Cerebral Artery Hypoplasia in a Select Adult Kenyan Population

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Abstract

Background  Hypoplasia of cerebral arteries predisposes to stroke and cerebral aneurysms which have an increased incidence in sub-Saharan Africa. The frequency and pattern of cerebral artery hypoplasia, however, shows population variations, and data from the African population remain scanty.

Objectives  This study aimed to determine the percentage of hypoplasia in the anterior, middle, and posterior cerebral, anterior and posterior communicating, basilar, and vertebral arteries.

Materials and Methods  Sections of the basilar, vertebral, posterior, and anterior communicating arteries and anterior, middle, and posterior cerebral arteries were taken, processed for histology, and examined with a light microscope at ×40. The images of the vessels were taken by a photomicroscope and circumference analyzed with the aid of Scion image analyzer. The average diameter of 10 sections was taken to be the diameter of the artery in analysis. Hypoplasia was then defined as internal diameter ≤1 mm. Photographs of representative samples of asymmetry were taken, data were analyzed using SPSS, and gender differences were analyzed using the Student’s test. Results were presented in tables.

Results  Two hundred and eighteen formalin-fixed brains of adult Kenyans at the Department of Human Anatomy, University of Nairobi, were studied. Of the 218, 48 brains (22%) did not have vessels with any form of hypoplasia while 170 (78%) did have vessels. Of these, anterior circulation hypoplasia (anterior cerebral artery and posterior communicating artery) was seen in 100 brains (46%) and posterior circulation hypoplasia (middle and posterior cerebral, basilar, and vertebral arteries) in 69 brains (32%).

Conclusion  Cerebral arterial hypoplasia is frequent in the select adult Kenyan population.

Keywords

► anterior
► cerebral artery
► hypoplasia
► posterior

Introduction

Hypoplasia in cerebral arteries has been shown to alter hemodynamics in the affected arteries as well as the normal arteries in the same vascular bed.1,2 It further influences the pattern of cerebral blood flow3 and predisposes to atherosclerosis of large and small cerebral arteries alike, causing stroke and transient ischemic attacks.1,2,4 Hypoplasia also causes cerebral aneurysms,5 may be associated with deformities of other intracranial arteries,6,7 and can be confused for pathological arterial occlusion.4 Cerebral hypoplasia has the potential to cause cerebral hypoperfusion and thus predisposes to cognitive dysfunction and Alzheimer’s disease.9 Knowledge on cerebral hypoplasia is important during instrumentation of arteries as well as mitigating complications of endovascular treatment and prognostication of cerebrovascular
Arachnoid mater was gently peeled from the base of the brain to expose the basilar, vertebral, posterior, middle, and anterior cerebral arteries and the posterior and anterior communicating arteries. Two-millimeter specimens taken from each of the arteries were then fixed in 10% formalin and processed for paraffin embedding and sectioning. Ten 5-μm serial sections from each arterial site were stained with hematoxylin/eosin and examined with the help of a Leica DM3000 light microscope at ×40. The images taken by the photomicroscope were digitized. Subsequently, the internal circumference of each of the 10 sections from each site of the artery was determined using Scion image analyzer version 1.46. To do this, the image was first set to scale; then, with the help of the line tool, a line was drawn round the lumen of the artery to give a value equivalent to the circumference. Only complete sections were included. The diameter (in millimeters) was calculated from the formula $D = C/\pi$, where $D$ is the diameter, $C$ is the circumference, and $\pi = 3.14$. The average diameter of the 10 sections was taken to be the diameter of that artery.

Artery hypoplasia was defined as internal diameter ≤1 mm. Photographs of representative samples of asymmetry were taken using a high-resolution digital camera. Data were analyzed using Statistical Package for the Social Science (SPSS; IBM, New York, United States) for Windows. Gender differences were analyzed using the Student’s test at 95% confidence intervals where value of ≤0.05 was taken as significant. Results were presented in tables.

**Results**

Of the 218 dissected brain specimens, 170 presented with hypoplasia. The remaining 48 did not exhibit hypoplasia. Of the 170, anterior cerebral artery hypoplasia was recorded in 13 (6%) brains with 87 (40%) showing posterior communicating artery (PCoA) hypoplasia, 26 (12%) showing posterior cerebral artery hypoplasia, 6 (3%) showing basilar artery hypoplasia, and 37 (17%) showing vertebral artery hypoplasia (VAH). All the arteries studied, except middle cerebral, displayed hypoplasia. The findings have been summarized (►Tables 2 and 3 and ►Fig. 1–5).

**Discussion**

Data from our study revealed that of all the arteries studied, the middle cerebral artery did not exhibit hypoplasia.
This finding is similar to prevailing literature from other populations.\textsuperscript{12}

Cerebral artery hypoplasia was more common in the anterior circulation (46\%). This is consistent with contemporary literature reports.\textsuperscript{3,15} The mechanisms by which cerebral artery hypoplasia occurs are considered to be related to hemodynamic factors. In this case, the differential growth of the various parts of the brain will continuously change the hemodynamic demands and consequently the flow patterns in the cerebral arteries.\textsuperscript{16} It is, therefore, conceivable that if a selected part of the brain does not develop, the change in the hemodynamic demand in that area will be reduced as noted by Van Overbeeke et al.\textsuperscript{17} The frequency of anterior circulation (anterior cerebral artery and PCoA) hypoplasia varied between arteries.

The frequency of A1 hypoplasia is reported to range between 1 and 15\%,\textsuperscript{18-20} The A1 segment is the principal supplier of collateral blood flow and origin to striate arteries, which supply the hypothalamus, septum pellucidum, and corpus striatum. Hypoperfusion may, therefore, affect functioning in these areas. Further, in patients with hypoplastic A1 segments, total cerebral blood flow within the ipsilateral internal carotid is usually lower than in the contralateral internal carotid artery (ICA).\textsuperscript{14} This may cause global cerebral hypoperfusion. Accordingly, A1 hypoplasia is a risk factor for stroke-related vascular diseases,\textsuperscript{20,21} has been implicated in mild cognitive impairment,\textsuperscript{22,23} and may present with monoplegia, abulia, and urinary incontinence. It is also a risk factor for the occurrence of anterior communicating artery (ACoA) aneurysms.\textsuperscript{24} In the current study, A1 hypoplasia occurred in 6\%, which was notably higher when compared with the Polish (3\%),\textsuperscript{25} the Indians (4\%),\textsuperscript{10} and the Sri Lankans (5\%) (\textsuperscript{\textendash}Table 4). It was, however, lower when compared with the Taiwanese (15\%).\textsuperscript{19} The relatively higher prevalence observed among Kenyans as compared with many of the other populations may explain the high prevalence of aneurysms of ACoA.\textsuperscript{11,27} Pertinent to this suggestion is the observation that A1 hypoplasia predisposes to ACoA aneurysm.\textsuperscript{5}

Posterior cerebral artery was hypoplastic in 12\% of the cases. This was higher than that recorded in the American\textsuperscript{28} (6.3\%), Indian\textsuperscript{29} (5.29\%), Polish\textsuperscript{12} (4\%), and Pakistani\textsuperscript{30} (0\%) populations (\textsuperscript{\textendash}Table 5). It was notably lower when

![Fig. 1](image)

**Fig. 1** (A) Unilateral hypoplasia of A1 segment of the left anterior cerebral artery. Note the asterisk which highlights the variant artery. (B) Unilateral hypoplasia of A1 segment right anterior cerebral artery. Note the asterisk which highlights the variant segment. (C) Unilateral hypoplasia of A2 segment of anterior cerebral artery 2. Abbreviations: AC 1, first part of anterior cerebral; BA, basilar artery; MCA, middle cerebral artery.
compared with the German (37.5%) population and other populations. The higher prevalence in the Kenyan setting as compared with most of the other populations may predispose to bilateral paramedian thalamic strokes and ischemic strokes, which have been reported to be high in Africa.

The 40% incidence of PCoA hypoplasia observed in the current study is lower to the 51% reported for the Sri Lankan population. It is, however, much higher than that noted in Korean (19.35%), Dutch (28%), Indian (23.3%), and Polish (24%) populations and previous study on the Kenyan population (►Table 6). The high variability even among ethnically related Caucasian populations suggests that epigenetic factors are involved in the causation of this variation.

Hypoplasia of PCoA increases the risk of atherosclerosis of large and small intracranial arteries and hence ischemic posterior circulatory strokes.

Basilar artery hypoplasia has been reported to be a rare occurrence frequently linked to persistent carotid–basilar communication or correlated with the presence of a large PCoA with persistent flow from the carotid to vertebrobasilar system. Cases of these variations are scarce in the literature with a case study being reported in the Italian population and 1 case of 62 specimens being noted in the Spanish population. In our setting, the basilar artery was hypoplastic in 3% of the sample population. Basilar artery hypoplasia has been shown to occur following persistent axial nonfusión of the distal basilar artery, which develops
from the caudal division of the ICA to the posterior inferior cerebellar artery termination of the vertebral artery. Basilar artery hypoplasia has been linked to chronic brain hypoperfusion and a subsequent posterior circulation insufficiency.

Table 4  Frequency of hypoplasia on anterior cerebral artery in different populations

<table>
<thead>
<tr>
<th>Reference</th>
<th>Population</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chuang et al, 2007</td>
<td>Taiwanese</td>
<td>15</td>
</tr>
<tr>
<td>De Silva et al, 2009</td>
<td>Sri Lankan</td>
<td>5</td>
</tr>
<tr>
<td>Klimek-Piotrowska et al, 2016</td>
<td>Polish</td>
<td>1.0</td>
</tr>
<tr>
<td>Makowicz et al, 2013</td>
<td>Polish</td>
<td>3</td>
</tr>
<tr>
<td>Iqbal, 2013</td>
<td>Indian</td>
<td>4</td>
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<tr>
<td>Current study</td>
<td>Kenyan</td>
<td>6</td>
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</table>

Table 5  Frequency of hypoplasia of posterior cerebral artery in various populations

<table>
<thead>
<tr>
<th>Reference</th>
<th>Population</th>
<th>Frequency</th>
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<tr>
<td>Förster et al, 2014</td>
<td>German</td>
<td>37.5</td>
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<tr>
<td>Alpers et al, 1959</td>
<td>American</td>
<td>6.3</td>
</tr>
<tr>
<td>Gunnal et al, 2016</td>
<td>Indian</td>
<td>5.29</td>
</tr>
<tr>
<td>Klimek-Piotrowska et al, 2016</td>
<td>Polish</td>
<td>4</td>
</tr>
<tr>
<td>Siddiqi et al, 2013</td>
<td>Pakistani</td>
<td>0</td>
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<tr>
<td>Puchades-Orts et al, 1976</td>
<td></td>
<td>11.3</td>
</tr>
<tr>
<td>Milenković et al, 1985</td>
<td></td>
<td>7.68</td>
</tr>
<tr>
<td>Iqbal, 2013</td>
<td>Indian</td>
<td>6</td>
</tr>
<tr>
<td>Current study</td>
<td>Kenyan</td>
<td>12</td>
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</tbody>
</table>

Fig. 4  Basilar artery hypoplasia.

Fig. 5 (A) Mild left vertebral artery hypoplasia. (B) Mild right vertebral artery hypoplasia. Abbreviations: BA, basilar artery.
The current study revealed a 17% prevalence of VAH, higher than those reported for most Caucasian\textsuperscript{41–47} populations (\textit{Table 7}). The variations of these vessels, similar to the above, have been shown to predispose to lacunar infarcts and strokes that have a high prevalence in our setting. This VAH-associated risk is equivalent to that of other conventional risk factors such as hypertension, diabetes, smoking, and dyslipidemia.\textsuperscript{4} Accordingly, nearly 30% of the Kenyan population may be at risk of posterior circulatory stroke and the other complications. This implies that in patients who present with vertebrobasilar insufficiency, VAH should be considered.

**General Remarks**

The frequency of hypoplasia varies between populations. These variations are probably genetically determined, develop early in embryonic life, and persist in postnatal life.\textsuperscript{13} It is also worth noting that in our setting, hypoplasia was predominant in the anterior circulation, specifically in the PCoA.

**Conclusion**

The frequency of cerebral arterial hypoplasia is high in the Kenyan population and is more common in the anterior circulation. Due care should be taken during neuroradiological, investigative, and interventional procedures; and patients should be followed up when presenting with cerebrovascular disease.

**Funding**

None.

**Conflict of Interest**

None declared.

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**References**


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**Table 6** Frequency of hypoplasia of posterior communicating artery in various populations

<table>
<thead>
<tr>
<th>References</th>
<th>Population</th>
<th>Prevalence (%)</th>
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<tbody>
<tr>
<td>Chuang et al, 2008\textsuperscript{44}</td>
<td>Korean</td>
<td>19.35</td>
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<tr>
<td>De Silva et al, 2009\textsuperscript{46}</td>
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<td>51</td>
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<tr>
<td>Dzięrzanowski et al, 2014\textsuperscript{48}</td>
<td>Polish</td>
<td>24</td>
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<tr>
<td>Krabbe-Hartkamp et al, 1998\textsuperscript{46}</td>
<td>Dutch</td>
<td>28</td>
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<tr>
<td>Saha et al, 2013\textsuperscript{47}</td>
<td>Indian</td>
<td>23.3</td>
</tr>
<tr>
<td>Siddiqi et al, 2014\textsuperscript{48}</td>
<td>Pakistani</td>
<td>39.5</td>
</tr>
<tr>
<td>Windle 1888\textsuperscript{49}</td>
<td>British</td>
<td>25</td>
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<tr>
<td>Sinkeet et al, 2010\textsuperscript{49}</td>
<td>Kenyan</td>
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<td>Iqbal, 2013\textsuperscript{49}</td>
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<td>10</td>
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<td>Current study</td>
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**Table 7** Frequency of vertebral artery hypoplasia in various populations

<table>
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<tr>
<th>References</th>
<th>Population</th>
<th>Prevalence of VAH (%)</th>
</tr>
</thead>
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<td>Chuang et al, 2008\textsuperscript{44}</td>
<td>Taiwanese</td>
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<tr>
<td>Oder et al, 1998\textsuperscript{42}</td>
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<tr>
<td>Park et al, 2007\textsuperscript{44}</td>
<td>Korean</td>
<td>26.5</td>
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<td>Peterson et al, 2010\textsuperscript{45}</td>
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<tr>
<td>Thierfelder et al, 2014\textsuperscript{46}</td>
<td>American</td>
<td>15.6</td>
</tr>
<tr>
<td>Hu et al, 2013\textsuperscript{47}</td>
<td>Chinese</td>
<td>10</td>
</tr>
<tr>
<td>Current study</td>
<td>Kenyan</td>
<td>17</td>
</tr>
</tbody>
</table>

Abbreviation: VAH, vertebral artery hypoplasia.
27 Nabaweesi-Batuka J, Kitungu PK, Kiboi JG. Pattern of cerebral aneurysms in a Kenyan population as seen at an urban hospital. World Neurosurg 2016;87:255–265
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