brain injuries and was not directly related to oronasal bleeding.

DISCUSSION

Nasal bleeding is classified into two types according to the bleeding sites: anterior and posterior. Anterior nasal bleeding, which occurs at Kiesselbach's plexus, is usually less severe, self-limited, and easily managed with conventional methods. Posterior nasal bleeding often occurs in an inaccessible region and is difficult to control with nasal packing or cautery.[1,8,22,23] The cause of both anterior and posterior nasal bleeding is usually spontaneous. Traumatic intractable oronasal bleeding differs from spontaneous nasal bleeding in its clinical presentation. The difference is the location and pathologic nature of the bleeding points, the multiplicity and severity of bleeding, the difficulty in controlling the bleeding, the associated injuries, and the patient's general condition.

Computed tomography is the primary diagnostic tool for severe craniofacial trauma because it discloses intracranial abnormalities such as intracranial hemorrhages, brain contusions, edema, pneumocephalus, and foreign bodies. CT examination is indispensable before and after embolization to detect such abnormalities. Although CT scans show maxillomandibular fractures in detail, they do not define exact oronasal bleeding points. Selective angiography, however, is useful for delineating bleeding points,[4,24] and it sometimes demonstrates multiple bleeding points. Contrary to some previous reports,[14,16] the presumption of the laterality and the location of oronasal bleeding points is often misleading, especially in traumatic bleeding.[4] For this reason, bilateral internal and external carotid angiography must be performed.[4,6,17,21,24,25] When nasal packing is effective, however, extravasation is not always demonstrated.[4,6,8,21,24–26] In cases of severe facial injuries, there are usually multiple bleeding points other than those demonstrated as extravasation of the contrast material when the packing is effective.

Tight nasal and oral packing should be performed first in any patient with oronasal bleeding. Such packing, however, was ineffective in our series. Some possible explanations for the failure of the packing are: (a) bleeding is not only from the nasal cavity but from the oral cavity; (b) an extensive maxillomandibular fracture caused a large communication between the nasal and oral cavities; or (c) in patients with comminuted facial fractures, there are no solid walls for the

packing to buttress against, merely soft tissue. Given the flexibility of soft tissue, it is difficult to pack the areas of hemorrhage tight enough to stop the bleeding.

In addition to conventional methods, ligation of the external carotid artery is an alternative treatment,[3,10,27] but this is not always effective because of abundant collateral circulation in the external carotid territory.[8,28] The ligation of the internal maxillary artery through a transantral approach is also an alternative,[1–3,19,22,23,27,29] but it is sometimes ineffective,[2,8,10,12] and is difficult, especially when the patient is critically ill with active bleeding. Surgical ligation of the external carotid artery or the internal maxillary artery is indicated when interventional neuroradiologists are not available. It should be noted, however, that unilateral ligation may be ineffective and that the internal maxillary artery is not always the causative vessel, as shown in this study. If interventional neuroradiologists are available, embolization has distinct advantages over surgical ligation: distal access near bleeding points, demonstration of bleeding points, control of multiple bleeding points, repeatability, no necessity for general anesthesia, and short procedure time.[7,10,11,13,14,18,19,21,30] Although endovascular treatment is not free of risks,[11,12,15,17–19,31,32] when an experienced neuroradiologist performs the treatment, the risks of inadvertent migration or reflux of the embolic materials, blindness, facial nerve palsy, and soft-tissue necrosis are slight. Furthermore, embolization should be started promptly when conventional treatments have failed to control oronasal bleeding because continuing to administer such treatments may threaten the patient's life and require large amounts of blood. In fact, based on our experience with delayed intervention in our earlier series, we have shifted to prompt endovascular treatment to control oronasal bleeding. In four recent cases, we decided to perform embolization in the early stage of the patient's admission and did not strictly observe the conventional methods.

Although embolization was effective at controlling intractable oronasal bleeding in all of the patients in this series, embolization had to be performed within a limited time in most patients because of their poor general condition. Although complete angiographic hemostasis of all bleeding points is ideal, endovascular occlusion of the more proximal vessels in addition to incomplete distal embolization is often effective at reducing pressure at the bleeding points.[30] During intervention, associated injuries should be taken into account because these influence the patient's chance of survival. They include intracranial hemorrhage, brain contusion, thoracic trauma, abdominal trauma, renal trauma, and orthopedic injuries. Both oronasal bleeding and bleeding from other parts of the body may require massive blood transfusions. Delaying blood transfusions may result in irreversible hypovolemic shock. Furthermore, when there are multiple fractures in the body, the use of muscle relaxants for general anesthesia may cause additional blood loss. Immediately after embolization, subsequent surgical intervention or embolization may be necessary for other conditions, such as intracranial hemorrhages or lesions in the abdominal or pelvic areas. This occurred in three patients in this series. Theoretically, oronasal packing can be removed immediately after successful embolization.[15,17] Although this may be true for spontaneous nasal bleeding, in cases of severe craniofacial injuries it should be delayed for several days because there are multiple bleeding points. Hemostasis is obtained by a pressure balance between the lacerated vessels (arteries and veins) and the packing, and progressive local hemostasis by thrombus formation is expected for several days.

The cause of the bleeding is vessel laceration or transection by trauma, which is different from spontaneous bleeding. If a bleeding point is far from the tip of the microcatheter, embolization using polyvinyl alcohol particles is first carried out. Then, coil occlusion of the more proximal vessel is carried out. In the case of spontaneous nasal bleeding, the proximal portion of the internal maxillary artery should not be occluded, so that embolization can be repeated in the future. For traumatic bleeding, however, it is unnecessary to preserve this proximal vessel, and occlusion of the artery with platinum microcoils is feasible. Although there are many types of platinum microcoils, we think that Tornado-type coils (spiral-shaped fibered coils with increasing helical diameter; Cook Inc., Bloomington, Ind) are the most useful for this purpose and that they reduce the time required to obtain complete occlusion of the vessel compared with other types of coils.

In a hypovolemic state or under the administration of vasoconstriction drugs such as dopamine or noradrenaline, the external carotid territory may show extensive vasospasm. This may give the false impression of vessel laceration when segmental vasospasms are observed. Embolization to these vasospastic vessels may cause reflux of the embolic materials to the internal carotid artery, resulting in the serious complication of cerebral infarction. A pressure injection of the particulate embolic materials should be avoided. Furthermore, embolization under real-time fluoroscopic control, as in the road-map technique, is especially important under these circumstances.

In the case of bleeding from ethmoidal arteries, it is theoretically possible to embolize through ophthalmic arteries using microcatheters.[33] This is not always feasible, however, and there is a high probability that it will cause blindness because of inadvertent embolization to retinal and posterior ciliary arteries. Surgical clipping of ethmoidal arteries is one alternative.[1,8,10,16,18,27,28] If the bleeding is minor, conservative treatment is another option.

It is vital to keep the airway open by endotracheal intubation or tracheostomy during the interventional procedure.[2] In the case of severe facial injury, it should be noted that a clot that may cause obstruction of the airway later may already be in the trachea. Reintubation, for any reason, is extremely difficult because of intraoral blood and massive edema of the soft tissue. In this situation, a tracheostomy is preferable to endotracheal intubation.

In conclusion, embolization is an acceptable treatment for traumatic intractable oronasal bleeding when conventional treatments have failed. Early recognition and treatment of the associated injuries may improve the patient's chance of survival, and close cooperation between neurointerventionists,

critical care physicians, otolaryngologists, and maxillofacial surgeons is imperative.

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