

# Contralateral External Carotid Artery as Collateral to Internal Carotid Artery in a Patient with Common Carotid Artery Occlusion

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## Abstract

Common carotid artery (CCA) occlusion is sometimes associated with collateral flow through carotid bulb that preserves patency of internal carotid artery (ICA). Usually ipsilateral external carotid artery (ECA) anastomotic branches supply the ICA. But in this case, we found the contralateral ECA role in collateralization of the ipsilateral ICA to CCA occlusion using color carotid duplex.

## INTRODUCTION

Occlusion of the common carotid artery (CCA) is generally associated with occlusion of the ipsilateral internal carotid artery (ICA) and external carotid artery (ECA). Sometimes, however, collateral circulation to the ECA may preserve patency of the ICA via retrograde perfusion through the bulb (1, 2, 3). This collateral flow can be maintained through ECA anastomotic branches such as superior and inferior thyroidal arteries, deep cervical artery, descending branch of occipital artery (4), superior and inferior labial arteries (5) or an aberrant ICA branch (6).

There is no patient report of natural connection of contralateral ECA to ipsilateral ICA as collateral in CCA occlusion patient to restore ICA flow. But this anastomosis surgically can be induced in some patients with CCA occlusion (7). This external carotid to external carotid crossover anastomosis may have application in the management of squamous cell carcinoma involving the common carotid or in the treatment of carotid artery blowout (8).

These patients may suffer ongoing transient ischemic attacks and risk for stroke (1). Most authors agree to say that color flow duplex imaging has now become the hallmark to detect a patent ICA in spite of a CCA occlusion (2). Recognition of this pathologic variant may allow for effective surgical intervention (1). Such situation should not be ignored since bypass surgery can easily allow for effective restoration of

flow (2).

## CASE REPORT

A 60-year-old, white man presented with Broca aphasia and right central hemifacial weakness. He did not history of hypertension, diabetes mellitus, smoking, or transient ischemic attack. Physical examination showed only absent carotid pulsation in left side of his neck.

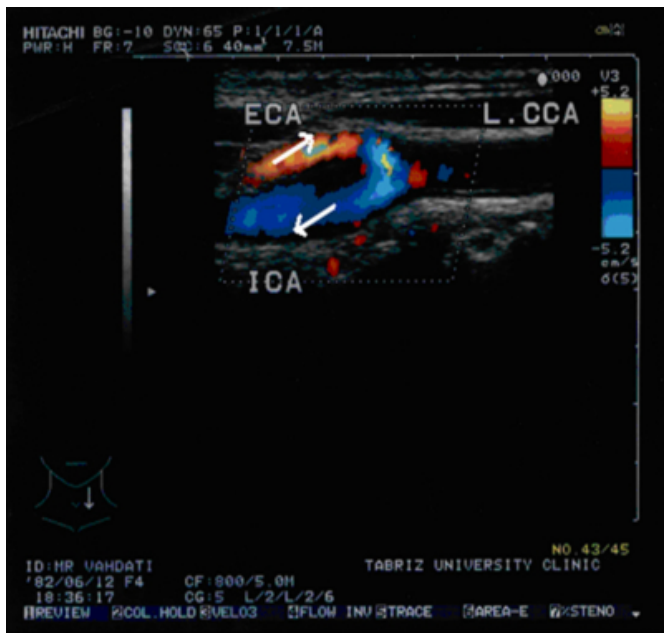
First day brain CT scan was normal. Subsequent brain CT obtained on 5<sup>th</sup> day of admission demonstrated a hypodense lesion in left paraventricular area.

Carotid duplex revealed the absence of the blood flow in left CCA. The blood flow in the left ECA was reversed and fed the left ICA (fig.1).

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**Figure 1**

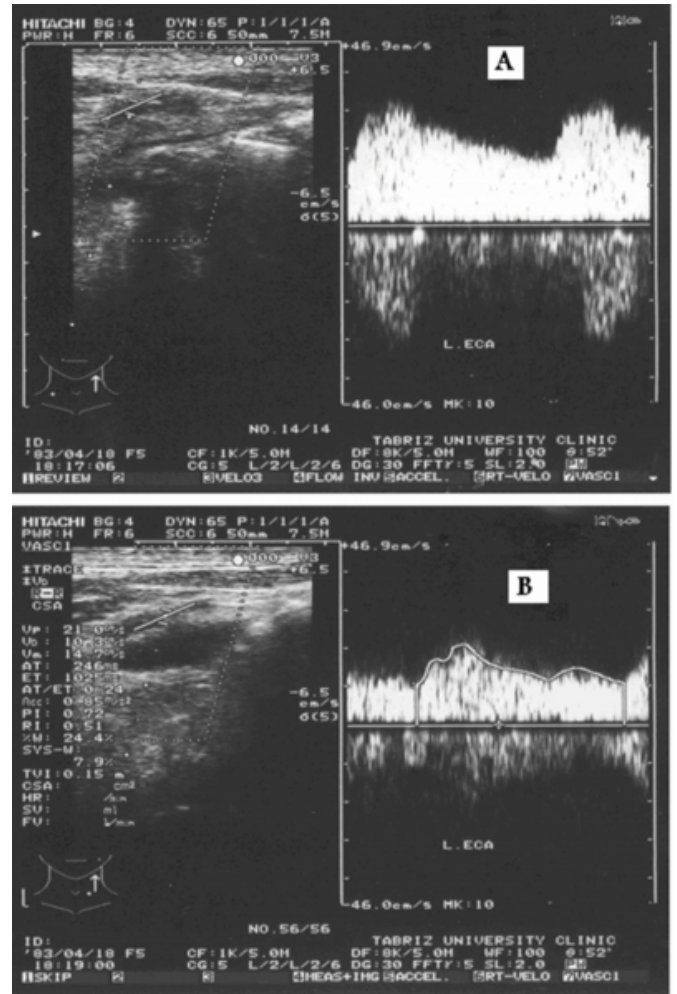
Figure 1: Retrograde flow in carotid bulb through external carotid artery (ECA).



With compression on the contralateral ECA in the mandibular angle blood flow in the left ECA was decreased (fig2 A,B).

**Figure 2**

Figure 2: Spectral wave of left external carotid artery before (A) and after (B) compression of the contralateral ECA in the mandibular angle that leads to decrease of blood flow velocity.

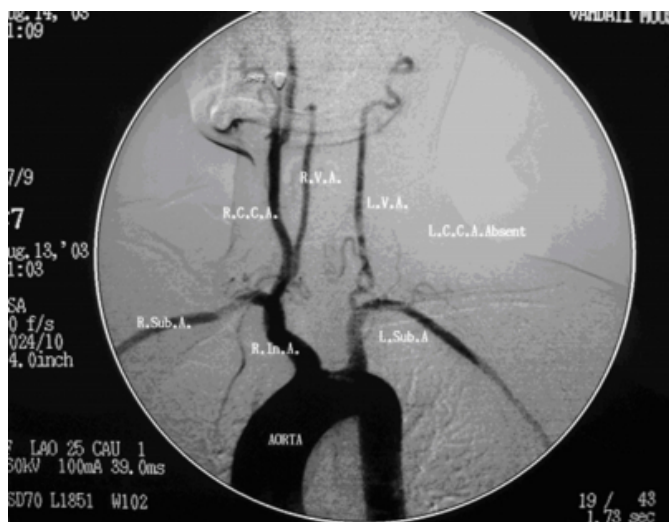


In transcranial Doppler, the left CCA flow could not be detected. Flow velocity of right anterior cerebral artery increased and direction of left anterior cerebral artery was reverse. The left middle cerebral flow velocity decreased.

In brain four vessels angiography, the left CCA could not be catheterized and the patency of ICA and ECA could not be shown (fig.3).

**Figure 3**

Figure 3: Aortic angiography: Left common carotid artery could not be catheterized.



**DISCUSSION**

In a swine model, occlusion of CCA alone was unable completely to halt ICA antegrade blood flow, while CCA and ECA occlusion completely stopped ICA antegrade flow (9). In a study for evaluation of the hemodynamic of the collateral circulation, the main collateral pathway to the ligated ECA region was the ipsilateral occipital artery through Richter's anastomosis from the vertebral artery in the case of unilateral ECA ligation, and was the contralateral carotid artery in the case of unilateral CCA, ECA, and internal carotid artery resection. The superior and inferior labial arteries were important as the collateral pathway from the contralateral ECA (3). Determination of a patent ICA in CCA occlusion is important in diagnosis and surgical treatment (1, 2, 3). Conventional angiography has some difficulties in detection of CCA occlusion and ICA patency, same as our reported case (10, 11, 12, 13). Delayed angiographic views of the bulb allowing for late collateral vessel filling can be informative (1). A data collection study including the review of angiograms and duplex scans suggests that carotid duplex is more sensitive for detecting ICA flow after common carotid artery occlusion than routine contrast angiography(6).

In recent studies Carotid duplex scanning is more sensitive in diagnosis of CCA occlusion and patent ICA (2,6, 11).

In our patient, it seems that the right ECA plays as an important collateral for the left ECA and patency of the left ICA.

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