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KIMMERLE ANOMALY AS A DYNAMIC CRANIOCERVICAL NEUROVASCULAR COMPRESSION SYNDROME: AN INTEGRATIVE REVIEW AND PROPOSED DIAGNOSTIC–THERAPEUTIC FRAMEWORK

Abstract

Background. Kimmerle anomaly, also termed ponticulus posticus or arcuate foramen, is an osseous bridge over the vertebral artery groove of the atlas. Although usually incidental, it may become clinically relevant when the V3 segment of the vertebral artery, periarterial sympathetic plexus, venous plexus, or C1–C2 neural structures are constrained during head rotation.

Materials and methods. A structured literature synthesis was conducted using biomedical publications, emphasizing systematic reviews, meta-analyses, computed-tomography studies, surgical reports published through July 2026. Evidence was organized according to morphology, dynamic vascular behavior, symptom reproducibility, collateral circulation, and treatment response.

Results. Reported prevalence varies because of population differences and imaging methodology; modern CT-based analyses identify ponticulus posticus as a common anatomical variant. In practice, morphology alone does not establish causality. The strongest evidence of clinical significance is concordance between a compatible posterior-circulation or suboccipital syndrome, reproducible positional symptoms, structural narrowing around the V3 segment, and demonstrable flow reduction during provocative testing. Thin-slice CT defines osseous anatomy, CT angiography or MR angiography evaluates vascular relationships, duplex or transcranial Doppler provides dynamic screening, and rotational digital subtraction angiography remains the reference test when necessary.

Conclusion. Clinically significant Kimmerle anomaly should be regarded as a dynamic craniocervical neurovascular compression syndrome. Conservative management is appropriate for most patients, whereas microsurgical decompression or stabilization should be reserved for hemodynamically verified, refractory cases.

Keywords: Kimmerle anomaly; ponticulus posticus; arcuate foramen; vertebral artery; V3 segment; rotational vertebral artery syndrome; Bow Hunter syndrome; CT angiography; dynamic ultrasonography; craniovertebral junction.

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АНОМАЛИЯ КИММЕРЛИ КАК ДИНАМИЧЕСКИЙ КРАНИОЦЕРВИКАЛЬНЫЙ НЕЙРОВАСКУЛЯРНЫЙ КОМПРЕССИОННЫЙ СИНДРОМ: ИНТЕГРАТИВНЫЙ ОБЗОР И ПРЕДЛОЖЕННАЯ ДИАГНОСТИКО-ТЕРАПЕВТИЧЕСКАЯ МОДЕЛЬ

Аннотация

Актуальность. Аномалия Киммерли, также обозначаемая как задний мостик атланта, ponticulus posticus или дугообразное отверстие, представляет собой костную перемычку над бороздой позвоночной артерии на задней дуге C1. В большинстве случаев она является

случайной анатомической находкой, однако при ограничении сегмента V3 позвоночной артерии, периартериального симпатического сплетения, венозного русла или нервных структур C1–C2 может приобретать клиническое значение, особенно при поворотах головы.

Материалы и методы. Выполнен структурированный анализ биомедицинских публикаций с приоритетом систематических обзоров, метаанализов, компьютерно-томографических исследований, работ по динамической визуализации сосудов и хирургических серий, опубликованных до июля 2026 года. Данные систематизированы с учётом морфологии костного мостика, динамики кровотока, воспроизводимости позиционных симптомов, состояния коллатерального кровообращения и ответа на лечение.

Результаты. Частота выявления существенно зависит от популяции и метода визуализации; современные КТ-исследования подтверждают, что ponticulus posticus является распространённым анатомическим вариантом. Сама морфология не доказывает причинную связь с жалобами. Наиболее убедительным критерием симптомности служит совпадение клиники задней циркуляции или субокципитальной боли с позиционной провокацией, структурным сужением вокруг V3 и объективным снижением кровотока. Тонкосрезовая КТ определяет костную анатомию, КТ-ангиография и МР-ангиография оценивают сосудистые взаимоотношения, ультразвуковые ротационные пробы используются для скрининга, а динамическая субтракционная ангиография остаётся референсным методом. Предложенный алгоритм позволяет уменьшить гипердиагностику, отделить случайную костную находку от функционально значимого сосудисто-неврального конфликта и обосновать выбор терапии.

Заключение. Симптомную аномалию Киммерли следует рассматривать как динамический краниоцервикальный нейроваскулярный компрессионный синдром. Большинству пациентов показана консервативная тактика; операция оправдана при доказанной гемодинамической значимости и рефрактерных симптомах.

Ключевые слова: аномалия Киммерли; задний мостик атланта; дугообразное отверстие; позвоночная артерия; сегмент V3; ротационный синдром позвоночной артерии; синдром лучника; КТ-ангиография; динамическая ультрасонография; краниовертебральный переход.

KIMMERLI ANOMALIYASI DINAMIK KRANIOTSERVIKAL NEYROVASKULYAR KOMPRESSIV SINDROM SIFATIDA: INTEGRATIV SHARH VA TAKLIF ETILGAN DIAGNOSTIK-DAVOLASH MODELI

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Annotatsiya

Dolzarblik. Kimmerli anomaliyasi, shuningdek atlasning orqa ko‘prikchasi, ponticulus posticus yoki arcuate foramen deb atalib, C1 umurtqa orqa yoyidagi umurtqa arteriyasi egati ustida hosil bo‘ladigan suyak ko‘prikchasidir. Ko‘pchilik holatlarda u tasodifiy anatomik topilma hisoblanadi, biroq V3 segment, periarterial simpatik chigal, venoz pleksus yoki C1–C2 nerv tuzilmalari cheklanganda, ayniqsa boshni burish vaqtida klinik ahamiyat kasb etishi mumkin. Maqsad. Kimmerli

anomaliyasining anatomiyasi, tarqalishi, patogenezi, klinik fenotiplari, diagnostikasi, differensial diagnostikasi va davolashiga oid zamonaviy ma’lumotlarni umumlashtirish hamda “anatomiya–gemodinamika–simptom” integrativ modelini taklif etish.

Material va usullar. 2026-yil iyuligacha e’lon qilingan biotibbiy manbalar, jumladan tizimli sharhlar, meta-tahlillar, kompyuter tomografik tadqiqotlar, dinamik tomir vizualizatsiyasi ishlari va jarrohlik kuzatuvlari strukturaviy ravishda tahlil qilindi. Ma’lumotlar suyak ko‘prikchasining morfologiyasi, qon oqimining dinamikasi, pozitsion simptomlarning qayta chaqirilishi, kollateral qon aylanishi va davolashga javob asosida tizimlashtirildi.

Natijalar. Aniqlanish chastotasi populyatsiya va tasvirlash usuliga bog‘liq; zamonaviy KT tadqiqotlari ponticulus posticus keng tarqalgan anatomik variant ekanini ko‘rsatadi. Morfologiyaning o‘zi simptomlar bilan sababiy bog‘liqlikni isbotlamaydi. Klinik ahamiyatning eng ishonchli belgisi orqa qon aylanishi simptomlari yoki suboksipital og‘riqning bosh holati bilan qayta chaqirilishi, V3 atrofidagi struktur torayish va provokatsion tekshiruvda qon oqimining objektiv pasayishi bilan mos kelishidir. Yupqa kesimli KT suyak anatomiyasini, KT-angiografiya va MR-angiografiya tomir munosabatlarini baholaydi, ultratovushli rotatsion sinamalar skrining uchun qo‘llanadi, dinamik raqamli subtraksion angiografiya esa referens usul bo‘lib qoladi.

Xulosa. Simptomatik Kimmerli anomaliyasi dinamik kraniotservikal neyrovaskulyar kompression sindrom sifatida baholanishi kerak. Ko‘pchilik bemorlarda konservativ taktika yetarli, jarrohlik esa gemodinamik ahamiyat isbotlangan va refrakter simptomli holatlarga ajratiladi.

Kalit so‘zlar: Kimmerli anomaliyasi; ponticulus posticus; arcuate foramen; umurtqa arteriyasi; V3 segment; rotatsion umurtqa arteriyasi sindromi; Bow Hunter sindromi; KT-angiografiya; dinamik ultratovush; kraniovertebral soha.

Introduction. Kimmerle anomaly is an osseous variation of the atlas in which a partial or complete bony bridge forms over the groove occupied by the third, suboccipital segment of the vertebral artery. In contemporary anatomical terminology, the structure is most often called ponticulus posticus; arcuate foramen, foramen arcuale, posterior ponticle, pons posticus, retroarticular vertebral artery ring, and Kimmerle anomaly are established synonyms. The variation converts an open arterial sulcus into a partly constrained groove or a complete osteofibrous canal. Depending on its morphology, this canal may contain the V3 segment of the vertebral artery, accompanying veins, the periarterial sympathetic plexus, and structures related to the first cervical nerve. This neurovascular proximity explains why a small osseous bridge can have important clinical and surgical consequences [1, 2, 3]. The atlas lies within the highly mobile atlanto-occipital and atlantoaxial complex. After leaving the C1 transverse foramen, the vertebral artery turns posteromedially across the superior surface of the posterior arch before entering the dura. Rotation and extension change the length, curvature, and contact pressure of the V3 segment. In most individuals these changes are accommodated by arterial mobility, contralateral vertebral flow, and posterior communicating arteries. If the vessel is tethered within an arcuate foramen, movement may cause focal stenosis, disturbed flow, repetitive adventitial irritation, or transient occlusion. Clinical expression ranges from suboccipital pain and cervicogenic dizziness to rotational vertebrobasilar insufficiency, transient ischemic attacks, or rarely posterior-circulation stroke [3, 6, 10]. The main diagnostic problem is attribution rather than detection. Ponticulus posticus is common in asymptomatic populations, and prevalence varies according to

imaging method, population, and the definition of incomplete ossification. Large meta-analyses and CT cohorts confirm that it is not a rare malformation. Therefore, an image alone cannot establish the cause of headache, vertigo, tinnitus, visual disturbance, disequilibrium, or syncope. Conversely, static neutral-position imaging may miss a dynamic lesion. Thin-slice CT defines bone accurately, but it cannot prove functional arterial compromise; a normal resting Doppler study or neutral CT angiogram does not exclude rotational compression [4, 7, 14].

A clinically meaningful diagnosis requires concordance among four elements: a compatible posterior-circulation or suboccipital syndrome, reproducibility in a specific head position, an anatomical bridge capable of restricting the V3 segment or adjacent neural structures, and objective dynamic dysfunction. Collateral circulation and alternative diagnoses must also be assessed. Pathogenesis may be hemodynamic, arterial-wall related, neurogenic, nociceptive, or mixed. A complete ring may limit arterial excursion, whereas an incomplete spur may act as a focal contact point. Repetitive trauma may theoretically promote dissection or thrombosis, while irritation of the C1 nerve, suboccipital nerve, or sympathetic plexus may produce pain without measurable ischemia [11, 12, 20]. The anomaly also has independent surgical relevance. During C1 lateral mass, pedicle, or posterior-arch screw placement, a complete bridge may falsely appear to be a thick posterior arch and may conceal the vertebral artery. Recent CT angiography and morphometric studies emphasize individualized bone–vessel mapping before craniovertebral junction fixation [5, 11, 16]. This article integrates anatomy, dynamic hemodynamics, clinical phenotype, collateral reserve, and treatment response into a practical framework for distinguishing incidental ponticulus posticus from clinically significant Kimmerle anomaly.

Materials and Methods. The manuscript was developed as a structured integrative review rather than a formal systematic review or meta-analysis. Literature concerning Kimmerle anomaly, ponticulus posticus, arcuate foramen, V3 vertebral artery compression, rotational vertebral artery syndrome, Bow Hunter syndrome, C1 instrumentation, and craniovertebral vascular anatomy was examined. Priority was given to systematic reviews, meta-analyses, large CT or cone-beam CT cohorts, three-dimensional CT angiography studies, dynamic Doppler or angiographic investigations, and surgical reports available through July 2026. Historically important earlier publications were retained when they established terminology, prevalence, clinical associations, or surgical risk [2, 3, 10, 18]. Evidence was organized into five domains: morphology; vascular behavior in neutral and provocative positions; clinical phenotype; collateral reserve; and response to conservative or surgical treatment. Morphology included complete and incomplete bridges, laterality, bilateral forms, and combined ponticulus posticus–ponticulus lateralis. Vascular analysis included luminal caliber, arterial dominance, waveform changes, and positional patency. Clinical analysis included vertigo, presyncope, syncope, diplopia, oscillopsia, tinnitus, ataxia, posterior headache, occipital neuralgia, and ischemic events.

Because the evidence is heterogeneous and dominated by observational studies, case reports, and small surgical series, no pooled therapeutic effect was calculated. Cross-sectional associations with headache or migraine were interpreted cautiously. A clinically persuasive relationship was defined by simultaneous structural plausibility, symptom concordance, positional reproducibility, and objective

hemodynamic or neural evidence. The phenotype matrices and diagnostic sequence presented below are an original synthesis of published evidence and do not represent a new patient cohort.

Results. Ponticulus posticus arises from the superior surface of the posterior arch of C1 and bridges toward the posterior portion of the superior articular mass. The complete form creates a true arcuate foramen; the incomplete form produces a partial spur or arch. Unilateral, bilateral, complete, incomplete, and mixed configurations occur. Some patients also have ponticulus lateralis, creating an additional lateral site of restriction. CT classifications that describe each side as absent, incomplete, or complete are more informative than a simple present-or-absent label [4, 7, 14, 19]. Its origin remains debated. Traditional explanations emphasize ossification of the posterior atlanto-occipital membrane or related ligamentous structures. The presence of complete bridges in young individuals, bilateral symmetry, ethnic variation, and associated atlas anomalies suggests a developmental or genetic component. Age-related progression in some cohorts indicates that mechanical loading and ligamentous mineralization may modify expression. A combined developmental–biomechanical model is therefore reasonable [2, 3, 18].

Table 1. Morphological Classification and Potential Clinical Significance of Kimmerle Anomaly

Morphological category	Imaging definition	Potential functional consequence	Main surgical implication
Incomplete ponticulus posticus	Partial spur over the V3 groove	Focal contact during rotation; possible neural irritation	May be underestimated without multiplanar CT
Complete ponticulus posticus	Closed osseous canal over the groove	Restricted arterial excursion and possible rotational stenosis	Can be mistaken for a safe, thick posterior arch
Bilateral form	Complete or incomplete bridge on both sides	Bilateral restriction modified by dominance and collateral flow	Requires bilateral vascular mapping
Mixed form	Complete bridge on one side, incomplete on the other	Asymmetric positional hemodynamics	Screw trajectory must be individualized
Associated ponticulus lateralis	Posterior and lateral osseous bridges	Multilevel or circumferential tethering	Decompression may require broader exposure

Prevalence estimates vary widely because plain radiographs detect complete rings more reliably than small incomplete bridges, whereas CT and cone-beam CT identify subtle ossification. Earlier meta-analysis estimated overall prevalence near 16.7%, with complete forms around 9.3%. A larger meta-analysis of 55,985 subjects found complete foramen arcuale in approximately 9% and incomplete forms in approximately 14%. A CT study of 2,917 patients reported ponticulus posticus in 22.5%. Recent CT-focused syntheses continue to support a combined prevalence near one fifth, although geographic, sex, and laterality differences remain inconsistent [15, 17, 27]. The frequency of the osseous finding is much higher than the frequency of symptomatic disease. Morphology should consequently be considered a risk substrate, not a diagnosis. A wide complete canal may be clinically

silent, whereas a small incomplete bridge may be important if its edge compresses a dominant artery during ordinary movement. Functional significance depends on bridge geometry, arterial diameter and mobility, head position, contralateral vertebral capacity, intracranial collaterals, and neural sensitivity [3, 10, 18, 27].

Four overlapping phenotypes can be distinguished. In the dynamic hemodynamic phenotype, rotation or extension reversibly narrows the V3 segment. Symptoms start within seconds of the provocative position and improve after returning to neutral. Positional vertigo, diplopia, oscillopsia, gait unsteadiness, dysarthria, presyncope, or syncope are more persuasive than isolated nonspecific headache. Risk increases when the affected artery is dominant, the contralateral artery is hypoplastic or occluded, or posterior communicating arteries are inadequate [4, 14, 19, 25]. In the arterial-wall injury phenotype, repetitive contact may damage the adventitia or endothelium and promote dissection, mural thrombus, distal embolization, or fixed stenosis. Evidence specific to Kimmerle anomaly is limited, but rotational vertebral artery syndromes associated with C1 abnormalities and reported stroke cases support biological plausibility. Symptoms may persist outside the provocative position once ischemia or thromboembolism has occurred [2, 6, 10, 13].

The neurogenic–nociceptive phenotype reflects irritation of the C1 nerve, suboccipital nerve, periarterial sympathetic fibers, or upper-cervical soft tissues. Patients may present with occipital pain, cervicogenic headache, neck stiffness, local tenderness, or tinnitus without demonstrable hypoperfusion. Studies have reported associations with headache and migraine, but referral bias, symptom overlap, and cross-sectional designs limit causal interpretation. The mixed phenotype combines positional dizziness or visual symptoms with suboccipital pain and cervical muscle spasm [11, 15, 17, 20].

Table 2. Proposed Clinical Phenotypes of Symptomatic Kimmerle Anomaly

Proposed phenotype	Dominant mechanism	Typical presentation	Objective support	Main pitfall
Dynamic hemodynamic	Reversible V3 stenosis or occlusion	Positional vertigo, diplopia, ataxia, presyncope, syncope	Dynamic ultrasound, CTA, MRA, or DSA	Diagnosing vascular disease from nonspecific dizziness
Arterial-wall injury	Dissection, thrombosis, or embolism after repetitive trauma	Posterior-circulation TIA or stroke	Diffusion-positive lesion, vessel-wall abnormality, thrombus	Expecting all symptoms to disappear in neutral position
Neurogenic–nociceptive	C1/suboccipital neural or myofascial irritation	Occipital pain, cervicogenic headache, local tenderness	Pain reproduction, blocks, absence of ischemic evidence	Attributing primary migraine to an incidental bridge
Mixed neurovascular	Combined arterial restriction and neural irritation	Pain with positional dizziness,	Concordant structural and dynamic findings	Treating pain while overlooking flow compromise

		tinnitus, or visual disturbance		

History should define the exact movement, direction and angle of rotation, latency, reproducibility, duration, and recovery in neutral position. Collapse, focal deficit, diplopia, dysarthria, limb ataxia, or transient visual disturbance requires urgent vascular evaluation. Examination should include cranial nerves, nystagmus, cerebellar function, gait, long-tract signs, cervical range of motion, occipital tenderness, and bilateral arm blood pressure. Provocative testing must stop immediately if neurological symptoms appear and should be avoided when dissection or recent ischemia is suspected [4, 7, 14]. Differential diagnosis includes benign paroxysmal positional vertigo, vestibular migraine, Ménière disease, persistent postural-perceptual dizziness, orthostatic hypotension, arrhythmia, subclavian steal syndrome, atherosclerotic vertebral stenosis, vertebral dissection, Chiari malformation, craniovertebral instability, cervical spondylosis, cervicogenic dizziness, occipital neuralgia, primary headache disorders, demyelinating disease, and posterior fossa lesions. Symptomatic Kimmerle anomaly becomes more credible when these alternatives are excluded and when symptoms are both position-specific and anatomically concordant [7, 14, 19].

Plain lateral radiography may reveal a complete arcuate foramen but lacks sensitivity for incomplete forms. Thin-slice CT with axial, sagittal, coronal, and three-dimensional reconstructions is the preferred technique for defining bridge completeness, laterality, thickness, canal size, associated atlas anomalies, and dimensions relevant to instrumentation. Cone-beam CT may detect the anomaly incidentally in orthodontic imaging, but it does not replace dedicated neurovascular assessment [3, 6, 15, 18]. CT angiography evaluates luminal caliber, vertebral dominance, contralateral hypoplasia, anomalous entry, fenestration, persistent first intersegmental artery, and the relationship of V3 to the bony canal. MR angiography avoids radiation and can be combined with diffusion-weighted brain imaging and vessel-wall sequences. Nevertheless, neutral CTA or MRA may be normal in a dynamic syndrome. Carefully selected rotational CTA or MRA can demonstrate positional narrowing, but movement must be controlled to minimize ischemic risk and artifact [6, 11, 12, 20]. Duplex ultrasonography and transcranial Doppler provide noninvasive dynamic screening. Gradual rotation may reveal reduced end-diastolic velocity, waveform dampening, marked reduction in mean velocity, or flow cessation. Recent studies of rotational vertebral artery syndrome support quantified ultrasound assessment, but results remain operator-dependent and the V3 segment may be difficult to insonate. A negative study cannot definitively exclude dynamic compression [1, 3, 10, 13]. Rotational digital subtraction angiography remains the reference examination when severe symptoms persist, noninvasive tests are discordant, or surgery is planned. It can show the precise level of stenosis, delayed filling, collateral compensation, and restoration of patency in neutral position. Because the procedure is invasive and provocative, it should use the minimum rotation needed to reproduce the clinically relevant position in an experienced center [6, 13, 28].

Table 3. Proposed Diagnostic Algorithm for Clinically Significant Kimmerle Anomaly

Diagnostic stage	Preferred investigation	Main question	Clinical interpretation

Clinical triage	Positional history and neurological examination	Are symptoms compatible and reproducible?	Nonpositional symptoms reduce causal probability
Osseous definition	Thin-slice CT with 3D reconstruction	What is the morphology and laterality?	Establishes substrate, not causality
Vascular mapping	CTA or MRA	Which artery is dominant and are collaterals adequate?	Identifies high-risk anatomy and competing disease
Dynamic screening	Duplex or transcranial Doppler	Does flow fall in the provoking position?	Concordant changes justify escalation
Definitive confirmation	Rotational DSA or selected dynamic CTA/MRA	Is focal C1-related stenosis reproducible?	Strongest evidence of functional compression
Surgical planning	High-resolution bone–vessel CTA fusion	How can decompression or fixation avoid the artery?	Essential before complex C1 surgery

Treatment. Incidentally discovered ponticulus posticus requires no treatment. Patients should be informed that the bridge is common and usually benign. Mild nonischemic symptoms without objective dynamic flow compromise are managed conservatively through avoidance of extreme repetitive rotation, correction of provocative posture, appropriate analgesia, treatment of coexisting migraine or vestibular disorders, and supervised rehabilitation. High-velocity upper-cervical manipulation should be avoided when vascular compromise has not been excluded [1, 3, 8]. Medication should target the established disorder rather than the bony finding. Antiplatelet therapy may be indicated after transient ischemic attack, dissection, or ischemic stroke according to cerebrovascular standards, but is not routinely justified for an asymptomatic bridge. A collar may temporarily limit provocative motion, although prolonged immobilization can cause stiffness and deconditioning. Surgery is reserved for disabling or dangerous symptoms, failure of appropriate conservative management, and convincing clinicoradiological concordance. Microsurgical or minimally invasive resection of the bridge releases the V3 segment while preserving motion. Case reports and small series describe improvement, but evidence is limited by selection bias and inconsistent outcomes. Risks include vertebral artery injury, venous bleeding, neural injury, instability, scar-related retethering, and persistent symptoms [15, 17, 27]. Fusion may be preferable when rotational occlusion is caused primarily by atlantoaxial instability or pathological translation. Stabilization removes the provocative motion but sacrifices cervical rotation and introduces instrumentation risk. Endovascular treatment has a limited role because the lesion is usually external and dynamic. Management should be individualized by a team involving neurology, neurosurgery or spine surgery, neuroradiology, vascular imaging, and rehabilitation specialists.

Table 4. Proposed Treatment Selection Matrix

Clinical scenario	Preferred strategy	Rationale	Evidence limitation

Incidental bridge	Observation and documentation	High prevalence and usually benign course	Limited natural-history data
Nonspecific symptoms without dynamic findings	Treat alternative diagnoses	Morphology alone does not prove causality	Rare vascular cases may initially be subtle
Reproducible symptoms with noninvasive flow reduction	Activity modification and specialist vascular work-up	Symptom–hemodynamic concordance suggests functional significance	Ultrasound is operator-dependent
Refractory V3 compression without instability	Surgical decompression	Removes the osseous constraint and preserves motion	Small series and case reports
Compression with C1–C2 instability	Stabilization with individualized vascular planning	Eliminates pathological motion	Reduced rotation after fusion
Ischemia with dissection or thromboembolism	Standard cerebrovascular treatment plus mechanical evaluation	Addresses immediate and recurrent risk	Timing of definitive surgery is not standardized

Discussion. The principal conclusion is that Kimmerle anomaly should not be treated as a binary radiographic diagnosis. It is an anatomical substrate whose relevance emerges only when it produces a reproducible neurovascular conflict. This view reconciles the high prevalence of ponticulus posticus in asymptomatic cohorts with well-documented dynamic vertebral artery compression. The bridge is common, symptomatic compression is uncommon, and severe ischemic complications are rare [4, 7, 14, 19]. The proposed anatomy–hemodynamics–symptom framework improves diagnostic discipline. Anatomy establishes plausibility, hemodynamics establishes functional effect, and symptoms establish clinical expression. No single domain is sufficient. A complete ring may be silent because its canal is wide or collateral flow is robust. An incomplete spur may be symptomatic because its free edge impinges on a dominant artery. A Doppler change at an extreme position never used in daily life may have little relevance, whereas neurological symptoms at moderate rotation are important even if neutral imaging is normal. Confidence rises when an ordinary head position reproduces both symptoms and objective flow reduction at the expected C1 site [6, 13, 28]. Bow Hunter syndrome should be distinguished from Kimmerle anomaly. Bow Hunter syndrome describes rotational vertebral artery compromise and may result from osteophytes, disc disease, tumors, fibrous bands, instability, arterial variants, or C1 bridges. Kimmerle anomaly is one structural cause. The distinction determines treatment: isolated bridge compression may be decompressed, atlantoaxial instability may require fusion, and subaxial degenerative compression may require a different approach [5, 11, 16].

Headache data require caution. Meta-analyses and cross-sectional studies suggest associations with headache or migraine in selected populations, but classification, imaging, and referral methods vary. Migraine and cervical myofascial pain are common, and many studies lack dynamic vascular assessment. A vascular mechanism is more plausible when occipital headache is position-dependent

and accompanied by posterior-circulation symptoms. A neural mechanism is more likely when focal tenderness or occipital neuralgia occurs without ischemic evidence [8, 9, 25]. Dynamic ultrasound may become the preferred screening bridge between static imaging and invasive angiography, provided protocols are standardized. Reports should document the insonation site, rotation direction and angle, baseline asymmetry, symptoms, and contralateral artery. The most useful finding is not an isolated numerical threshold but a reproducible, symptom-concordant, side-specific waveform change. Advanced CTA, MRA, vessel-wall MRI, and bone–vessel fusion may further characterize arterial tethering, dissection, and surgical anatomy [6, 11, 12, 20]. Surgical evidence demonstrates feasibility but not broad indications. Improvement after decompression has been reported in highly selected patients with rotational dizziness, syncope, or documented V3 compression, yet publication bias and inconsistent outcomes limit generalization. Prospective registries should record bridge dimensions, arterial dominance, provocative angle, dynamic flow, neurological reproduction, operative technique, arterial patency, complications, quality of life, and long-term recurrence [2, 6, 18, 27]. The anomaly has unequivocal importance before C1 instrumentation even when asymptomatic. A complete bridge can conceal the vertebral artery and alter standard landmarks. Preoperative evaluation should include atlas dimensions, ponticulus morphology, high-riding or anomalous vertebral arteries, persistent first intersegmental artery, fenestration, and unusual posterior inferior cerebellar artery origin. Navigation may improve execution, but it cannot replace individualized vascular analysis [4, 6, 14].

This synthesis is limited by inconsistent terminology, heterogeneous definitions of incomplete ossification, retrospective symptom studies, nonstandardized dynamic testing, and small surgical cohorts. The proposed framework is therefore a clinical reasoning model, not a validated score. Future multicenter research should standardize morphology, arterial dominance, collateral anatomy, provocative angle, flow change, symptom latency, recovery time, and patient-reported outcomes.

Conclusion. Kimmerle anomaly is a common atlas variation with uncommon but potentially important neurovascular consequences. Its presence on radiography or CT should not automatically be interpreted as the cause of headache, dizziness, tinnitus, or syncope. Clinically significant disease requires concordance among a compatible phenotype, reproducible positional symptoms, anatomically plausible restriction of the V3 vertebral artery or adjacent neural structures, and objective dynamic dysfunction. Thin-slice CT defines bone, CTA or MRA characterizes vascular anatomy and collateral reserve, dynamic ultrasound provides noninvasive screening, and rotational digital subtraction angiography remains the reference test when definitive confirmation is necessary. The original contribution of this article is an anatomy–hemodynamics–symptom framework with phenotype-based diagnostic and treatment matrices. The model separates an incidental bridge from a functional craniocervical compression syndrome and reduces both overdiagnosis and underrecognition. Conservative care is appropriate for most patients. Decompression is justified only when symptoms are disabling or dangerous, conservative treatment has failed, and dynamic vascular or neural conflict is convincingly demonstrated. Fusion is reserved for instability-driven compression. Recognition of ponticulus posticus is mandatory before C1 instrumentation because it can conceal the vertebral artery and distort surgical landmarks.

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