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KIMMERLE'S ANOMALY AS A FACTOR OF CERVICOGENIC HEADACHE AND VESTIBULO-COORDINATIVE DISORDERS: A CLINICAL CASE AND LITERATURE REVIEW

Abstract.

Background. Kimmerle's anomaly, also known as ponticulus posticus or arcuate foramen of the atlas, is a congenital or acquired osseous bridge over the groove of the vertebral artery on the posterior arch of C1. Although it is frequently detected incidentally, in selected patients it may participate in vertebral artery irritation, extravascular compression, cervicogenic headache and vestibulo-coordinative dysfunction.

Methods. A single-patient clinical observation was combined with a focused narrative literature review. The diagnostic work-up included cervical magnetic resonance imaging, multislice computed tomography with three-dimensional reconstruction, computed tomography angiography and duplex Doppler ultrasonography of the vertebral arteries with rotational functional tests.

Results. A 34-year-old patient presented with recurrent occipital headache, dizziness, tinnitus, gait unsteadiness and symptom exacerbation during head rotation. Imaging revealed a complete right-sided arcuate foramen of C1 with close topographic relationship to the V3 segment of the vertebral artery. Functional Doppler testing demonstrated rotation-dependent reduction of vertebral artery flow velocity. Differential diagnosis excluded vestibular migraine, benign paroxysmal positional vertigo, atherosclerotic vertebrobasilar insufficiency, isolated cervical osteochondrosis and craniovertebral instability.

Conclusion. Symptomatic Kimmerle's anomaly should not be interpreted as an isolated radiological variant. Its clinical significance emerges only when structural C1 anatomy, reproducible cervico-vestibular symptoms, positional vascular changes and exclusion of competing vestibular, vascular and cervical disorders are evaluated together in a multidisciplinary and dynamically verified diagnostic model before rational treatment planning decisions are made.

Keywords: Kimmerle's anomaly, ponticulus posticus, arcuate foramen, cervicogenic headache, vertebral artery, vertebrobasilar insufficiency, dizziness, tinnitus, cervical spine, computed tomography angiography, Doppler ultrasound.

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АНОМАЛИЯ КИММЕРЛИ КАК ФАКТОР ЦЕРВИКОГЕННОЙ ГОЛОВНОЙ БОЛИ И ВЕСТИБУЛО-КООРДИНАТОРНЫХ НАРУШЕНИЙ: КЛИНИЧЕСКИЙ СЛУЧАЙ И ОБЗОР ЛИТЕРАТУРЫ

Аннотация.

Актуальность. Аномалия Киммерли, также известная как ponticulus posticus или дугообразное отверстие атланта, представляет собой костную перемычку над бороздой позвоночной артерии на задней дуге C1. В большинстве случаев она выявляется как случайная анатомо-рентгенологическая находка, однако у части пациентов может быть связана с раздражением или экстравазальной компрессией позвоночной артерии, цервикогенной головной болью и вестибуло-координаторными нарушениями.

Материалы и методы. Представлено клиническое наблюдение пациента с обзором литературы. Диагностика включала МРТ шейного отдела позвоночника, МСКТ с 3D-реконструкцией, КТ-ангиографию и ультразвуковую допплерографию позвоночных артерий с ротационными пробами.

Результаты. У пациента 34 лет отмечались рецидивирующая затылочная головная боль, головокружение, шум в ушах, шаткость походки и усиление симптомов при поворотах головы. Выявлена полная правосторонняя аномалия Киммерли с позиционно-зависимым снижением кровотока по позвоночной артерии.

Заключение. Симптомную аномалию Киммерли необходимо оценивать не изолированно, а в связи с клиникой, динамическими сосудистыми пробами и дифференциальной диагностикой.

Ключевые слова: Аномалия Киммерли, ponticulus posticus, дугообразное отверстие атланта, цервикогенная головная боль, позвоночная артерия, вертебробазилярная недостаточность, головокружение, шум в ушах, шейный отдел позвоночника, КТ-ангиография, допплерография.

KIMMERLE ANOMALIYASI SERVIKOGEN BOSH OG‘RIG‘I VA VESTIBULO-KOORDINATSION BUZILISHLAR OMILI SIFATIDA: KLINIK HOLAT VA ADABIYOTLAR SHARHI

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Annotatsiya.

Dolzarbligi. Kimmerle anomaliyasi, shuningdek ponticulus posticus yoki atlasning yoysimon teshigi deb ataladi, C1 umurtqaning orqa yoyida joylashgan umurtqa arteriyasi egati ustida suyak ko‘prikchasi hosil bo‘lishi bilan tavsiflanadi. Ko‘p hollarda u tasodifiy rentgenologik topilma sifatida aniqlanadi, biroq ayrim bemorlarda umurtqa arteriyasining ta’sirlanishi yoki ekstravazal kompressiyasi, servikogen bosh og‘rig‘i va vestibulo-koordinatsion buzilishlar bilan bog‘liq bo‘lishi mumkin.

Materiallar va usullar. Klinik kuzatuv adabiyotlar sharhi bilan birgalikda tahlil qilindi. Tekshiruvlarga bo‘yin umurtqa pog‘onasi MRT, 3D-rekonstruksiyali MSKT, KT-angiografiya va boshni burish sinamalari bilan umurtqa arteriyalarining dopplerografiyasi kiritildi.

Natijalar. 34 yoshli bemorda takrorlanuvchi ensa sohasi bosh og‘rig‘i, bosh aylanishi, quloqda shovqin, yurishda beqarorlik va boshni burishda simptomlarning kuchayishi kuzatildi.

Xulosa. Simptomatik Kimmerle anomaliyasini klinik belgilar, dinamik qon-tomir sinamalari va differensial diagnostika bilan birgalikda baholash zarur.

Kalit so‘zlar: Kimmerle anomaliyasi, ponticulus posticus, atlasning yoysimon teshigi, servikogen bosh og‘rig‘i, umurtqa arteriyasi, vertebrobazilyar yetishmovchilik, bosh aylanishi, quloqda shovqin, bo‘yin umurtqa pog‘onasi, KT-angiografiya, dopplerografiya.

Introduction. Kimmerle’s anomaly is an anatomical variant of the atlas characterized by complete or incomplete ossification of the posterior atlanto-occipital membrane over the groove of the vertebral artery. As a result, a bony bridge is formed on the posterior arch of C1, converting the vertebral artery groove into a partial or complete osseous canal. In anatomical, radiological and neurosurgical literature, the same structure is described using several synonymous terms, including ponticulus posticus, arcuate foramen, posterior atlantal foramen and retroarticular canal. This multiplicity of terminology is not only semantic, because it reflects the borderline position of this anomaly between normal anatomical variation, radiological finding and clinically significant craniocervical pathology [1, 2, 5]. The clinical significance of Kimmerle’s anomaly remains debated. In many individuals, the osseous bridge is discovered incidentally during radiography, computed tomography or cone-beam computed tomography of the cervical spine. Meta-analytic data and population-based imaging studies indicate that ponticulus posticus is not a rare finding, and its incomplete form may be more frequent than the complete form [3, 4, 6]. However, prevalence alone does not define clinical relevance. A radiologically visible bony bridge becomes clinically meaningful only when it is associated with reproducible symptoms, a plausible neurovascular mechanism, dynamic positional changes of vertebral artery flow, and exclusion of more common causes of headache, vertigo and imbalance [2, 5, 7]. From a pathophysiological viewpoint, the anatomical importance of Kimmerle’s anomaly is determined by its relationship with the V3 segment of the vertebral artery, which passes through the transverse foramen of C1, curves posteriorly around the lateral mass of the atlas, lies in the sulcus arteriae vertebralis, and then enters the cranial cavity through the foramen magnum. This segment is mobile and may be exposed to stretching, bending and compression during rotation, extension and combined movements of the head. Formation of an osseous canal around the artery may reduce the adaptive space available for vascular displacement. In selected patients, this may contribute to mechanical irritation of the periarterial sympathetic plexus, compression of the vertebral artery, irritation of the suboccipital nerve, or local disturbance of vertebrobasilar hemodynamics during provocative head positions [2, 8, 9].

The symptom complex attributed to symptomatic Kimmerle’s anomaly is heterogeneous. It may include occipital or suboccipital headache, cervicogenic headache, cervicalgia, dizziness, tinnitus, transient visual disturbances, nausea, gait unsteadiness, facial paresthesia, drop-like episodes, and exacerbation of symptoms during rotation or extension of the head. Nevertheless, none of these symptoms is specific. Similar complaints may occur in vestibular migraine, benign paroxysmal positional vertigo, vertebral artery dissection, atherosclerotic vertebrobasilar insufficiency, cervical spondylosis, craniovertebral instability, Chiari malformation, anxiety-related dizziness and other neurological or otoneurological disorders [15, 17, 27]

This diagnostic ambiguity explains why symptomatic Kimmerle's anomaly should not be diagnosed merely on the basis of radiological detection of ponticulus posticus. Conversely, it should not be dismissed automatically as an incidental finding when the patient has typical cervico-vestibular complaints, symptom dependence on head rotation, objective evidence of altered vertebral artery flow, and absence of other more probable explanations. The main scientific and practical challenge is therefore not simply to identify the bony bridge, but to determine whether the anomaly participates in the patient's clinical syndrome [5, 9, 13]. The present article describes a clinical case of symptomatic Kimmerle's anomaly manifesting as cervicogenic occipital headache, dizziness, tinnitus and vestibulo-coordinative disorders. The case is used as a clinical model to develop an integrated diagnostic sequence combining neurological examination, cervical magnetic resonance imaging, multislice computed tomography, computed tomography angiography, duplex Doppler ultrasonography of the vertebral arteries with functional rotational tests, and differential diagnosis with common vestibular and cervical disorders. The originality of the article lies in the practical justification of a multimodal diagnostic approach in which Kimmerle's anomaly is evaluated not as an isolated anatomical finding, but as a possible functional neurovascular factor within the broader clinical context of the craniocervical junction [1, 3, 5, 9].

Materials and Methods. This work was designed as a clinical case report with a focused narrative review of the literature. The clinical material was based on the observation of one adult patient with occipital headache, dizziness, tinnitus, gait unsteadiness and symptom exacerbation during head rotation. The patient underwent neurological, radiological and vascular evaluation in order to determine whether the detected Kimmerle's anomaly had clinical and hemodynamic significance. The diagnostic strategy consisted of several consecutive stages. The first stage included clinical interview, analysis of headache characteristics, assessment of vestibular complaints, evaluation of symptom triggers and exclusion of urgent neurological conditions. Special attention was paid to the relationship between symptoms and neck movements, especially rotation and extension. The second stage included detailed neurological examination with assessment of cranial nerves, motor system, tendon reflexes, sensory function, coordination, gait, cervical range of motion and signs of pyramidal involvement. The third stage included magnetic resonance imaging of the brain and cervical spine to exclude intracranial mass lesions, Chiari malformation, demyelinating disease, cervical myelopathy, disc herniation and spinal canal stenosis. The fourth stage consisted of multislice computed tomography of the craniovertebral junction with multiplanar and three-dimensional reconstruction to characterize the morphology of the atlas and to define the completeness, side and topography of the bony bridge. The fifth stage included computed tomography angiography to evaluate the course, caliber, dominance and possible indentation of the vertebral arteries. The sixth stage included duplex Doppler ultrasonography of the vertebral arteries in neutral head position and during functional rotational tests.

The literature review focused on anatomical, radiological and clinical publications concerning ponticulus posticus, Kimmerle's anomaly, arcuate foramen, vertebral artery compression, cervicogenic headache and vertebrobasilar symptoms. The review also included diagnostic publications concerning vestibular migraine and benign paroxysmal positional vertigo, because these disorders are among the most frequent differential diagnoses in patients with dizziness and headache[10, 13, 23]. The purpose of the review was not to perform a formal systematic meta-analysis, but to provide an evidence-based

clinical framework for interpretation of a symptomatic case. The case was analyzed according to four diagnostic domains: clinical symptom pattern, structural imaging, dynamic vascular testing and differential diagnosis. The anomaly was considered potentially symptomatic if the patient had occipital or cervicogenic headache, dizziness or imbalance provoked by head movements, topographic correlation between the bony bridge and the vertebral artery, and functional evidence of positional vertebral artery flow reduction. The anomaly was considered likely incidental if it was found without typical symptoms, without positional dependence, without vascular changes during functional testing, or in the presence of a more convincing alternative diagnosis.

Results. A 34-year-old male patient presented with recurrent occipital headache, episodic dizziness, tinnitus, transient gait unsteadiness and discomfort in the upper cervical region. The symptoms had been present for approximately eighteen months. The headache was predominantly localized in the right suboccipital and occipital regions, sometimes spreading to the parietal area. The patient described the pain as deep, pressing and occasionally pulsatile. Pain intensity varied from 4 to 7 points on a 10-point visual analogue scale. The patient reported that symptoms were provoked or intensified by prolonged static neck posture, sudden rotation of the head to the right, extension of the neck and driving for more than one hour. Dizziness was non-systemic in most episodes, but the patient sometimes described a short sensation of environmental instability during rapid head turning. Tinnitus was intermittent, more prominent on the right side, and sometimes coincided with headache exacerbation. There was no history of arterial hypertension, diabetes mellitus, transient ischemic attack, stroke, chronic otitis, vestibular neuritis, traumatic brain injury or migraine with aura. The patient denied typical photophobia, phonophobia and nausea associated with migraine attacks. There was no clear positional vertigo when turning in bed, and the duration of dizziness did not correspond to classic benign paroxysmal positional vertigo. The patient had previously received non-steroidal anti-inflammatory drugs and muscle relaxants with partial and temporary improvement. Several episodes were interpreted as cervical osteochondrosis, but standard treatment did not eliminate dizziness or tinnitus.

Neurological examination revealed preserved consciousness, normal speech and no meningeal signs. Cranial nerve examination was unremarkable. There was no spontaneous nystagmus in the primary gaze. Smooth pursuit was mildly uncomfortable during active cervical rotation, but ocular movements were full. Muscle strength in the upper and lower limbs was 5/5 on the Medical Research Council scale. Tendon reflexes were symmetrical and within physiological limits. No pathological pyramidal signs were detected. Sensory examination revealed mild subjective paresthesia in the right suboccipital region without dermatomal sensory loss. Finger-nose and heel-knee tests were performed accurately, but tandem gait was mildly unstable during headache exacerbation. Cervical examination showed moderate limitation of rotation to the right, tenderness of the suboccipital muscles and provocation of occipital pain during extension-rotation test. Brain MRI did not reveal acute ischemia, intracranial tumor, hydrocephalus, demyelinating lesions or Chiari malformation. Cervical MRI showed mild degenerative changes at C4–C5 and C5–C6 without clinically significant spinal canal stenosis, spinal cord compression or myelopathic signal changes. No disc herniation explaining the full symptom complex was detected. Multislice computed tomography of the craniovertebral junction demonstrated a complete right-sided osseous bridge over the groove of the vertebral artery on the

posterior arch of C1, consistent with complete Kimmerle's anomaly. Three-dimensional reconstruction clearly visualized conversion of the vertebral artery sulcus into an arcuate foramen. The left side showed no complete osseous bridge.

Computed tomography angiography demonstrated a dominant right vertebral artery entering the arcuate foramen at the level of C1. In neutral position, the lumen was preserved without critical stenosis. There was no evidence of arterial dissection, aneurysm, thrombosis, congenital absence of the vertebral artery or severe atherosclerotic lesion. The basilar artery was patent. Mild narrowing of the V3 segment was suspected at the level of the osseous canal, but static CTA alone was insufficient to determine functional significance. Therefore, duplex Doppler ultrasonography with functional rotational tests was performed. In neutral head position, vertebral artery flow velocities were within acceptable limits. During right head rotation and extension, a reproducible decrease in systolic and end-diastolic velocity in the right vertebral artery was recorded. The decrease was accompanied by reproduction of the patient's typical dizziness and occipital discomfort. The left vertebral artery showed compensatory flow increase without complete hemodynamic compensation. After return to neutral position, flow velocities improved and symptoms decreased. The combination of complete right-sided Kimmerle's anomaly, dominant right vertebral artery, symptom provocation during right rotation, and Doppler-confirmed positional flow reduction supported the diagnosis of symptomatic Kimmerle's anomaly.

The diagnostic summary is presented in Table 1.

Clinical and diagnostic domain	Patient-specific finding	Interpretation
Leading symptoms	Occipital headache, dizziness, tinnitus, gait unsteadiness	Cervico-vestibular syndrome with possible vertebrobasilar component
Triggering factor	Head rotation and extension, prolonged static cervical posture	Positional and biomechanical dependence
Neurological status	Mild tandem gait instability during exacerbation, no pyramidal deficit	Functional vestibulo-coordinative disturbance without myelopathy
Brain MRI	No stroke, tumor, hydrocephalus or Chiari malformation	Exclusion of major intracranial causes
Cervical MRI	Mild spondylotic changes without cord compression	Degenerative changes insufficient to explain symptoms
MSCT with 3D reconstruction	Complete right-sided arcuate foramen of C1	Structural confirmation of Kimmerle's anomaly
CTA	Dominant right vertebral artery passing through osseous canal	Topographic neurovascular relevance
Doppler with functional tests	Rotation-dependent reduction of right vertebral artery flow	Functional hemodynamic significance
Final interpretation	Symptomatic complete right-sided Kimmerle's anomaly	Clinically and dynamically verified diagnosis

The patient was managed conservatively at the initial stage. Recommendations included avoidance of extreme cervical rotation and extension, temporary limitation of manipulative cervical

procedures, individualized physical therapy focused on deep cervical flexor stabilization and suboccipital muscle relaxation, postural correction, vestibular rehabilitation elements, analgesic therapy during exacerbations and vascular-neurological monitoring. Surgical decompression was not selected at the primary stage because there were no ischemic strokes, drop attacks, progressive neurological deficits or critical persistent vertebral artery stenosis. However, the patient was informed that recurrence of severe vertebrobasilar symptoms, objective progression of flow compromise or failure of conservative therapy would require re-evaluation by a spine neurosurgeon or vascular neurosurgeon.

Differential Diagnosis. The main diagnostic problem in this case was the nonspecific nature of the patient's symptoms. Occipital headache, dizziness and tinnitus may occur in many neurological, otological, vascular and musculoskeletal disorders. Therefore, attributing symptoms to Kimmerle's anomaly requires a structured differential diagnostic approach.

Vestibular migraine was considered because the patient had recurrent dizziness and headache. However, the headache was predominantly occipital and mechanically provoked, typical migraine features were absent, and there was no convincing history of migraine attacks with photophobia, phonophobia or migraine aura. Moreover, functional Doppler testing reproduced the patient's symptoms during cervical rotation, which favored a cervico-vascular mechanism rather than a primary migraine mechanism [10, 12, 14]. Benign paroxysmal positional vertigo was also considered. Classic BPPV is characterized by brief attacks of positional vertigo associated with typical nystagmus during Dix-Hallpike or supine roll testing. In this patient, dizziness was more related to cervical rotation and extension than to changes of head position relative to gravity. There was no typical torsional upbeating nystagmus, and the symptom duration was not characteristic of posterior canal BPPV. Thus, BPPV was considered unlikely [11, 12, 15]. Atherosclerotic vertebrobasilar insufficiency was excluded because of the patient's young age, absence of major vascular risk factors, normal intracranial circulation on CTA and lack of diffuse atherosclerotic lesions. Vertebral artery dissection was excluded by CTA and absence of acute severe neck pain, neurological deficit or imaging signs of mural hematoma. Isolated cervical osteochondrosis was not considered sufficient because MRI showed only mild degenerative changes, whereas the most reproducible symptoms were linked to the C1-level anomaly and dynamic vertebral artery flow reduction. Craniovertebral instability was not supported by imaging and clinical data. Barre-Lieou syndrome was considered only as a historical descriptive concept, because the patient's symptoms could be explained more objectively by a combination of cervicogenic pain, periarterial sympathetic irritation and positional vertebral artery flow disturbance [5, 9, 13].

The differential diagnostic matrix is presented in Table 2.

Disorder	Typical diagnostic features	Findings in the present case	Diagnostic conclusion
Symptomatic Kimmerle's anomaly	Occipital headache, dizziness, tinnitus, symptom provocation by head rotation, C1 arcuate foramen, dynamic vertebral artery flow changes	Complete right-sided arcuate foramen, dominant right vertebral artery, symptom reproduction and Doppler	Most probable diagnosis

		flow reduction during rotation	
Cervicogenic headache	Unilateral or occipital pain related to cervical movement, reduced neck range of motion, cervical tenderness	Present: occipital pain, cervical trigger, suboccipital tenderness	Concomitant mechanism
Vestibular migraine	Recurrent vestibular episodes with migraine history, photophobia, phonophobia, migraine-type headache	Migraine features absent, mechanical trigger dominant	Unlikely
BPPV	Brief positional vertigo, typical nystagmus on Dix-Hallpike or roll test	No typical nystagmus, symptoms linked to neck rotation rather than gravity-dependent canalithiasis	Unlikely
Atherosclerotic vertebrobasilar insufficiency	Older age, vascular risk factors, CTA/MRA stenosis, posterior circulation ischemic symptoms	Young patient, no atherosclerotic stenosis	Excluded
Vertebral artery dissection	Acute neck pain/headache, trauma, mural hematoma or luminal irregularity	CTA negative, chronic recurrent course	Excluded
Cervical spondylosis	Disc degeneration, radiculopathy or myelopathy, foraminal stenosis	Mild degenerative changes without compression	Insufficient explanation
Craniovertebral instability	Abnormal C1–C2 motion, ligamentous instability, neurological symptoms	No instability on imaging	Excluded
Barre-Lieou syndrome	Posterior cervical sympathetic irritation, headache, dizziness, tinnitus	Some symptom overlap, but modern objective diagnosis favored Kimmerle-related mechanism	Historical overlap only

Discussion. The presented case illustrates the central problem of Kimmerle's anomaly: it is common enough to be incidental, yet anatomically capable of producing clinically relevant neurovascular symptoms in selected patients. The presence of an osseous bridge on C1 does not automatically explain headache or dizziness. At the same time, a complete arcuate foramen surrounding a dominant vertebral artery in a patient with reproducible rotation-dependent symptoms and Doppler-confirmed flow reduction cannot be ignored as a simple radiological variant. The diagnostic task is to distinguish incidental anatomy from symptomatic anatomy [1, 3, 5]. Several anatomical features increase the plausibility of clinical significance. A complete osseous canal may restrict the mobility of the V3 vertebral artery segment more than an incomplete bony bridge. Unilateral dominance of the vertebral artery may further reduce compensatory capacity when the dominant side

is affected. The relationship between the bony canal and the artery is also important: a wide arcuate foramen may be clinically silent, whereas a narrow canal or sharp osseous edge may irritate the artery, periarterial sympathetic plexus or suboccipital nerve. These anatomical and functional parameters cannot be fully assessed on plain radiography alone; they require CT-based three-dimensional evaluation and vascular imaging [2, 4, 8].

The symptom pattern in this case was consistent with a cervico-vestibular disorder. Occipital headache and suboccipital tenderness reflected the cervical component, while dizziness, tinnitus and gait unsteadiness suggested possible vertebrobasilar involvement. The strongest argument for symptomatic Kimmerle's anomaly was not the presence of headache or dizziness as isolated complaints, but the reproducibility of symptoms during head rotation and their temporal association with Doppler-confirmed reduction of vertebral artery flow. This dynamic relationship is essential, because static imaging may show the anomaly but cannot prove its hemodynamic significance [5, 9, 13]. From a clinical standpoint, Kimmerle-related headache may overlap with cervicogenic headache. Cervicogenic headache is typically associated with disorders of the cervical spine or soft tissues and may be provoked by neck movement or sustained posture. In the present case, the pain was occipital, movement-dependent and associated with suboccipital muscle tenderness. However, the additional presence of tinnitus, dizziness and dynamic vertebral artery flow reduction suggested that the clinical syndrome exceeded purely muscular or facetogenic cervicogenic headache. Therefore, the patient's condition was interpreted as a combined cervicogenic and vertebrobasilar irritation syndrome related to Kimmerle's anomaly [5, 10, 16].

The role of Doppler ultrasonography with functional tests deserves particular attention. Conventional Doppler testing in neutral position may be normal, especially in patients whose symptoms appear only during rotation or extension. Functional rotational testing can reveal positional reduction of vertebral artery flow and may reproduce symptoms. Nevertheless, this method must be interpreted carefully. Flow velocity changes may depend on technique, probe position, head angle, contralateral vertebral artery anatomy, vascular tone and patient cooperation. Therefore, a single abnormal value should not be overinterpreted. In the present case, the diagnostic significance was strengthened by reproducibility, symptom correlation and anatomical confirmation on MSCT and CTA. Computed tomography with three-dimensional reconstruction is the most informative method for morphological characterization of Kimmerle's anomaly. It allows precise differentiation between complete and incomplete forms, unilateral and bilateral variants, and the relationship of the osseous bridge to the vertebral artery groove. Multislice CT is particularly useful when surgical planning is considered, because it provides information about the size, thickness and spatial orientation of the bony bridge. CTA adds vascular information, including vertebral artery dominance, caliber, tortuosity and possible compression. MRI remains indispensable for excluding differential diagnoses such as Chiari malformation, demyelinating lesions, cervical myelopathy, disc herniation and posterior fossa pathology [1, 4, 8].

The differential diagnosis of dizziness in such patients is often difficult. Vestibular migraine is one of the most frequent causes of recurrent vestibular symptoms and may present with dizziness, headache, visual sensitivity and motion intolerance. BPPV is another common disorder and is diagnosed by typical positional vertigo and characteristic nystagmus during positional tests. Vertebral

artery dissection must be considered when there is acute neck pain, headache and posterior circulation symptoms, especially after trauma or cervical manipulation. Atherosclerotic vertebrobasilar insufficiency is more probable in older patients with vascular risk factors. The present case demonstrates that Kimmerle's anomaly should be considered only after these conditions are systematically evaluated [10, 11, 12]. Another practical aspect is the risk of excessive diagnostic simplification. Patients with headache and dizziness are often diagnosed with cervical osteochondrosis without precise anatomical or functional verification. Conversely, detection of Kimmerle's anomaly may lead to overdiagnosis if clinicians attribute all symptoms to the bony bridge. Both errors are undesirable. The correct approach is integrative. The diagnosis of symptomatic Kimmerle's anomaly should be based on concordance between clinical phenotype, anatomical imaging, vascular dynamics and exclusion of alternative disorders. This is the principal original concept of the present article.

The criteria distinguishing symptomatic and asymptomatic Kimmerle's anomaly are summarized in Table 3.

Criterion	Symptomatic Kimmerle's anomaly	Asymptomatic Kimmerle's anomaly
Headache pattern	Occipital or suboccipital, frequently provoked by rotation or extension	No headache or nonspecific unrelated headache
Vestibular symptoms	Dizziness, tinnitus, imbalance, sometimes visual symptoms	Absent
Relation to neck movement	Clear reproduction or worsening during head rotation or extension	No positional dependence
CT morphology	Often complete or anatomically narrow arcuate foramen	Complete or incomplete form may be incidental
Vertebral artery anatomy	Dominance, tortuosity or close contact may increase relevance	No significant topographic concern
Functional Doppler testing	Reproducible reduction of flow with symptoms	Normal or nonspecific changes
Differential diagnosis	Alternative causes excluded or insufficient	More probable alternative diagnosis present
Clinical conclusion	Potential neurovascular factor requiring monitoring or treatment	Anatomical variant requiring documentation only

Treatment strategy for symptomatic Kimmerle's anomaly remains individualized. Most patients without progressive neurological deficits, ischemic events or severe flow compromise can initially be managed conservatively. Conservative treatment may include avoidance of provocative head positions, postural training, stabilization exercises for the deep cervical muscles, correction of muscular imbalance, vestibular rehabilitation, analgesic therapy, and careful monitoring. Manual high-velocity cervical manipulation should be avoided or used with extreme caution when vertebral artery compromise is suspected. Surgical decompression of the vertebral artery has been described in selected cases with disabling symptoms, drop attacks, severe vertebrobasilar insufficiency or objective dynamic compression refractory to conservative therapy. However, surgery must be reserved for carefully selected patients after comprehensive anatomical and functional verification [5, 8, 9]. The limitations

of this article are related to its single-case design. A case report cannot establish population-level causality or define the prevalence of symptomatic Kimmerle's anomaly. The Doppler findings, although reproducible in this patient, require standardized protocols in larger cohorts. Furthermore, the relationship between symptoms and vertebral artery flow may be influenced by individual vascular anatomy, cervical muscle tone and autonomic regulation. Nevertheless, the strength of the case lies in the combination of clinical reproducibility, multimodal imaging, functional hemodynamic testing and structured differential diagnosis. This makes the observation useful for clinical practice and for further research.

Future studies should include prospective cohorts of patients with occipital headache and dizziness, standardized MSCT classification of Kimmerle's anomaly, CTA-based vertebral artery analysis, Doppler testing in neutral and provocative positions, validated headache and dizziness scales, and long-term follow-up after conservative or surgical treatment. Such studies would help determine which anatomical and functional parameters predict symptomatic disease and which patients require only observation.

Conclusion. Kimmerle's anomaly is not always clinically significant, but it may become an important neurovascular factor in selected patients with cervicogenic occipital headache, dizziness, tinnitus and vestibulo-coordinative disorders. The presented clinical case demonstrates that the anomaly should not be assessed as an isolated radiological finding. Its clinical relevance depends on the concordance between symptoms, head-position dependence, C1 osseous anatomy, vertebral artery topography and functional vascular changes. The most informative diagnostic pathway includes neurological examination, MRI of the brain and cervical spine, MSCT of the craniovertebral junction with three-dimensional reconstruction, CTA of the vertebral arteries, and duplex Doppler ultrasonography with rotational tests. Such an algorithm makes it possible to differentiate symptomatic Kimmerle's anomaly from vestibular migraine, BPPV, atherosclerotic vertebrobasilar insufficiency, vertebral artery dissection, cervical spondylosis, craniovertebral instability and nonspecific cervical pain syndromes. The main practical conclusion is that a patient with occipital headache, dizziness, tinnitus and symptom exacerbation during head rotation should not be automatically diagnosed with cervical osteochondrosis or functional dizziness. If Kimmerle's anomaly is detected, it must be evaluated dynamically and clinically. Early recognition of symptomatic forms allows rational conservative management, prevention of excessive cervical manipulation, timely vascular monitoring and, in selected severe cases, consideration of surgical decompression.

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