Spontaneous carotid blowout of the common carotid artery in a chronically immunosuppressed transplant patient

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A 37-year-old man with a history of hypertension, type 1 diabetes mellitus, and end-stage kidney disease maintained on azathioprine and tacrolimus status-post combined kidney-pancreas transplant 1.5 years prior presented with 4 days of abdominal pain, nausea, vomiting, and diarrhea. He was admitted for cytomegalovirus (CMV) viremia (>85,000 copies/mL) and concern for acute hepatitis. He was started on ganciclovir antiviral therapy; however, on hospital day 6, despite improvement in his viremia (>42,500 copies/mL), he developed sudden left neck pain and swelling with tracheal deviation. Notably, there was no known history of neck surgery or instrumentation. A computed tomography scan demonstrated active extravasation from his left common carotid artery (CCA) (Fig 1). Vascular surgery was consulted and emergently took the patient to the operating room. This case is published with the consent of the patient.

Given the patient’s emergent status neuromonitoring was unavailable; however, with his stroke-free history and otherwise stable preoperative neurological status, it was deemed unnecessary to perform carotid shunting. A standard oblique incision anterior to the sternocleidomastoid with subsequent lateralization of the muscle was performed to expose the left CCA in the usual fashion. An expanding hematoma was immediately encountered deep to the sternocleidomastoid muscle. Vascular control of the CCA was established proximal to the hematoma and a 5-mm circular defect was identified in the proximal CCA with surrounding friable tissue approximately 2.5 cm in length. Additionally, the surrounding tissue had significant inflammatory change that tracked further medially. The defect was controlled with digital pressure and the patient was heparinized to a therapeutic activated coagulation time of 250. After circumferential dissection of the defected segment, a clamp was placed on a healthy segment of distal CCA, and dissection of the bifurcation was deemed unnecessary. Owing to the degree of inflammation, the deep neck space was further interrogated with the assistance of otolaryngology but revealed no mucosal violation of the esophagus or trachea.

Several tissue specimens from this area were sent for pathology and culture. In total, a 2.4 cm × 0.7 cm segment of damaged CCA was resected to healthy edges and replaced with a nonreversed greater saphenous vein interposition graft. The patient was reversed with protamine at the conclusion of surgery. On postoperative day 1, the patient had an acute-onset right upper extremity weakness and was found to have several small acute cortical infarcts in the left frontal and parietal lobes. Imaging of the graft confirmed patency, and his weakness resolved completely by postoperative day 3.

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ABSTRACT
Carotid blowout (CB) is a life-threatening surgical emergency with a mortality rate of up to 60%. CB is commonly seen in head and neck cancer patients after surgical and radiation therapy; other causes include iatrogenic, traumatic, or infectious etiologies. We report an unusual case of spontaneous CB presumed to be caused by cytomegalovirus (CMV) in a chronically immunosuppressed transplant recipient. Given the significant mortality of CB and the prevalence of post-transplant CMV, this case highlights an area of further investigation regarding the association between CMV and carotid pathology, as well as the need to include CB as a potential infectious complication in the immunosuppressed population. (J Vasc Surg Cases Innov Tech 2022;8:715-8.)

Keywords: Carotid blowout (CB); Carotid blowout syndrome (CBS); Cytomegalovirus (CMV); Chronic immunosuppression; Transplant

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The final pathology demonstrated focal myxoid change (Fig 2, A and B), small vessel acute vasculitis within the adjacent soft tissue, and Verhoeff-Van Gieson staining (Fig 2, C) revealed focal elastic fiber disruption. However, immunohistochemical staining for CMV (Fig 2, D), and wound cultures were negative and demonstrated no evidence of atherosclerosis, calcification, or chronic inflammation. Given his active viremia, the transplant infectious diseases team attributed his CB to CMV infection and he was treated with intravenous ganciclovir for 6 weeks followed by lifelong suppression with oral valganciclovir. The patient was discharged on postoperative day 5 and a 1-year postoperative surveillance computed tomography scan confirmed patency.

**DISCUSSION**

Herein we present a case of spontaneous carotid blowout (CB) with presumed etiology of CMV viremia. To our knowledge, this is the first reported case of CB as a complication of CMV viremia in the literature. CB is most often seen in patients with a history of radiation for head and neck cancer. In the majority of cases, that is, 80% to 90% of CB, radiation leads to adventitial fibrosis and obliteration; however, our patient had no such history. Infection is an additional predisposing factor to carotid injury and CB, but these cases are predominantly secondary to retropharyngeal abscesses from bacterial species such as actinomycosis and methicillin-resistant *Staphylococcus aureus* among others. Although viremia is common in immunocompromised individuals, there remains a paucity of literature directly linking viral infection to CB. Specifically, CMV viremia is more typically known to cause pneumonitis, colitis, esophagitis, uveitis, and nephritis, which makes its presentation in our patient quite noteworthy. A growing body of literature has linked CMV viremia with atherosclerotic plaque vulnerability, thrombosis, acute coronary syndromes, and carotid artery stenosis. However, these complications of CMV were not present in our patient, who otherwise did not have any history or imaging findings consistent with intimal disease or atherosclerosis. In contrast, Swain et al used a canine model to suggest that preservation of the adventitia is the most important factor in preventing arterial rupture from infection. Although no clear linkage could be found in our patient, our working hypothesis is that the patient’s chronic immunosuppression and acutely severe CMV viremia altered the integrity of the arterial wall and predisposed him to CB.

There are no other reported cases of spontaneous CB owing to infectious etiologies in transplant or immunosuppressed patients. Given that CMV causes active infections in at least one-third of transplant recipients and the high mortality of CB, this case highlights the need for additional investigation into the strength of the association between CMV and carotid pathology including CB.

**CONCLUSIONS**

CB is a rare and life-threatening surgical emergency. We present a unique case of spontaneous CB in a transplant patient with CMV as the presumed infectious etiology. There is no known predilection of CMV infections...
affecting the carotid vasculature. Patients with a history of chronic immunosuppression and/or chronic bacterial, fungal, or viral infection may be at increased risk for the development of unusual infectious complications such as spontaneous CB. Prompt recognition and expeditious surgical treatment is key to achieving positive clinical outcomes.

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