KIMMERLE ANOMALY

Mukhammadov Nuriddin Askarovich

Samarkand State Medical University, Department of Neurosurgery

Abstract: *The Kimmerle anomaly (KA) is a congenital or acquired osseous bridge at the craniovertebral junction, forming a bony canal that can compress the vertebral artery (VA), occipital nerve, and venous plexus. This review summarizes current literature on the epidemiology, pathogenesis, clinical manifestations, and diagnostic approaches of KA based on data from PubMed and eLibrary. The anomaly is found in 16–19% of the population and can manifest as vertebrobasilar insufficiency (VBI), cervicogenic headaches, and cochleovestibular disorders, especially after cervical spine strain or trauma. Diagnosis relies on radiography, MSCT, and MSCT angiography, while MRI and Doppler ultrasound are used to assess hemodynamic significance. Differential diagnosis includes atherosclerotic stenosis, vertebral artery tortuosity, and vestibular disorders. Understanding the anatomical and clinical characteristics of KA is critical for timely recognition and prevention of vascular and neurological complications.*

Key words: *Kimmerle anomaly, vertebral artery, vertebrobasilar insufficiency, craniovertebral junction, MSCT angiography, compression syndrome.*

Introduction. In 1923, H. Hayek described an anomaly of the craniovertebral junction, which is a bony bridge [1]. It is localized between the posterior edge of the articular process of C1 and the posterior border of its arch and forms an opening through which the vertebral artery (VA) and occipital nerve pass [2]. In 1930, A. Kimmerle (Hungary) described this anomaly in detail, drawing attention to the fact that this change can lead to vertebrobasilar insufficiency (VBI), vertebral pain syndrome and acute cerebrovascular accident (ACE). Subsequently, it was called Kimmerle anomaly (KA) [3]. Then, H. Krayenbuhl and MG Yasargil, while studying radiographs of the cervical spine (CS), discovered this pathology in patients with vertebrobasilar insufficiency (VBI) [4]. AC is an exostosis of the atlanto-occipital membrane located in the area of the craniovertebral junction, which forms

a bony bridge between the articular process of C1 and its posterior arch (posterior or medial bridge) and/or the articular and transverse process of C1 (lateral bridge) [5]. In AC, the V3 segment of the VA is compressed in the bony canal instead of its usual location in the sulcus arteriae vertebralis. The vertebral vein, posterior branch of the C1 rootlet and sympathetic endings may also be included in the bony canal. AC has several synonyms in the scientific literature: foramen arcuate, foramen arcuate atlantis, foramen retroarticulare, perior, ponticulus posterior et ponticulus lateralis atlantis, ponticuli posticus, canalis Bildungidunge; in an incomplete variant of the structure - clinoid bridge [6]. In ICD-10 it is classified as: Q76.4, I65.0, M47.0, M53.0, M85.0, G45.0, G99.2.

Objective: To analyze modern scientific literature on the epidemiology, pathogenesis, diagnosis of AK, presented in the eLibrary and PubMed libraries.

Epidemiology. Along with Arnold-Chiari malformation, platybasia, basilar impression, C1 assimilation, hypoplasia and aplasia of the posterior arch of C1, AC is one of the most common anomalies of the craniovertebral junction. AC occurs in 18.8% of cases according to sectional findings, 17.2% when analyzing MSCT, 16.6% according to spondylography [7]. The disease occurs equally in both men and women (15.8% and 14.6%, respectively) [8]. In a study of 305 cases of congenital AC, according to N.A. Shchikunov et al. (2014), bilateral AC was present in 42% of cases. A completely symmetrically formed canal was observed in 29% of cases, and an incomplete one – in 13%. In 19% of cases, bilateral asymmetric AC was detected, and unilateral AC - in 39%. Of the unilateral ones, left-sided was noted (30%), less often - right-sided (9%). In the case of left-sided PA canal, it was incomplete in 16% of cases, complete - in 14% [9]. The degree of ossification of the PA groove in AC is presented in Table 1. The clinical picture can manifest itself with ossification of the ligament of classes III and IV. PATHOGENESIS The pathogenesis of VBN in AC remains complex [10]. PA never occupies the entire volume of the foramen (usually about 35% of its volume). The fibrous tissue surrounding the PA, its venous and sympathetic plexus, and in the region of the posterior arch C1 and the occipital nerve, serve as a framework for them [11]. AC acquires clinical significance due to

the loss of elasticity of the VA affected by atherosclerosis or vasculitis, periarterial cicatricial process, the degree of ossification of the ligament, the presence of other anomalies of the craniovertebral region, the discrepancy between the width of the VA and the size of the opening in the bone canal, the presence of osteochondrosis of the CS, etc. [12]. In addition to the mechanical impact on the VA in AC, the ligament also reduces the mobility of the V3 segment of the VA during VA movement with the development of its compression [13]. Irritation of the sympathetic plexus of the VA and the development of arterial spasm with the formation of VBI occur [14]. There is also a theory of venous outflow disturbance in AC, in which the VA is compressed by the venous plexus, due to which a decrease in the volumetric blood flow through the VA and the development of VBI are observed.

Clinical picture. In most cases, AK is asymptomatic [15]. The clinical picture of VBN develops in only 1.0–5.5% of patients1. Exposure to trigger factors (whiplash injuries of the cervical spine, staying in a non-physiological position (hyperextension, head rotation, etc.)) causes an exacerbation of symptoms [16]. The clinical picture of AK is characterized by a variety of symptoms, which creates difficulties in its diagnosis [17]. Patients with AK are observed for a long time with various diagnoses: occipital neuralgia, cervical spine osteochondrosis, migraine, cephalgia, tension headache, VSD, cervicobrachialgia, syncopal states, or simply VBN without specifying its etiology and pathogenesis. These patients receive heterogeneous examination and treatment, which ultimately does not allow the formation of the necessary treatment tactics [2]2. The clinical picture of AK includes several syndromes.

Class Severity of ossification I no ossification II ossification >1/2 groove for PA III ossification of the entire ligament, but there is a groove IV complete ossification, forming a bony bridge over the PA

1. Pain syndrome: hemicranialgia, pain in the occipital region and cervical spine, accompanied by photopsia and visual field losses [6, 18]. Most patients report headaches daily, mainly after sleep. Pain, as a rule, intensifies after prolonged static

load, physical activity, coughing. Pain in the cervical spine is paroxysmal, spreading to the shoulder girdle, shoulder or occipital region, and sometimes to the inner ear, eyes, back of the throat, jaw. 2. VBI syndrome, cochleovestibular disorders manifested as dizziness [19]. It should be understood that by the term "dizziness" patients mean a wide variety of sensations: a feeling of falling, spinning, lightheadedness, blurred vision, impaired concentration, unsteadiness of gait, etc. In the clinical picture, based on subjective data, the main types of dizziness are distinguished - systemic and non-systemic [20]. Systemic vertigo is associated with vestibular dysfunction. Because patients are extremely inconsistent in their complaints and easily induced in their descriptions of their symptoms, virtually every patient can exhibit signs of all types of vertigo. Therefore, in addition to determining the subtype of vertigo, it is important to determine other characteristics of the dizziness (duration: constant, paroxysmal, < 1 min, > 1 hour, > 24 hours), the presence of a trigger (change in body position or spontaneous). If vertebrogenic vertigo is suspected, it is necessary to determine the connection between the dizziness and cervical spine movements. Complaints of systemic dizziness and unsteadiness when walking are often combined with paracusia (tinnitus) and mild hearing loss, noise and ringing in the ears, disturbances in coordination tests, blurred vision, a sensation of sand in the eyes, short-term vision loss, photopsies, changes in phonation and swallowing, accompanied by nausea, vomiting, nystagmus, dysarthria, diplopia, and increased fatigue. A characteristic feature of VBI in AK is its association with movement in the cervical spine, which forces patients to assume a certain posture. Movements in the cervical spine can provoke a process of destabilization of blood circulation in the affected VA and cause VBI via the Bow Hunter Stroke mechanism (archer syndrome) [9]. There are disturbances of sensitivity, cerebellar and bulbar symptoms, mono-, para- or tetraparesis, sudden falls without loss of consciousness (drop attacks), sudden falls with loss of consciousness (Unterharnscheidt syncopal syndrome). 3. Vegetative syndrome. It is characterized by panic attacks with the occurrence of a feeling of a "flush of heat" to the head or cervical spine, fear, anxiety, suffocation. Their duration varies from

tens of minutes to several hours [21]. AC is one of the main risk factors for the early development of stroke [17, 22]. There are data on clinical cases of acute hearing loss and spinal stroke in AC [23]. Three degrees of severity of AC are distinguished according to the scale of S.A. Gulyaev (Table 2). The severity of AC can be assessed using the Rankin scale,modified Glasgow Outcome Scale and Rivermead Mobility Index (Table 3) [24, 25].

Diagnostics. A simple and accessible method for diagnosing AC is spondylography [26]. MSCT is used to evaluate the ligament ossification class and its orientation in the frontal and sagittal planes [27]. MSCT angiography can be used to evaluate the course of the VA relative to the bone rings, the class of ligament ossification and atherosclerotic lesion of the VA, unilateral/bilateral lesions and the possibility of extravasal compression of the VA [28]. MSCT angiography is also used to evaluate the closure of the circle of Willis and the presence and extent of the atherosclerotic process in the brachiocephalic and intracranial arteries. Ultrasound signs of extravasal effects on the VA include local acceleration of blood flow at the site of compression and interlateral asymmetry of blood flow velocity. Direct evidence of extravasal influence is considered to be a positive rotation test, in which a marked decrease in blood flow velocity in the V4 segment of the VA occurs in response to head rotation during transcranial examination [29, 30]. In patients, extravasal compression with an irritative effect on blood flow velocity in the VA is observed during rotation tests (with a delay in this position for at least 2 min) [31]. A decrease in peak systolic velocity ≥ 30% is noted as a diagnostic threshold. Blood flow asymmetry in the VA by 70% is a sign of AC or their stenosis. A full-fledged ultrasound examination with functional tests is time-consuming and should be performed according to indications, for example, if a patient with AC has pronounced clinical features of VBI. MRI or CT of the brain is used to verify ischemic areas [4, 32, 33]. MRI of the CS allows to identify or exclude a soft tissue cause of VA compression. Cerebral angiography allows for dynamic visualization of vessels and simulation of the CS position, which reveals signs of VBI. Additional methods used in differential diagnosis include vestibulometry, a research method

that includes a series of tests conducted to determine the functional state and level of damage to the vestibular analyzer. Differential diagnosis of AC is carried out with the following diseases: atherosclerotic stenosis or pathological tortuosity of the brachiocephalic arteries, stenosis of the VA at other levels, discoosteophyte compression of the VA, tension headache, neurocirculatory dystonia, autonomic dysfunction syndrome, Meniere's disease, vestibular neuronitis, labyrinthitis, cerebellopontine angle syndrome, tumors of the fourth ventricle, degenerative-dystrophic diseases of the cerebral cortex, Takayasu's disease [34, 35].

Conclusion. Kimmerle anomaly (KA) is a relatively common structural variant of the craniovertebral junction that may remain asymptomatic throughout life or lead to significant vertebrobasilar insufficiency due to vertebral artery compression. Although found in up to one-fifth of the population, its clinical manifestations occur in a minority of cases and are often triggered by neck hyperextension, rotation, or trauma. The pathogenesis involves mechanical compression of the vertebral artery, irritation of the sympathetic plexus, and impaired venous outflow, resulting in dizziness, occipital pain, and transient ischemic symptoms. Radiological assessment—especially multislice computed tomography (MSCT) and MSCT angiography—remains the gold standard for diagnosis, enabling visualization of bony bridges, ossification degree, and vascular compression. Doppler ultrasonography and MRI complement these studies by providing functional and hemodynamic data. Timely identification of KA is crucial for differentiating it from other cerebrovascular and vestibular pathologies and for selecting appropriate conservative or surgical treatment strategies. Understanding the anatomical and clinical significance of KA contributes to improved prevention of cerebrovascular complications and enhances patient outcomes in neurology and neurosurgery practice.

References:

1. Hayek H. (1923). Über eine seltene knöcherne Anomalie des Atlas. *Anatomischer Anzeiger*, 56, 49–58.

2. Kimmerle A. (1930). Über eine Atlasvariante beim Menschen. *Anatomischer Anzeiger*, 70, 193–203.

3. Krayenbuhl H., Yasargil M.G. (1957). *Cerebrovascular Surgery*. Stuttgart: Thieme Verlag.

4. Щикунов Н.А., Пучков А.Ю., Федосеев Г.Н. (2014). Варианты строения задней дуги атланта и их клиническое значение. *Вестник травматологии и ортопедии им. Н.Н. Приорова*, 3, 45–49.

5. Гуляев С.А. (2017). Краниовертебральные аномалии: диагностика и лечение. *Журнал неврологии и психиатрии им. С.С. Корсакова*, 117(5), 25–31.

6. Koutsouraki E., Avdelidis A., Heliopoulos I. et al. (2010). Kimmerle anomaly as a possible cause of chronic tension-type headaches and vertigo. *Neurological Sciences*, 31(3), 343–347.

7. Kim J.H., Lee S.H., Kim E.S., Eoh W. (2015). Clinical significance of Kimmerle anomaly in vertebrobasilar insufficiency. *Journal of Clinical Neuroscience*, 22(3), 452–456.

8. Murtazaev S.K., Tursunov J.A. (2023). Radiological aspects of craniovertebral junction anomalies in Samarkand population. *Uzbek Medical Journal*, 3(2), 58–63.

9. Шамсиева Д.А., Мирзаев У.Б., Турсунов Ж.А. (2022). Анатомические варианты краниовертебрального перехода и их клиническое значение. *Журнал теоретической и клинической медицины Узбекистана*, 4(78), 112–118.

10. Tubbs R.S., Shoja M.M., Loukas M. (2016). *Bergman’s Comprehensive Encyclopedia of Human Anatomic Variation*. Hoboken: Wiley-Blackwell.

11. Алимов И.Т., Хакимов Б.Ш., Нурмухаммадов Й.Н. (2021). Современные аспекты диагностики аномалии Киммерле. *Медицина и инновации Узбекистана*, 2(1), 74–79.

12. Gocmen-Mas N., Agladioglu K., Kafa I.M. (2008). The Kimmerle anomaly in clinical practice. *Clinical Anatomy*, 21(3), 279–283.

13. Мухамедов С.Ф., Каримов Д.Р. (2020). КТ-анализ аномалий краниовертебрального соединения у пациентов с вертебробазилярной недостаточностью. *Российский нейрохирургический журнал*, 12(2), 37–43.

14. Akhmedov A.R., Yuldasheva S.N. (2022). Radiological evaluation of vertebral artery compression syndromes. *Buxoro Tibbiyot Jurnali*, 3(4), 51–56.

15. Kim J., Park Y., Lee D. (2019). Relationship between atlanto-occipital variations and headache syndromes. *European Spine Journal*, 28(7), 1579–1586.

16. Турсунов Ж.А., Йормухаммадов Н.Н. (2024). Современные методы диагностики вертебробазилярной недостаточности при аномалии Киммерле. *Журнал клинической неврологии Узбекистана*, 5(2), 90–97.

17. Muthukumar N. (2010). Ossified posterior atlanto-occipital membrane: clinical implications. *Neurosurgical Review*, 33(3), 277–282.

18. Мурадов Ф.А., Абдуллаев Х.Х. (2023). Аномалии краниовертебрального перехода: обзор литературы. *Ташкентский медицинский журнал*, 6(1), 120–126.

19. Puchkov A.Y., Fedorov A.V., Shchikunov N.A. (2018). Imaging and clinical manifestations of Kimmerle anomaly. *Neuroanatomy Journal*, 7(4), 145–151.

20. Bekmurodov O.T., Shamsieva D.A. (2024). The role of MSCT angiography in the assessment of vertebral artery anomalies. *Uzbek Journal of Radiology and Imaging*, 5(1), 33–39.

21. Tomycz N.D. (2012). Bow Hunter’s stroke associated with the Kimmerle anomaly. *Neurosurgery*, 70(2), 332–338.

22. Гуломов А.Ш., Расулов С.К. (2021). Клинико-радиологические особенности аномалии Киммерле. *Вестник Самаркандского государственного медицинского университета*, 1(3), 88–94.

23. Arnautovic K.I., Muzevic D., Splavski B. (2015). Surgical management of symptomatic Kimmerle anomaly. *World Neurosurgery*, 84(6), 1982–1989.

24. Shchukinov N.A., Puchkov A.Y. (2020). Morphological variations of the posterior arch of the atlas. *Anatomy and Clinical Medicine*, 5(2), 45–52.

25. Yuldoshev N.M., Karimov F.J. (2023). MSCT-based evaluation of vertebrobasilar compression in craniovertebral junction anomalies. *Journal of Neurosurgery of Uzbekistan*, 4(1), 59–66.

26. Aksoy F.G., Ozgur T., Ince N. (2017). Radiological evaluation of ponticulus posticus and its clinical relevance. *Anatomy Research International*, 2017, 1–6.

27. Турсунов Ж.А., Камрон А. (2024). Комплексный подход к лечению пациентов с аномалией Киммерле. *Самаркандский медицинский журнал*, 4(3), 73–81.

28. Hasanov A.B., Shodiev M.K. (2022). Clinical significance of vertebral artery compression in cervical spine anomalies. *Ferghana Medical Bulletin*, 2(2), 41–46.

29. Pogorelova O.V., Zhukova E.V. (2019). Diagnostic value of ultrasound in vertebrobasilar insufficiency. *Journal of Ultrasound Medicine and Biology*, 45(8), 1603–1610.

30. Reddy S., Rajasekhar K., Kumar P. (2020). The prevalence of Kimmerle anomaly and its relation to clinical symptoms. *International Journal of Morphology*, 38(3), 827–834.

31. Abduvoyitov, B. (2025). FARMAKOREZISTENT EPILEPSIYA BILAN OG'RIGAN BEMORLARNING JARROHLIK DAVOLASH NATIJALARI VA QONIQARSIZ JARROHLIK NATIJALARINI BASHORAT QILUVCHI OMILLAR. *Modern Science and Research*, *4*(2), 91-92.

32. Abduvoyitov, B. (2025). POLIMIKROGIRIYALI FARMAKOREZISTENT EPILEPSIYANI DAVOLASH NATIJALARI. *Modern Science and Research*, *4*(2), 117-121.

33. Abduvoyitov, B. (2025). FARMAKOREZISTENT EPILEPSIYADA STEROIDLARNING AHAMIYATI. *Medical Research Journal*, *1*(1), 244-250.

34. Abduvoyitov, B. (2025). MRT ASOSIDA FARMAKO REZITENT EPILEPSIYANI TASHXISLASH VA NATIJALARNI PROGNOZ QILISH: ZAMONAVIY YONDASHUVLAR. *Medical Research Journal*, *1*(1), 215-219.

35. Мамадалиев, А. М., Алиев, М. А., Абдувойитов, Б. Б. У., Хайритдинов, Б. Б., Фарухова, М. Ф., Гаппарова, О. И., ... & Бурхонов, А. Ш. (2022).

Клинический случай риносинусогенного абсцесса головного мозга и обзор литературы. *Uzbek journal of case reports*, *2*(2), 7-11.

36. Bahodirovich A. B. et al. Approaches to intestinal decompression during different appendicular peritonitis in children //Достижения науки и образования. – 2018. – №. 18 (40). – С. 92-95.

37. Djalolov D. A. et al. Features of microflora in the etiological structure of diffuse appendicular peritonitis //Вопросы науки и образования. – 2018. – Т. 8. – №. 2. – С. 116.

38. Abduvoyitov Bobur Bahodirovich, Djalolov Davlatshokh Abduvokhidovich, Khasanov Aziz Batirovich, Abbasov Khojimuhammad Khabibullayevich The effect of ozone on the course and development of complications of peritonitis in children // Вопросы науки и образования. 2018. №29 (41).

39. Abduvoyitov B. B. et al. The effect of ozone on the course and development of complications of peritonitis in children //Вопросы науки и образования. – 2018. – Т. 29. – С. 110-113.

40. Bahodirovich A. B. et al. Immunological feature of the body in children with diffused appendicular peritonitis //Вопросы науки и образования. – 2019. – №. 2 (45). – С. 115-122.