A 16-month-old female child presented with a single episode of epistaxis (~100 mL) and intermittent high-grade fever. The child also had a single episode of blood-stained vomitus the previous day. No history of trauma or other bleeding manifestations was present. Clinical examination revealed severe pallor, tachycardia, and hypertension (104/70 mm Hg). Local examination of the nasal cavity and pharynx showed no obvious pathology. The epistaxis was considered secondary to hypertension. Further episodes of epistaxis in the hospital were managed with anterior and posterior nasal packing. The child developed hypotension and was stabilized with intravenous fluids and packed red cell transfusion. Computed tomography angiogram (CTA) revealed a large pseudoaneurysm (24 × 14 mm) from the left cervical segment of the left internal carotid artery (ICA) with hemotoma in the left parapharyngeal space. There was no contrast opacification of distal ICA beyond the pseudoaneurysm. There were no infarcts in bilateral cerebral hemispheres. After informed consent from the parents, digital subtraction angiography (DSA) was done under sedation using left common femoral artery access and 4F diagnostic catheter. DSA of the left ICA showed a slow filling of left ICA distal to the pseudoaneurysm. Right ICA injection showed normal filling of left middle cerebral artery and anterior cerebral artery via the Circle of Willis. Coil embolization of the pseudoaneurysm neck and adjacent parent left ICA was done using coils (Nester, 4 × 14 mm). Postprocedure check angiogram revealed complete occlusion of the pseudoaneurysm neck and left ICA. The procedure was uneventful. The next day, nasal packing was removed and there were no further episodes of epistaxis. She had persistent hypertension that was initially managed by sodium nitroprusside infusion and gradually changed over to oral amlodipine. On follow-up, she had mild right hemiparesis that improved in 3 months of almost 5/5 power. Due to her hypertension, vasculitis workup was done, but it turned out to be negative.

Extracranial ICA pseudoaneurysms are rare in children. They are usually a complication of deep neck space infection but can also be caused by trauma, vasculitis, and iatrogenic procedures.1 These patients can deteriorate rapidly if there is bleeding into the nasal cavity or pharynx. The youngest previously reported case of ICA pseudoaneurysm coiling was by Roos and Butler in a 2-year-old child.2

The earlier reports of cervical internal carotid pseudoaneurysms in pediatric age group were due to infections including tonsillitis, impaction of food material (like fishbone), vasculitis, collagen vascular disorders, and iatrogenic procedures apart from trauma.3–6 Singhal et al7 have reported two cases of ICA pseudoaneurysm formation in an 8- and 2-year-old children after fine-needle aspiration of cervical lymph nodes. Jean Roger et al8 and Karim et al9 have reported pseudoaneurysm formation in the common carotid arteries following fishbone impaction. Both these cases underwent surgical repair with good outcome. Sundarrajan et al10 recently reviewed the treatment of large infectious extracranial carotid artery pseudoaneurysms in children and found 26 patients. This included 17 males (65.4%) and the median age of
The presentation was 4 years. In this review, 23 (88.5%) pseudoaneurysms were on the ICA and 2 (7.7%) were on the external carotid artery. One pseudoaneurysm was located on the common carotid artery. The most common presentation was hemorrhage that occurred in eight patients (30.8%) and the most common causative organism was Streptococcus sp. (9 cases out of 15 cases with pathogen report). Of the 18 patients treated with endovascular technique, 14 underwent coil embolization of the parent vessel, 3 underwent carotid stenting, and 1 underwent vascular plug deployment. Reported postprocedural neurological deficits include facial nerve palsy, vocal cord paralysis, Horner’s syndrome, and hemiparesis.

In their review of eight cases of pediatric neck arterial pseudoaneurysms, Balasundaram et al have suggested a management protocol based on presentation and collateral flow.

In emergency conditions with a good intracranial collateral flow, parent vessel occlusion can be done.

Previous reports also mention about watershed infarct in the ipsilateral hemisphere with some functional impairment after endovascular coil embolization of parent ICA for pseudoaneurysm, though the intracranial collateral flow was angiographically adequate. The child in our report developed mild right hemiparesis postprocedure at 1 week. If the collateral circulation is not adequate, then the parent artery may be preserved by stent-graft or surgical ligation of pseudoaneurysm and end-to-end anastomosis. Coil embolization into the pseudoaneurysm sac is not preferred as it may erode into the mucosa and bleed profusely. Other novel methods used in treatment of these pseudoaneurysms include using n-butyl cyanoacrylate (glue) in a case of spontaneous ICA pseudoaneurysm in a 3-year-old child.

Intraaneurysmal thrombin injection is usually not done in carotid pseudoaneurysms due to the chances of recurrence and distal embolization. Complications following endovascular management include coil migration, recanalization of the pseudoaneurysm, and embolic stroke. The data on the long-term follow-up and outcome is limited due to fewer number of reported cases. The etiology of the pseudoaneurysm in our case was not clear and though the child’s hypertension was considered due to vasculitis, the workup was negative.

Conflict of Interest
None declared.

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