Introduction
Patients with isolated lower cranial nerves paralysis will usually present to the otolaryngologist. Vascular aetiology high in the neck is a less common compared to tumours, trauma and infection.

Case 1
A 77 y.o. female presented with a history of paralysis of cranial nerves 9 -12 for a month. Neurological examination was otherwise unremarkable. Laboratory studies were normal. Native scanning of the brain, cranial base and neck revealed asymmetry of the parapharyngeal space. Contrast enhanced CT Revealed coiling of the right internal carotid and tortuosity on the contralateral side.

Case 2
45 y.o. female with similar paralysis that progressed and subsided in the course of two weeks after a period of excessive vomiting. Contrast enhanced CT and MRI, EMG, neurological and laboratory studies were all unremarkable. Second case made us reconsider the first one and ICAD was suspected in both cases but was not proved. Regression of symptom may confirms such an etiology in both cases.

Discussion
A dissection may be subintimal or subadventitial leading to ischaemic symptoms or aneurysmal dilatation and cranial nerve palsies. Most patients present with cerebral ischaemia and rarely with CN palsies – 5%. This may be underestimated because only 21 % of the cases with CN palsies also show cerebral ischaemia. ICAD can either occur spontaneously or following minor trauma or physical effort, such as coughing vomiting (case 2), etc. Congenital tissue defects such as fibromuscular dysplasia, Ehlers-Danlos syndrome, Marfan’s syndrome, ICA redundancy (case1), etc. are associated with an increased risk of spontaneous dissections of the carotid and vertebral arteries.

Conclusion
ICAD is a well-recognized, but not frequent cause for lower cranial nerves palsies. It must be considered in the absence of other neurological pathology or tumour. However, in both cases we failed to prove it, because of lack of strong suspicion.