Ischaemic necrosis of the tongue secondary to cardiogenic shock: a case series.
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INTRODUCTION
Ischaemic tongue necrosis is rare, with fewer than 15 reported cases in the world literature, all related to giant cell (temporal) arteritis. We previously reported the first case of tongue gangrene as a sequel of profound cardiogenic shock, and now present our updated experience. Our institution’s cardiac surgery service has accrued a total of 4 patients with ischemic necrosis of the tongue as an end-organ manifestation of profound cardiogenic shock.

METHODS
We present the case histories and photographs of four patients (2005-2008) in which profound cardiogenic shock eventuated in tongue necrosis (photographs were unavailable in one case).

RESULTS
Four patients in our institution’s cardiovascular surgery intensive care unit were noted to develop ischemic necrosis of the tongue. All 4 patients had experienced protracted courses of profound cardiogenic shock requiring high-dose vasopressor support and urgent cardiac surgery. Intra-aortic balloon pump (IABP) therapy was required in 3 of 4 cases. One patient developed synchronous carotid arterial occlusive disease requiring intervention. Duration of orotracheal intubation was variable. All patients had concomitant signs of poor end-organ perfusion, including lower extremity ischemia and renal and hepatic failure. Ultimately, 3 of 4 patients died, with 1 patient surviving after sloughing of the entire oral tongue.

Details of representative cases are presented.

Case 1.
A 79 year old woman with hypertension and congestive heart failure presented in severe cardiogenic shock with a frail mitral valve leaflet. Vasopressor and inotrope support were initiated, and an intra-aortic balloon pump (IABP) placed after 12 hours. The tip of the oral tongue was noted to be dusky the next day. The patient underwent emergent valve replacement, the IABP was removed, and the otolaryngology service consulted on hospital day 3. At this point, the tip of the tongue was black. The erythrocyte sedimentation rate, checked for concern of giant cell arteritis, was not elevated. There was no evidence of pressure or venous congestion from the endotracheal tube. The patient also exhibited acute renal failure and hepatic failure as multi-organ manifestations of shock. Meticulous oral hygiene was initiated and broad-spectrum antibiotics continued. The patient was already on aspirin and coumadin for cardiac purposes. On day 11, the entire oral tongue had liquefied and began to separate from the base of tongue. This necrotic tissue separated by day 18. The patient was ultimately discharged to an acute nursing facility on hospital day 30. This case has been reported by our group in a previous publication1.

Case 3.
An 88 year old woman with coronary artery disease underwent cardiac catheterization which revealed obstructive left main coronary artery disease. During the procedure, the patient developed severe hypotension and bradycardia requiring placement of an IABP and transvenous pacemaker. The following day a 2-vessel CABG was performed and the IABP removed postoperatively. The patient was extubated the next day, but on postoperative day 4, developed atrial fibrillation and a massive stroke due to occlusive carotid arterial disease, requiring urgent carotid embolectomy. The patient developed critical lower extremity ischemia 5 days later, during which time the anterior aspect of the oral tongue was noted to be black. There was no evidence of compression by the teeth or endotracheal tube. The patient ultimately died from acute cerebral edema 2 days later.

Case 4.
A 91 year old woman with coronary artery disease experienced profound cardiogenic shock 8 days after urgent CABG. An IABP was placed for 48 hours. Two days later, the otolaryngology service was consulted for a black tip of tongue. There was no evidence of compression by the teeth or endotracheal tube. The patient simultaneously developed multi-organ failure, including renal and hepatic failure. Meticulous oral hygiene was instituted, and anti-platelet therapy was continued. The patient experienced a fatal cardiac arrest the following day.

DISCUSSION
Complete gangrene of the oral tongue has only been rarely reported, mainly in the context of giant cell arteritis, where lingual arteritis has been implicated2. Of 15 reported cases of tongue gangrene, the majority have been limited to the tip of the tongue. We have previously published one case (Case #1) of total tongue necrosis, as the first reported case related to shock.

Three of these 4 patients required intra-aortic balloon pump counterpulsation therapy for profound cardiogenic shock. It is unclear whether the IABP itself, or simply the severity of shock, was causative. Nevertheless, with a mortality of 75% (3 of 4) in this series, it appears that ischemic tongue necrosis due to shock is an end-organ sign carrying a grave prognosis.

In shock, hypoperfusion places distal end organs at risk of ischemia. In these patients, profound cardiogenic shock directly led to multi-organ failure. However, the head and neck enjoys a rich vascular supply, normally protective in low flow states.

However, recent evidence suggests that the IABP does not augment carotid arterial flow3-4, because the pump operates distal to the takeoffs of the carotid arteries. As a result, perfusion of the head, neck and brain is not improved with IABP support. The external carotid circulation is particularly susceptible, because of carotid artery physiology. In low flow states, blood is preferentially shunted from the high-resistance, muscular external carotid artery to the low-resistance internal carotid artery (which is perfused even in diastole). This phenomenon acts to protect cerebral circulation in states of shock, leaving the territories of the external carotid vulnerable.

Accordingly, in these patients, the head and neck remained hypoperfused for the entire duration of IABP support. The tongue, a muscular end-organ, was particularly susceptible. In the one patient who did not receive IABP therapy, the territories of the external carotid system were nevertheless susceptible in low-flow states, as the internal carotid artery is preferentially perfused.

None of these patients had a history, signs or symptoms of giant cell arteritis. Otorrhageal intubation cannot be ruled out as a contributing factor. Because of the IABP and, thus, low lingual artery pressures, the tongue may have been more susceptible to external compression causing arterial insufficiency. No cases of tongue ischemia following endotracheal intubation alone have been reported.

IABP patients should be carefully monitored for signs of head and neck hypoperfusion. We recommend that gangrene of the tongue be managed similarly to dry gangrene of the extremities – with wound care, and debridement of necrotic tissue only for infection. Prophylactic antibiotics covering oral flora and pain control may be indicated in these cases. If the underlying cause of ischemia has been addressed, anticoagulation is unlikely to be necessary. Finally, if there is any suspicion of compression by the endotracheal tube, an early tracheotomy should be considered.

REFERENCES