

Association of Hyoid Bone Elongation with a Twisted Carotid Bifurcation

Grigol Keshelava

gagakeshelava@gmail.com

Clinic Healthycore

Research Article

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Abstract

Purpose

The purpose of this study is to determine the frequency of coexistence of greater horn of hyoid bone (GHHB) elongation and twisted carotid bifurcation (TCB) and to hypothesize a possible developmental mechanism underlying this association.

Methods

One part of the study is monocentric and retrospective: we selected consecutive patients over a 4-year period in whom carotid entrapment by an elongated GHHB was identified. The second part consists of a structured electronic literature search of published case reports.

Results

In total, 24 patients were included. Cases with a normal carotid bifurcation (Group 1) numbered 10 (41.7%), while those with TCB (Group 2) numbered 14 (58.3%). In all patients of Group 2 the TCB was exclusively right-sided (100%), and GHHB elongation was either right-sided or bilateral. In contrast, in Group 1 GHHB elongation occurred on the right, bilaterally, or on the left. Our findings establish a clear regularity: when TCB and GHHB elongation coexist, the TCB is invariably right-sided and the elongation is right-sided or bilateral. This pattern does not apply when GHHB elongation occurs against a background of normal carotid bifurcation.

Conclusion

We propose that the most plausible explanation is a shared developmental disturbance of the third pharyngeal arch field, which can simultaneously produce GHHB elongation and excessive lateral migration of the external carotid artery, resulting in TCB. Right-sided predominance is consistent with the normal asymmetry of aortic arch remodeling. This hypothesis remains speculative in the absence of direct embryological series, yet it coherently integrates the clinical regularity we observed.

1. Introduction

The hyoid bone (HB) is a horseshoe-shaped bone that forms attachments with several muscles and assists tongue movement and swallowing. It is located between the thyroid cartilage and the mandible in the anterior neck, usually at the level of the C3 vertebra. The HB consists of a body and two pairs of projections—the greater and lesser horns. Ligaments attach the ends of the greater horn of the hyoid bone (GHHB) to the thyroid cartilage, while the styloid process attaches to the lesser horns [8].

The HB begins to ossify at the end of the third trimester and may never fully fuse until late in life [9]. Some authors suggest that elongation of the GHHB occurs during embryonic development [18]. During this period the second and third pharyngeal arch cartilages contribute to the formation of the styloid process as well as the lesser and greater horns of the hyoid bone. The body of the hyoid develops from the hyoid anlage without significant input from the second and third pharyngeal arch cartilages [4]. A basic knowledge of HB embryology is therefore essential for understanding the etiology of anatomical variants.

Throughout development the geometry of the carotid bifurcation (CB) changes considerably. Initially the internal carotid artery (ICA) appears as a lateral offshoot, whereas the external carotid artery (ECA) extends more directly from the common carotid artery (CCA) [31]. Consequently, the topographic relationships between the carotid arteries and the HB are expected to show a degree of variability over time. Various studies have confirmed that the transverse positioning of the carotid arteries and their interconnections may differ [22, 25].

Because the HB lies close to the carotid arteries, elongation of the GHHB can produce carotid vessel compression, stenosis, dissection, or pseudoaneurysm formation [30]. Carotid entrapment by the HB remains a rare and relatively under-recognized phenomenon. Anatomical variations of the HB occur in 4–30% of the general population [6], yet a sufficiently elongated GHHB that actually compresses the carotid arteries is uncommon. Both medical and surgical treatments have been described [13, 14, 19, 23].

Our attention was drawn to the observation that patients with GHHB elongation frequently also exhibited a twisted carotid bifurcation (TCB). The same association was noted when we examined similar clinical reports in the literature. The mechanism underlying the frequent coincidence of GHHB elongation and TCB remains unknown.

TCB itself occurs in 11.3–12.4% of the general population [12, 33]. Carotid bifurcation anatomy is customarily divided into three types: Type 1 (ICA lateral, ECA medial—the normal arrangement), Type 2 (ICA and ECA overlapping), and Type 3 (ICA medial, ECA lateral) [12]. Types 2 and 3 constitute TCB.

The purpose of this study is to determine the frequency of coexistence of GHHB elongation and TCB (Types 2 and 3) and to propose a coherent developmental hypothesis that may explain this association.

2. Materials and Methods

One part of the study is a monocentric, single-arm, retrospective analysis of longitudinal data. We selected consecutive adult patients referred with neurological disorders or carotid artery disease to the Department of Vascular Surgery of our clinic over a 4-year period (2020–2024) in whom carotid entrapment by an elongated GHHB was identified. Final data analysis was performed in August 2024.

The study population consisted of adult patients with a clinically suspected diagnosis of transient ischemic attack (TIA) or stroke, plus suggestive imaging findings on ultrasonography and computed

tomography angiography (CTA), who were found to have GHHB elongation. CTA was used to measure GHHB length, to define the topographic relationship of the horns to the carotid arteries, and to determine the anatomical type of the carotid bifurcation. Treatment strategy for elongated GHHB was guided by these investigations. Clinical outcomes were followed until August 2024. The study was conducted in accordance with the Declaration of Helsinki.

The second part of the study consisted of a structured electronic literature search of published case reports performed in PubMed, Web of Science, ResearchGate, and Google Scholar. The following keywords were used: *hyoid bone elongation*, *carotid entrapment*, *carotid compression*, *twisted carotid bifurcation*, *anatomical variation of the greater horn of hyoid bone*, and *Eagle syndrome*. Radiological images presented in the retrieved publications were interpreted with particular attention to whether GHHB elongation was associated with ipsilateral or contralateral TCB.

3. Results

3.1. Retrospective Institutional Series

Because carotid vessel compression by an elongated GHHB is rare, only four patients (1 male, 3 females; mean age 69 years) met inclusion criteria during the 4-year period. In the same interval a total of 277 carotid artery reconstructions were performed in our department.

In all four cases carotid entrapment by the GHHB coexisted with atherosclerotic stenosis of the ICA. In two patients the ICA was compressed, in one the ECA, and in one the elongated GHHB did not contact the carotid vessels. Surgical intervention (carotid endarterectomy plus resection of the elongated GHHB) was performed in two patients. One patient declined surgery and received antiplatelet therapy; in the remaining case there was no indication for operation.

Two patients had bilateral GHHB elongation with unilateral carotid entrapment. In one of these the vessels were in contact with the GHHB on the left while a TCB was present on the contralateral (right) side (Fig. 1).

In the second case the carotid arteries were compressed on the right and a TCB was also present on the right [13]. In the third case GHHB elongation was right-sided and contact with the vessels occurred on the right; the bifurcation was in normal position bilaterally [14]. In the fourth patient a right elongated GHHB did not contact the carotid arteries, yet a TCB was present on the right (Fig. 2).

Thus, association of TCB with GHHB elongation was noted in three of the four institutional cases, and every TCB was right-sided (Table 1).

Table 1
Summary of the retrospective institutional series: association of TCB with elongated GHHB. TCB = twisted carotid bifurcation; BNP = bilateral normal position.

Case	Age/Sex	GHHB elongation side	GHHB contact side	TCB side / BNP
1	68/F	Bilateral	Left	TCB right
2 [13]	72/F	Bilateral	Right	TCB right
3 [14]	67/M	Right	Right	BNP
4	70/F	Right	No contact	TCB right

3.2. Results of the Systematic Literature Search

Twenty-two case reports of ICA entrapment by the GHHB were identified in the English-language literature. Two of these did not describe the anatomy of the contralateral carotid bifurcation and were therefore excluded [26, 28]. Of the remaining 20 patients described across 16 studies, six were female and 14 were male; mean age was 56 years (range 29–85).

These 20 patients were divided into two groups: those with normal carotid bifurcation (Group 1) and those with TCB (Group 2) (Fig. 3, Table 2).

In five patients ICA compression was associated with atherosclerotic stenosis. In three reports the authors noted reversible changes of ICA position provoked by swallowing or neck rotation [20, 21, 34]. These cases were assigned to Group 1.

Group 1 comprised 9 patients (45.0%) and Group 2 comprised 11 patients (55.0%). In Group 1 unilateral GHHB elongation was observed in 7 cases and bilateral elongation in 2. In Group 2 bilateral elongation was noted in 5 patients, and the TCB was right-sided in every case (100%).

Table 2

Summary of the systematic literature search: association of TCB with elongated GHHB.
TCB = twisted carotid bifurcation; BNP = bilateral normal position.

N	Author	Age/Sex	GHHB elong. side	Contact side	TCB / BNP
1	Kölbel [17]	61/F	Bilateral	Right	TCB right
2	Ishikawa[11]	74/M	Right	Right	TCB right
3	Abdelaziz [1]	85/F	Right	Right	TCB right
4	Hong [10]	56/M	Right	Right	TCB right
5	Renard [29]	83/M	Bilateral	Left	TCB right
6	Ye [35]	80/M	Right	Right	TCB right
7	Liu [20]	81/M	Right	Right	TCB right
8	Ludt [21]	32/F	Bilateral	Right	TCB right
9	Martinelli [23]	68/M	Right	Right	TCB right
10	Yalcin [34]	70/M	Bilateral	Right	TCB right
11	Plotkin [27]	32/F	Bilateral	Right	TCB right
12	De Campos [5]	29/M	Right	Right	BNP
13	Liu [19]	39/M	Left	Left	BNP
14	Yukawa [36]	36/M	Right	Right	BNP
15	Mori (2011)[24]	61/M	Left	Left	BNP
16	Schneider [30]	47/M	Left	Left	BNP
17	Kinoshita [16]	70/M	Bilateral	Right	BNP
18	Tokunaga [32]	46/F	Right	Right	BNP
19	Kho [15]	29/F	Bilateral	Left	BNP
20	Kho [15]	34/F	Right	Right	BNP

3.3. Pooled Results

In total, combining the retrospective institutional series (n = 4) and the literature series (n = 20), the study included 24 patients (9 females, 15 males; mean age 58 years, range 29–85). None had documented connective-tissue disorders.

Group 1 (Normal carotid bifurcation): 10 patients (41.7%).

Group 2 (Twisted carotid bifurcation): 14 patients (58.3%).

Bilateral GHHB elongation occurred in 2 patients of Group 1 and in 7 patients of Group 2. In every patient of Group 2, the TCB was right-sided (100%), and GHHB elongation was either right-sided or bilateral. By contrast, in Group 1, GHHB elongation occurred on the right, bilaterally, or on the left.

4. Discussion

Typically the carotid arteries lie posterolateral to the HB and do not contact it. Nevertheless, carotid–hyoid topographic relationships display measurable variability. Duan et al. studied 74 intact hyoid bones and found mean lengths of the right and left GHHB of 31.4 ± 2.6 mm and 31.0 ± 2.5 mm, respectively [7]. Manta and colleagues categorized carotid–hyoid relationships into twelve distinct types, several of which place one or both carotid arteries medial to the GHHB (combined prevalence of medial types approximately 3%) [22].

In the study of Uno et al. TCB was located on the right side in 84.6% and on the left in only 15.4% of cases [33]. No fully satisfactory scientific explanation for this right-sided predominance has yet been offered.

Our attention was drawn to the frequent coincidence of TCB and GHHB elongation (58.3% of the pooled series). Two classical hypotheses exist for the etiology of TCB itself. The congenital hypothesis proposes that TCB results from excessive mediolateral migration of the ECA during embryogenesis. The alternative hypothesis attributes elongation and tortuosity of the carotid arteries to age-related atherosclerosis [12]. Because TCB is observed even in young individuals without atherosclerosis or arterial tortuosity, we regard the congenital explanation as more convincing. Neither hypothesis, however, accounts for the striking association with GHHB elongation or for the absolute right-sided laterality of that association.

4.1. Preferred Hypothesis: Shared Developmental Field of the Third Pharyngeal Arch

The most coherent explanation, in our view, is a disturbance of the common developmental field of the third pharyngeal arch. The greater horn of the hyoid bone arises principally from the cartilage of the third pharyngeal arch. The ICA and the carotid sinus derive from the third aortic arch artery together with the cranial portion of the dorsal aorta. The ECA originates more ventrally (from the ventral pharyngeal artery / remnants of the first and second arches) and subsequently migrates toward the third-arch territory to complete the definitive bifurcation [4]. If relevant signaling pathways, neural-crest cell migration, or mesenchymal condensation within the third-arch field are perturbed, two consequences may arise simultaneously:

1. Excessive growth or incomplete remodeling of the GHHB, producing elongation.
2. Excessive lateral migration of the ECA relative to the ICA, resulting in a twisted bifurcation (ICA medial, ECA lateral).

This “common-field” disturbance elegantly accounts for the non-random association we observed and for the fact that the association is not obligatory—normal bifurcation can still coexist with elongated GHHB when the migratory component of the disturbance is milder or absent (Fig. 4).

This interpretation also accounts for the fact that twisted carotid bifurcation can occur in the absence of hyoid elongation. An isolated disturbance limited to the third aortic arch artery (or its remodelling) may produce excessive lateral positioning of the external carotid artery without affecting the cartilaginous derivatives of the third pharyngeal arch. In contrast, a broader perturbation of the third pharyngeal arch developmental field — involving neural crest and mesenchyme — is more likely to affect both the greater horn of the hyoid and the adjacent vascular structures, resulting in the co-occurrence of GHHB elongation and TCB. The right-sided predominance of this association remains consistent with the normal asymmetry of aortic-arch remodelling. Literature data already show that more than 80% of isolated TCBs are right-sided [33]. This laterality most probably reflects the normal asymmetry of aortic-arch remodeling (persistence of the left fourth aortic arch as the definitive aortic arch, formation of the brachiocephalic trunk on the right). When GHHB elongation is superimposed on this asymmetric background, the morphogenetic effect becomes clinically detectable far more readily on the right. The identical developmental disturbance on the left may more often remain subclinical or produce only subtle topographic changes that do not meet the definition of TCB (Fig. 4).

Left panel: Bilateral embryonic aortic arches (I–VI) are shown in red. Black arrows indicate their remodeling into the definitive aortic arch and carotid bifurcations.

From the third aortic arch the internal carotid artery (ICA) is formed (both sides).

From the first and second aortic arches the external carotid artery (ECA) is formed (both sides).

On the right side the carotid bifurcation is shown in the twisted configuration; on the left side the bifurcation remains in the normal position.

The right fourth aortic arch contributes to the brachiocephalic trunk; the left fourth aortic arch forms the definitive aortic arch (red curved arrow).

Right-sided predominance of twisted carotid bifurcation (TCB) is consistent with the normal asymmetry of aortic-arch remodeling.

Right panel: Pharyngeal-arch cartilages (I–VI) are shown in gray. Black arrows indicate their contribution to the formation of the hyoid bone:

Second pharyngeal arch → upper part of the hyoid body and the lesser horns;

Third pharyngeal arch → lower part of the hyoid body and the greater horns (GHHB).

The diagram illustrates the anatomical basis for the proposed common-field disturbance: a developmental perturbation within the third pharyngeal arch territory can simultaneously produce elongation of the GHHB

and excessive lateral migration of the external carotid artery.

At the molecular level, experimental studies in animal models have identified several genes that regulate the development of the third pharyngeal arch and its derivatives. In particular, *Hoxa3* is expressed in neural crest cells that populate the third arch, as well as in the associated mesenchyme and endoderm. Homozygous *Hoxa3* null mice exhibit both malformations of the greater horn of the hyoid bone and bilateral regression of the third arch artery, resulting in anomalies of the carotid arterial system [2, 3]. *Tbx1*, a critical regulator of pharyngeal apparatus development, also influences the formation and remodelling of the third and fourth pharyngeal arches and their associated aortic arch arteries; disruption of *Tbx1* signalling (as observed in models of 22q11.2 deletion syndrome) primarily affects vascular patterning but can secondarily impact surrounding mesenchymal structures [37].

These literature-based findings from experimental models support the theoretical possibility that a localised perturbation within the third pharyngeal arch developmental field could simultaneously affect both its cartilaginous derivatives (greater horn of the hyoid) and the positioning of the carotid bifurcation. It must be emphasized, however, that the present study is based solely on clinical and radiological observations in adult patients together with a review of published case reports. No genetic or molecular analyses were performed in our cohort, and the involvement of specific genes such as *Hoxa3* or *Tbx1* in the association we describe remains entirely speculative.

4.2. Mechanical Influence During Development

An additional, not mutually exclusive, mechanism is mechanical interaction between the developing GHHB (and its surrounding mesenchyme) and the migrating ECA. An already elongated greater horn may physically constrain or deflect the pathway of the ECA, favoring a more lateral final position of that vessel and thereby a medialized ICA. Such mechanical guidance is especially plausible on the right side, where aortic-arch remodeling is asymmetric because of the presence of the brachiocephalic trunk.

Japanese reports have documented that movement of the hyoid during swallowing can transiently create or abolish an apparent TCB configuration [16]. This observation indicates that mechanical interaction between the GHHB and the carotid bifurcation remains possible after birth. An elongated GHHB increases the probability of such dynamic effects, particularly during swallowing or head rotation. Nevertheless, the absolute right-sided regularity and the presence of the association in young patients argue that the fundamental predisposition is established embryonically.

4.3 Less Probable Explanations

Purely age-related atherosclerotic lengthening of the carotid arteries is an unsatisfactory explanation: the association is observed in young adults, and the laterality is far too strict to be accounted for by random atherosclerotic remodeling. Likewise, a purely chance coincidence is improbable; a 58.3% coexistence rate in a selected series of GHHB elongation greatly exceeds the background prevalence of TCB ($\approx 12\%$) [12, 33].

5. Limitations

Our study establishes a clear morphological regularity: whenever TCB and GHHB elongation coexist, the TCB is exclusively right-sided and the elongation is either right-sided or bilateral. This regularity does not hold when GHHB elongation occurs against a background of normal carotid bifurcation; in that setting the elongation may be right-sided, bilateral, or left-sided.

We propose that the most logical explanation is a disturbance of the shared developmental field of the third pharyngeal arch (neural crest and mesenchyme), capable of producing both GHHB elongation and excessive lateral migration of the ECA. Right-sided dominance is explained by the normal asymmetry of aortic-arch remodeling. Mechanical factors, both embryonic and postnatal, may further amplify the association.

It must be emphasized that this hypothesis is speculative. Direct embryological evidence—serial human embryonic series focused on this specific association, or experimental models—does not yet exist. The absence of such evidence is not fatal for a hypothesis paper; on the contrary, the identification of a reproducible clinical pattern that invites a coherent developmental interpretation is precisely the value of the present work. Future studies correlating three-dimensional embryonic imaging, molecular markers of the third-arch field, and postnatal CTA anatomy will be required to test the hypothesis rigorously.

Declarations

Ethics approval statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of Clinic Healthycore.

Participant consent statement: Informed consent was obtained from all subjects involved in the study.

Ethical approval:

Not applicable (case report).

Competing interests:

The authors declare that they have no competing interests.

Patient consent

Informed consent has been obtained from the patient for publication of the case report and accompanying images.

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Author Contribution

G. Keshelava: project development, data collection, manuscript writing and review/approval.

Availability of data and materials:

All data supporting the findings of the current study are available upon reasonable request from the corresponding author.

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- ## Figures



CTA and intraoperative findings in a patient with bilateral GHHB elongation, left-sided carotid entrapment, and right-sided TCB. (A) Axial CTA showing relationships of the carotid arteries to the GHHB. (B) Atherosclerotic stenosis of the left ICA with entrapment of left ICA, ECA and ascending pharyngeal artery by the left GHHB. (C) Surgical exposure demonstrating the anatomical relationships (RGHHB- right great horn of hyoid bone; RECA- right external carotid artery; RAPA- right ascending pharyngeal artery; RICA- right internal carotid artery; LECA- left external carotid artery; LGHHB-left great horn of hyoid bone; LICA-left internal carotid artery; LAPA- left ascending pharyngeal artery; AP- atherosclerotic plaque; RCCA- right common carotid artery; LCCA- left common carotid artery) .

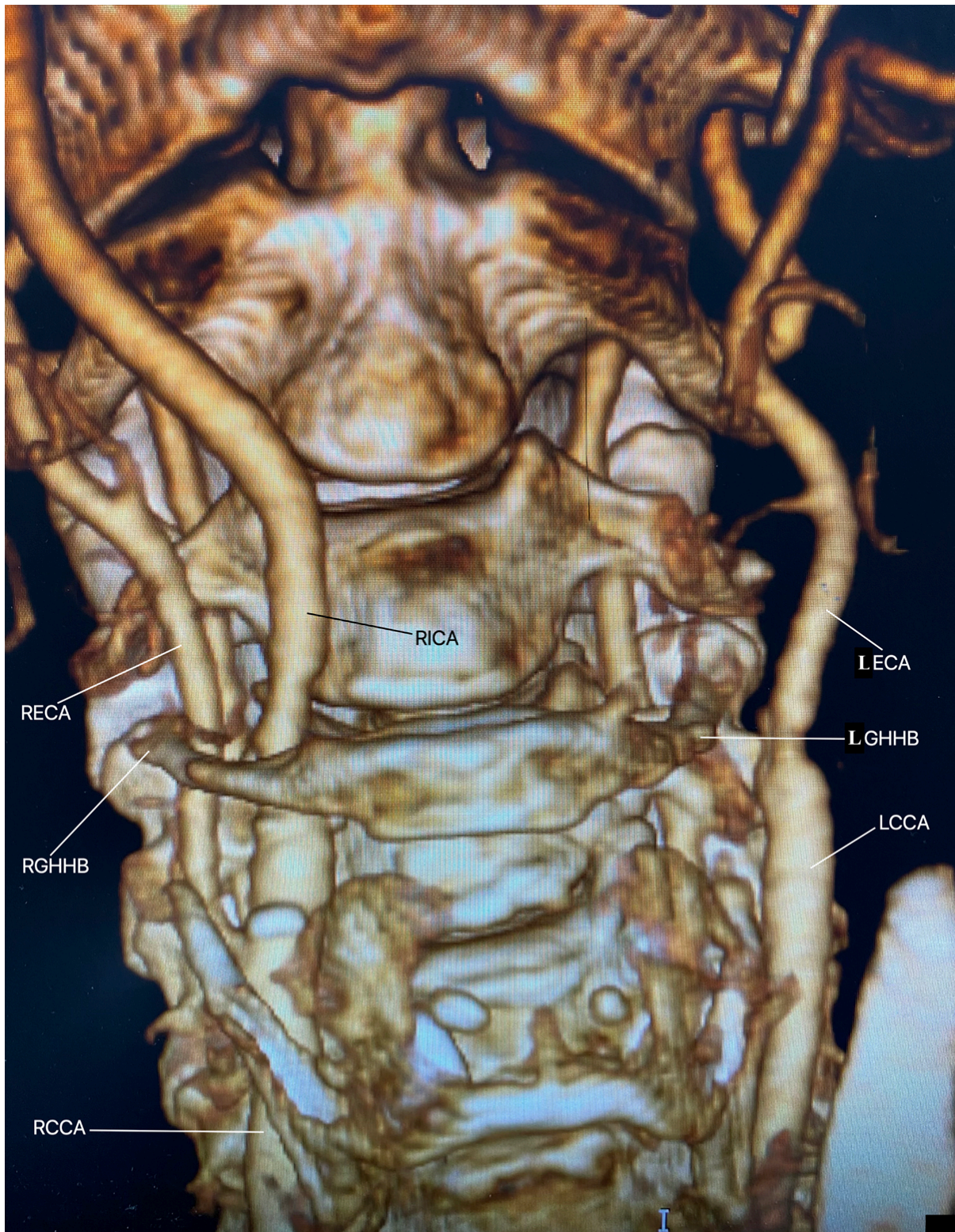


Figure 2

CTA demonstrating occlusion of the left ICA and elongation of the right RGHGB that does not contact the right carotid arteries; the right ICA is medialized (TCB) (RCCA- - right common carotid artery; RGHGB- right great horn of hyoid bone; RECA- right external carotid artery; RICA- right internal carotid artery; LECA- left external carotid artery; LGHGB- left great horn of hyoid bone; LCCA- left common carotid artery)

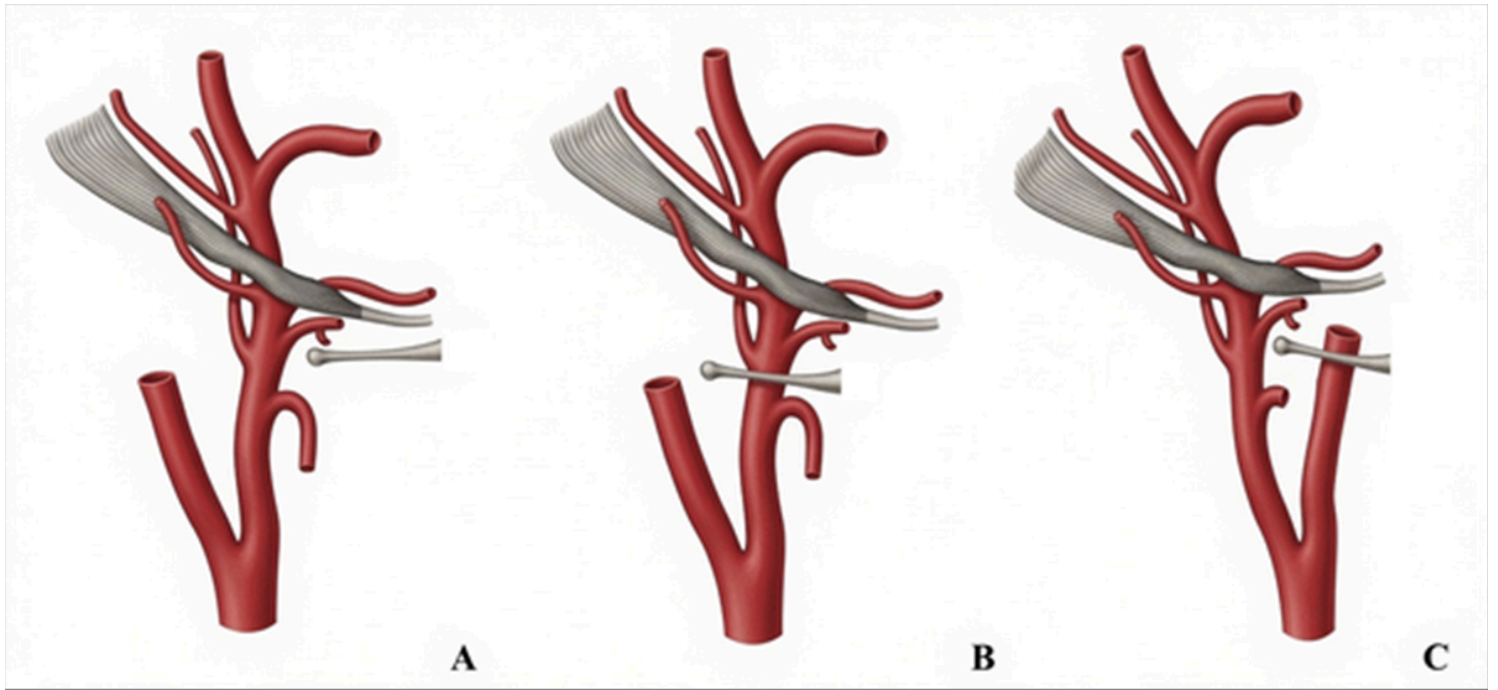


Figure 3

Schematic illustration of the relationship between carotid bifurcation and GHHB. (A) Normal position of the GHHB with normal carotid bifurcation. (B) Elongation of the GHHB with normal carotid bifurcation (Group 1). (C) Elongation of the GHHB with twisted carotid bifurcation (Group 2).

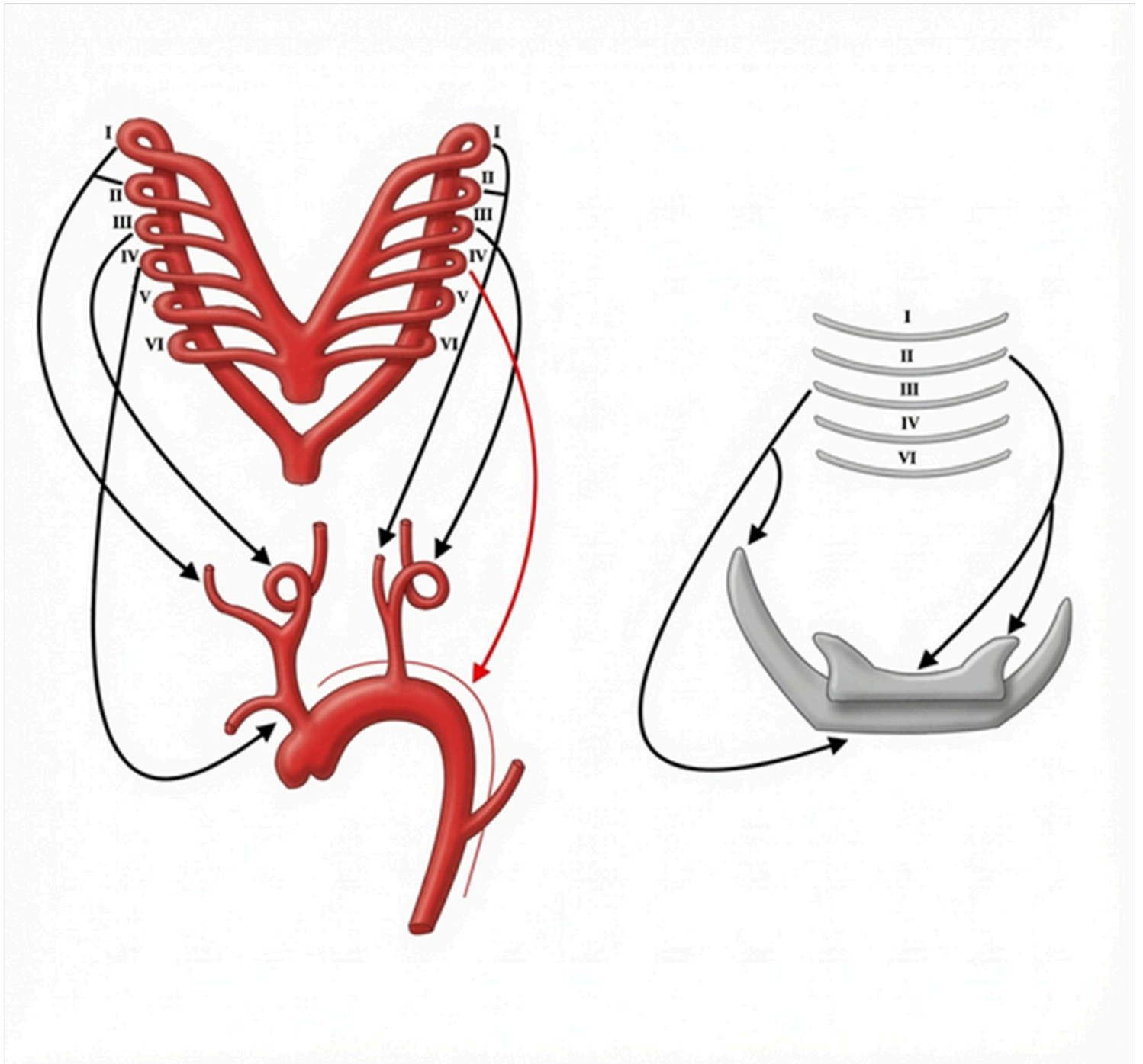


Figure 4

Schematic illustration of the embryonic origins of the carotid arterial system and the hyoid bone from the aortic and pharyngeal arches, supporting the hypothesis of a shared developmental field of the third pharyngeal arch.

Left panel: Bilateral embryonic aortic arches (I–VI) are shown in red. Black arrows indicate their remodeling into the definitive aortic arch and carotid bifurcations.

From the third aortic arch the internal carotid artery (ICA) is formed (both sides).

From the first and second aortic arches the external carotid artery (ECA) is formed (both sides).

On the right side the carotid bifurcation is shown in the twisted configuration; on the left side the bifurcation remains in the normal position.

The right fourth aortic arch contributes to the brachiocephalic trunk; the left fourth aortic arch forms the definitive aortic arch (red curved arrow).

Right-sided predominance of twisted carotid bifurcation (TCB) is consistent with the normal asymmetry of aortic-arch remodeling.

Right panel: Pharyngeal-arch cartilages (I–VI) are shown in gray. Black arrows indicate their contribution to the formation of the hyoid bone:

Second pharyngeal arch → upper part of the hyoid body and the lesser horns;

Third pharyngeal arch → lower part of the hyoid body and the greater horns (GHHB).

The diagram illustrates the anatomical basis for the proposed common-field disturbance: a developmental perturbation within the third pharyngeal arch territory can simultaneously produce elongation of the GHHB and excessive lateral migration of the external carotid artery.