Agenesis of the Left Internal Carotid Artery and Congenital Pituitary Aplasia

Abstract

Less than 0.01% of the overall society worldwide has been recorded to have internal carotid artery (ICA) agenesis, making it a remarkably uncommon embryonic vascular defect. Pituitary aplasia is another uncommon congenital disorder. In this article, we describe an incredibly rare pediatric case that had the development of one internal carotid artery and aplasia of the pituitary. The patient is a 2-and-a-half-year-old boy with dysmorphic features, such as frontal bossing, deep-seated eyes, low-set ears, depressed nasal bridge, head lags, and micropenis. He suffered generalized severe hypoglycemia and hypothyroidism on day 1 of life. He was hospitalized at our hospital for a workup and an MRI (Magnetic resonance image) of the brain to check the pituitary gland. His left internal carotid artery and carotid canal were missing, and the pituitary and Sella turcica revealed aplasia on MRI. A diagnosis of congenital panhypopituitarism coupled with agenesis of ICA was made. Here we present the first incidence of a verified complete pituitary aplasia in a patient with ICA agenesis. In the case of Congenital pituitary aplasia/hypoplasia, our experience indicates any vascular anomalies should be thoroughly evaluated. The formation of collateral circulation in unilateral ICA agenesis cases to complement the circulation and the presence of hormonal replacement therapy help such patients massively. It is not uncommon for unilateral ICA agenesis to remain asymptomatic because of the formation of collateral circulation that obtains the proper blood supply.

Keywords: *Agenesis, MRI, ICA, Congenital pituitary aplasia, Left internal carotid artery*

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Introduction

Agenesis of the internal carotid artery is an extremely uncommon neonatal abnormality.[1, 2] Less than 0.01% of the worldwide population has been reported to have this congenital vascular anomaly.[3-5] ICA Agenesis is described as the whole lack of the ICA and carotid canal; aplasia is defined as a lack of sections of the carotid canal and the ICA; and hypoplasia is defined as the narrowing of the carotid canal and the ICA. These are different possible varieties of findings in agenesis anomalies of the ICA.[3, 6] Collateral circulation forms during the embryonic period to compensate for the absence of blood supply in such cases. Therefore, the majority of instances of unilateral agenesis are asymptomatic.[4, 7] In the ICA agenesis, a 3:1 left side majority has been noted.[5, 8] We present a highly unusual example of pituitary aplasia accompanied by unilateral ICA.[9, 10]

Case description

The patient is a 2-and-a-half-year-old kid. He was delivered by cesarean section after forty weeks of gestation. His baby's weight at birth was 2,800 g, and the length of his body was 49.0 cm. There was no consanguinity. He suffered generalized severe hypoglycemia and hypothyroidism on day 1 of life. Thereafter, he went on to be admitted to the NICU (Neonatal intensive care unit) for 1 month and 15 days. He was transported to our hospital for a medical checkup. An examination revealed dysmorphic traits such as frontal bossing, deep-seated eyes, low-set ears, a depressed nasal bridge, head lags, and micropenis. The patient had no pyriform aperture stenosis and no solitary maxillary central incisor (SMCI). Cardiac and fundal examinations were normal. He had generalized severe hypoglycemia and hypothyroidism on day 1 of life. Thereafter, he was admitted to NICU for 1 month and 15 days. He was also admitted for a diagnostic work-up and MRI brain to evaluate the pituitary gland.

MRI findings showed the lack of the normal flow void of the left internal carotid canal and the first part of the left middle cerebral artery **(Figures 1 and 2)**. The pituitary gland is flat. No evident anterior or posterior pituitary gland tissue or pituitary stalk is observed. The pituitary gland's strong posterior signal is not detected in situ or

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ectopic. The left medial cerebral artery originates within the posterior circulation, and the left internal carotid artery cannot be seen. No intra, supra, retro or parasellar masses. Normal MRI appearance of the optic chiasm and corpus callosum. Otherwise, normal MR appearance of the brain parenchyma with no definite focal lesions. Diagnosis of congenital panhypopituitarism associated with agenesis of ICA was made.

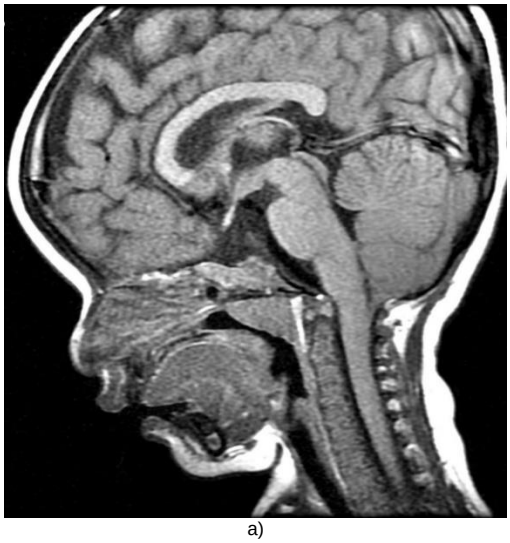
His growth and development have been normal since the beginning of the hormone replacement medication. He has no abnormalities in his nervous system. His eye fundus and eyesight are unharmed. Now, the patient is on growth hormone, thyroxine, hydrocortisone, and he is doing well.[11]

A 46, XY typical male karyotype was discovered by chromosomal analyses. No gene inconsistencies were found. A thorough evaluation was conducted, with a focus on endocrinology, metabolism, neurology, and cardiology **(Table 1)**.

Table 1. our patient's initial lab results

Test		Value	Reference Range
Thyroid function test	Cord TSH	0,01 uIU/ml	1 - 38.9
	TSH	0.005 uIU/mL	0.5 - 5.0
	FREE T3	2.8 pmol/L	12 – 30
	FREE T4	5.04 pmol/L	2.0 - 7.0
Androgens tests	Testosterone	2.35 nmol/L	2.602 - 13.877
	Follicle-stimulating hormone	0.01 mIU/ml	0 - 5.0
	Luteinizing hormone	0.04 mIU/ml	1.24 - 7.8
	Prolactin	0.57 ng/ml	0 – 17
Blood Sugar	Random Glucose	3.7 mmol\L	3.9 - 5.6
Cortisol testing	Cortisol	8.7 nmol/L	<28–717
ACTH stimulation test	ACTH	11.2 pg/ml	
	CORT at 0 min	4.6 nmol/L	
	CORT at 20 min	5.7 nmol/L	
	CORT at 30 min	7.8. nmol/L	
	CORT at 60 min	7.9 nmol/L	

ACTH: Adrenocorticotrophic hormone; TCH: Thyroid-stimulating hormone



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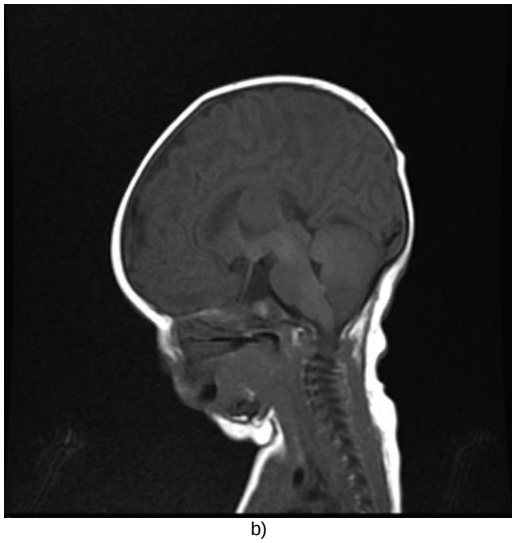

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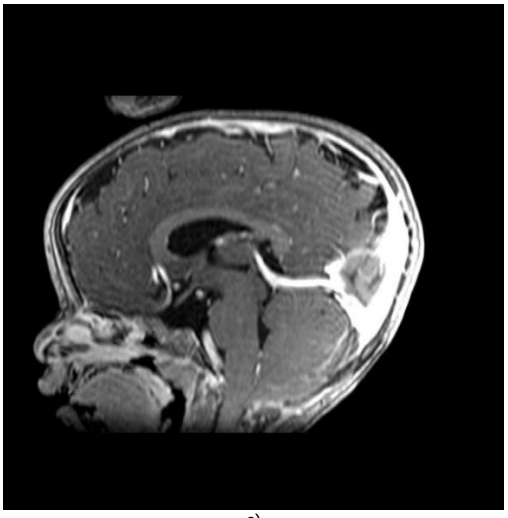

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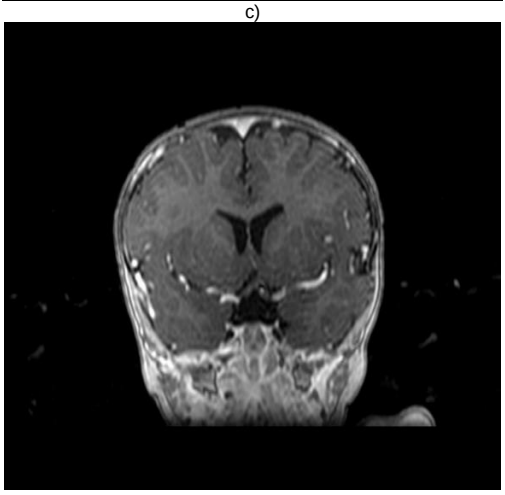
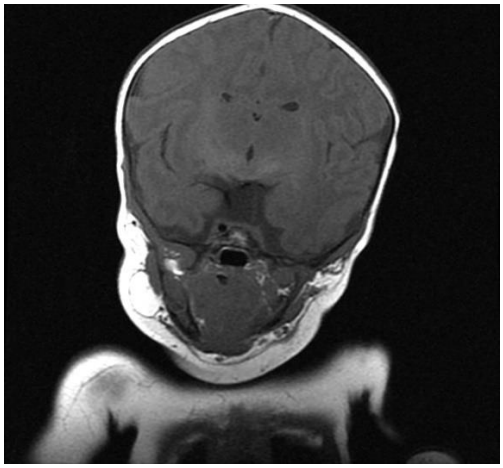


d)

Figure 1. a-c) figures showing MRI brain sagittal views of the patient where the pituitary sella is flat. No obvious anterior or posterior pituitary gland tissue or pituitary stalk is seen. The posterior pituitary bright signal is not visualized in site or ectopic. d) coronal MRI scan showing absence of pituitary gland tissue.

Generally, no intra, supra, retro, or parasellar masses. Normal MRI appearance of the optic chiasm and corpus callosum. Otherwise, normal MR appearance of the brain parenchyma with no definite focal lesions.



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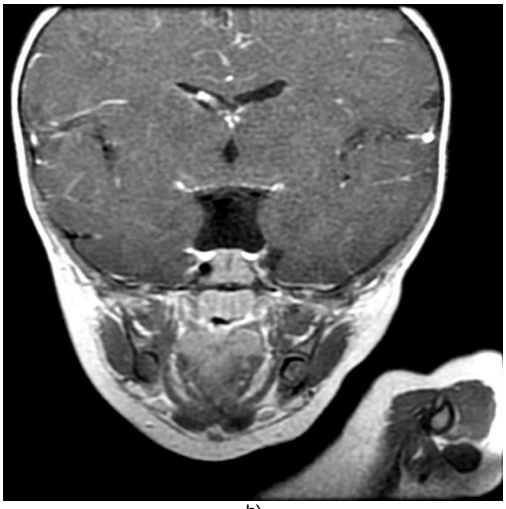

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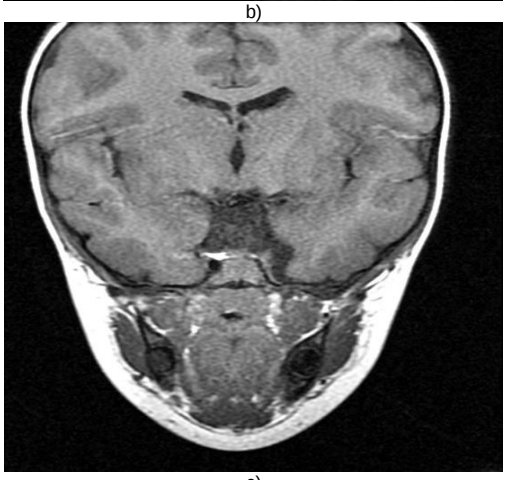

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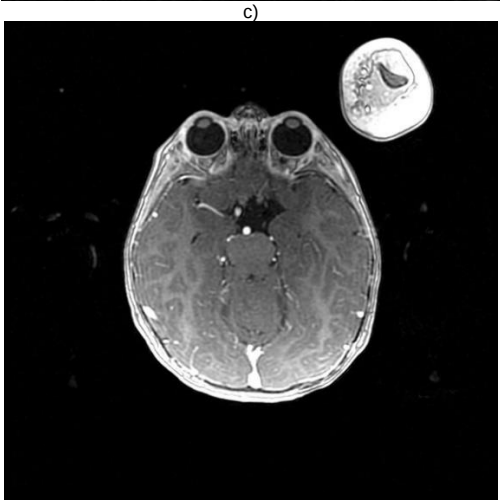

d)

Figure 2. a-c) show coronal MRI brain images, and d) is a view in which the left midcranial artery is coming from the posterior circulation and the left internal carotid artery is not visible (agenesis).

Results and Discussion

This case study describes a 2-and-a-half-year-old kid who had agenesis of the ICA, pituitary aplasia, and subsequent hypopituitarism, all of which were linked to various comorbidities.[12, 13] These two abnormalities are uncommon in general, and it is even more unusual for them to occur

together.[14, 15] Congenital hypopituitarism (CH) is an uncommon congenital condition that can present as multiple pituitary hormone involvement (combined pituitary hormone deficits; CPHD) or an isolated hormone absence, with isolated growth hormone deficiency (IGHD) being the most prevalent form.[8, 16]

During embryogenesis, variations in the structure of the arteries that carry blood to the brain during embryogenesis may take place, and these variations may affect how vascular disease develops. The first stage in which changes might occur is angiogenesis, which primarily involves the sprouting of parent arteries. The internal carotid artery, which initially forms in the 5th week of embryological life, supplies the primitive brain with all the blood it needs. The ICA begins to form during the 3 mm stage of developmental embryology. It develops from the initial and third aortic arches, the paired dorsal aorta, and other blood vessels. The root of the ICA is composed of the third aortic arch. The intermediate parts of the ICA emerge from the dorsal aorta at the initial and third aortic arches.[17]

The anterior neural ridge, the most anterior portion of the neural plate in the embryo, is the origin of the pituitary or adenohypophyseal placode, which marks the border between the anterior section of the ectoderm and the neuroectoderm. The neural plate cells that give birth to the telencephalon, hypothalamus, and posterior pituitary are located right next to the pituitary placode. The Rathke's pouch, an invagination of the oral ectoderm that will eventually give birth to the anterior and intermediate lobes of the pituitary, is located in the pituitary placode.[18]

A low number of cases of CPHD linked to ICA have been documented such as Inamo and Harada [19] and Tanaka *et al*.[20] In both cases, there was Pituitary hypoplasia associated with anomaly or absence of one of the ICA. There were also deficiencies in TSH, GH (Growth hormone), and ACTH. there were central diabetes insipidus and possible hypogonadism, too.[19, 20] In our case, the pituitary gland was completely absent, and both lobes were in association with left ICA agenesis. Similarly, our patient has TSH, GH, and ACTH deficiency. He also has a micropenis.

Generally, ICA agenesis is rarely connected to congenital hypopituitarism. Despite the fact that many of these publications did not address, or perhaps detailed insufficiently, the potential related endocrinological problems and treatments, all documented instances involved individuals with CPHD and the agenesis of ICA.[21] Due to this, many people have proposed that pituitary disorders have vascular causes. During development, the third aortic arch gives rise to the ICA. However, it is debatable whether the third aortic arch or the aortic sac serves as the origin of the common and external carotid arteries.[22] Most newborns who have this vascular defect are asymptomatic, and the rarity of pediatric ICA agenesis diagnoses suggests that artery collateral channels appear to maintain a sufficient level of brain perfusion.[23, 24]

The development abnormalities anencephaly, holoprosencephaly, septo-optic dysplasia, and empty-sella syndrome can all coexist with pituitary aplasia. A solitary maxillary central incisor or midfacial deformities imply a significant chance of growth hormone insufficiency. Although pituitary aplasia without brain or skull abnormalities is uncommon, a shortage of blood flow may cause congenital hypopituitarism by creating congenital pituitary hypoplasia (CPH).[25-27]

Congenital hypopituitarism is frequently difficult to diagnose. Testing for dynamic pituitary function in newborn infants is not frequently done. Infancy-related severe hypoglycemia brought on by GH and cortisol deficiency is often treated with both hormone replacement therapy.[28, 29]

Low blood levels of GH and cortisol seen in a baby with ketonuria, and hypoglycemia strongly suggests hypopituitarism. Because there are insufficient glycogen reserves in the liver, this type of hypoglycemia does not respond to glucagon. All of such patients had significant symptoms of hypoglycemia, but in instances 1 and 2, the appropriate replacement medication was administered too late, resulting in irreparable brain damage.[28] Each patient with such an anomaly differs in the endocrinological, anatomical, and clinical manifestations **(Table 2)** which contains a review of the previously reported similar cases in the published literature.

Table 2. The clinical and anatomical characteristics of patients with congenital hypopituitarism and internal carotid artery anomalies.

	Sex	Age at report	Pituitary MRI	Carotid MRI	Clinical Characteristics	Report date	Author
1	Not specified	Not specified	Not specified (except for normal posterior pituitary)	Absence of ICA and carotid Canal	Not specified.	1994	Triulzi [30]
2	M	18 months	Pituitary hypoplasia	Anomaly of right ICA	TSH, GH, ACTH deficiency. Central diabetes insipidus. Possible hypogonadism.	1996	Tanaka [20]
3	M	3 weeks	Absence of anterior pituitary with ectopic posterior pituitary.	Absence of the left ICA and carotid canal	GH, TSH, ACTH and gonadotropin deficiency. Microphallus.	1996	Shulman [31]

4	F	5 months	Absence of anterior pituitary and ectopic pituitary posterior lobe.	Absence of right common carotid artery, right ICA, right anterior cerebral artery.	TSH, ACTH, and GH deficiency. No evidence of diabetes insipidus. Genitalia were normal. Single central maxillary incisor.	1999	Kjellin [32]
5	M	23 years	Absence of anterior pituitary and ectopic pituitary posterior lobe	Absence of right ICA and carotid canal and A1 segment of the right anterior cerebral artery.	Congenital microphthalmia with cataract and coloboma of the right eye, encephalocele. Hormonal substitution treatments not specified.	1999	Blustajn [33]
6	M	37 years	Absence of anterior pituitary	Hypoplasia of right ICA and carotid canal.	Amblyopia of the left eye caused by an optic nerve coloboma, encephalocele. Hormonal substitution treatments not specified.	1999	Blustajn [33]
7	F	29 years	Pituitary hypoplasia and ectopic pituitary posterior lobe.	Absence of right ICA, carotid canal, and A1 segment of the right anterior cerebral artery.	TSH, ACTH, and GH deficiency. No evidence of diabetes insipidus. Hypogonadism. Chiari I malformation with syringomyelia.	2001	Mellado [34]
8	M	5 years	Absence of anterior pituitary and ectopic pituitary posterior lobe.	Absence of left ICA and carotid canal, A1 segment of the left anterior cerebral artery and the anterior communicating artery.	Retrognathia, microphallus, and cryptorchidism. TSH, ACTH, GH deficiency, but no evidence of diabetes insipid. Possible hypogonadism.	2002	Moon [35]
9	M	11 years	Pituitary hypoplasia	Absence of left ICA and carotid canal; hypoplasia of A1 segment of left anterior cerebral artery.	TSH, ACTH, GH deficiency. Central diabetes insipidus. Microphallus with possible hypogonadism	2003	Inamo [19]
10	F	10 years	Hypoplastic anterior pituitary, flat sella turcica, absent pituitary stalk.	Agenesis of the left ICA and the left carotid canal	GH, TSH, gonadotropin deficiency. No evidence of diabetes insipidus.	2012	Lamine [36]
11	F	7 Months	Adenohypophyseal hypoplasia with a lack of posterior pituitary hyperintensity	Absence of the left ICA	Desaturation episodes, recurrent respiratory infections. Short hands and feet. GH, ACTH, and TSH deficiency.	2012	Savasta [15]
12	F	2 years	Adenohypophyseal hypoplasia with a lack of posterior pituitary hyperintensity	Absence of the right ICA	GH, TSH, and gonadotropin deficiency. No clinical evidence of diabetes insipid.		
13	M	17 years	Adenohypophyseal hypoplasia with a lack of posterior pituitary hyperintensity.	Agenesis of the left ICA	GH deficiency. No other pituitary deficiencies. No clinical evidence of diabetes insipidus.	2015	Stage [8]
14	M	2 years	Absence of obvious anterior or posterior pituitary gland tissue or pituitary stalk.	Agenesis of left ICA	GH, TSH, and ACTH Deficiency. Frontal bossing, deep-seated eyes, low set ears, depressed nasal bridge, head lags, and micropenis.	2023	Our case

Conclusion

Here, we provide the first instance of a patient with ICA agenesis with a verified full pituitary aplasia. In the case of Congenital pituitary aplasia/hypoplasia, our experience indicates any vascular anomalies should be thoroughly evaluated. The formation of collateral circulation in unilateral ICA agenesis cases to complement the circulation and the presence of hormonal replacement therapy help such patients massively. It is not uncommon for the unilateral ICA agenesis to remain asymptomatic because of the formation of collateral circulation that obtains the proper blood supply.

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Conflict of interest

None.

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Ethics statement
None.

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