COMPREHENSIVE ANALYSIS OF CONSERVATIVE AND SURGICAL TREATMENT APPROACHES FOR KIMMERLE ANOMALY

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Abstract: *Kimmerle anomaly is a congenital or acquired bony bridge of the atlas (C1 vertebra) that may compress the vertebral artery and cause vertebrobasilar insufficiency (VBI). The aim of this study was to analyze the effectiveness of conservative and surgical treatment in patients with symptomatic Kimmerle anomaly. A total of 23 patients aged 20–58 years were examined and treated at the Neurosurgical Department of Samarkand State Medical University from 2018 to 2024. Diagnostic evaluation included duplex ultrasonography with functional tests, MSCT angiography of the craniovertebral junction, and MRI of the brain. Conservative therapy (vasoactive agents, neuroprotectors, physiotherapy, cervical immobilization) achieved partial regression of symptoms in 39% of patients; however, recurrence occurred in over half of these cases. Surgical decompression of the vertebral artery, performed via median or paravertebral intermuscular approaches, resulted in complete or significant regression of symptoms in 79% of operated patients, with restoration of vertebral artery flow confirmed by Doppler studies. The results demonstrate the high efficacy and safety of minimally invasive decompression as a definitive treatment method for patients with refractory vertebrobasilar insufficiency caused by Kimmerle anomaly.*

Keywords: *Kimmerle anomaly, vertebral artery compression, vertebrobasilar insufficiency, decompression surgery, minimally invasive approach, craniovertebral junction.*

Introduction. Kimmerle anomaly (also known as arcuate foramen or ponticulus posticus) is a congenital or acquired osseous variation of the atlas (C1 vertebra), characterized by the formation of a bony bridge between the posterior arch of the atlas and its superior articular process. This bridge forms a canal through which the vertebral artery (VA), the suboccipital nerve, and sympathetic fibers pass. The anomaly was first described by H. Hayek in 1923 and later detailed by A. Kimmerle in 1930, who emphasized its potential clinical significance as a cause of vertebrobasilar insufficiency (VBI), chronic headache, dizziness, and transient ischemic attacks.

Although the anatomical variation occurs in 12–30% of the population, only a small proportion of individuals develop clinical symptoms related to compression of the vertebral artery or adjacent neurovascular structures. The pathophysiological mechanism is thought to involve mechanical irritation or hemodynamic impairment of the VA during neck movements, leading to posterior circulation ischemia. Symptomatic cases may present with vertigo, occipital headache, tinnitus, visual disturbances, and, in severe cases, syncopal episodes provoked by head rotation or hyperextension.

Diagnosis of Kimmerle anomaly relies on a combination of imaging modalities, including X-ray of the cervical spine, CT or MSCT angiography of the craniovertebral junction, Doppler ultrasonography with functional tests, and MRI of the brain to exclude ischemic lesions. The choice of management strategy depends on the severity of the clinical manifestations and the degree of vascular compromise. Conservative treatment remains the first-line approach and includes pharmacotherapy (vasoactive agents, neuroprotectors, antioxidants, and muscle relaxants), physiotherapeutic methods (manual therapy, reflexotherapy, gentle cervical traction, local botulinum toxin injections), and cervical immobilization. However, despite symptomatic improvement in 40–90% of cases, recurrence rates remain high, with up to 50–60% of patients requiring repeated treatment courses.

Surgical decompression of the vertebral artery is considered the definitive treatment for refractory cases with documented hemodynamic compromise. Various surgical approaches—median posterior, paravertebral intermuscular, and minimally invasive tubular or endoscopic techniques—have been developed to relieve extravascular compression and restore vertebrobasilar flow. Recent studies demonstrate high efficacy of decompression procedures, with complete regression of neurological and vestibular symptoms and normalization of blood flow parameters in most patients.

Thus, understanding the anatomical variations, clinical presentation, diagnostic methods, and treatment strategies for Kimmerle anomaly is essential for timely recognition and appropriate management of this condition to prevent irreversible cerebrovascular complications.

Materials and Methods

Study Design and Patient Selection

The present study included patients diagnosed with symptomatic Kimmerle anomaly (KA) treated at the Neurosurgical Department of the Samarkand State Medical University from 2018 to 2024. The diagnosis was established based on clinical presentation, neurological examination, and radiological findings confirming the presence of an osseous bridge (ponticulus posticus) at the craniovertebral junction.

Inclusion criteria comprised:

1. Clinical symptoms suggestive of vertebrobasilar insufficiency (dizziness, occipital headache, tinnitus, unsteadiness, visual disturbances, or transient loss of consciousness).
2. Radiological confirmation of Kimmerle anomaly on CT or MRI.
3. Evidence of vertebral artery compression or hemodynamic alteration on duplex ultrasonography or CT/MR angiography.
4. Failure of conservative therapy for at least 6 months.

Patients with congenital anomalies of the cervical spine other than KA, vertebral fractures, or previous craniovertebral surgery were excluded.

Diagnostic Evaluation

All patients underwent comprehensive neurovascular assessment, including:

- **Ultrasound Doppler examination** of the vertebral arteries with functional tests (head rotation and extension) to evaluate changes in blood flow velocity and direction.

- **Multislice CT (MSCT) angiography** of the brachiocephalic and intracranial arteries to visualize the level of compression and determine the type of bony bridge (complete or incomplete).
- **MRI of the brain** to identify ischemic lesions or white matter hyperintensities associated with chronic vertebrobasilar insufficiency.
- **Neurological examination** assessing cranial nerve involvement, vestibular function, and cerebellar coordination.

Conservative Treatment Protocol

Before surgical consideration, all patients received standardized conservative therapy for a period of 3–6 months, including:

- **Pharmacotherapy:** muscle relaxants (tolperisone, tizanidine), antioxidants (mexidol, emoxypine), vasoactive agents (cinnarizine, betahistine), neuroprotectors (citicoline, piracetam), venotonics, and non-steroidal anti-inflammatory drugs (NSAIDs).
- **Physiotherapy:** reflexotherapy, soft cervical traction, local botulinum toxin type A injections for paraspinal muscle relaxation, vertebral artery block, and ozone therapy.
- **Cervical immobilization:** the use of a soft cervical collar (Shants collar) to restrict excessive head movement and reduce mechanical stress on the vertebral artery.

The effectiveness of conservative management was assessed by the reduction of symptoms and improvement of blood flow velocity on Doppler studies.

Surgical Indications and Technique

Surgical decompression was indicated in patients with:

- Progressive neurological or vestibular symptoms despite medical therapy.
- Documented vertebral artery compression with >50% flow reduction on dynamic Doppler or CT angiography.
- Persistent vertebrobasilar insufficiency and hemodynamic instability during head movement.

Two main surgical approaches were used depending on the laterality and configuration of the anomaly:

1. **Median posterior approach** — a skin incision was made along the midline from the external occipital protuberance to the spinous process of C3. Subperiosteal dissection exposed the posterior arch of C1 and the anomalous bony bridge, which was then resected using a high-speed diamond drill under microscopic magnification.
2. **Paravertebral intermuscular (minimally invasive) approach** — performed under intraoperative X-ray or neuronavigation guidance. A 3–4 cm paramedian skin incision was made along the posterior border of the sternocleidomastoid muscle at the level of the C1 arch. After gentle muscle splitting, a tubular retractor system was inserted perpendicular to the arcuate foramen. The bony bridge compressing the vertebral artery was removed under microscopic or endoscopic visualization.

Intraoperative ultrasonography was used in all cases to confirm decompression and restoration of pulsatile flow in the vertebral artery. Hemostasis was achieved with bipolar coagulation and hemostatic materials. Wound closure was performed in layers without drainage.

Postoperative Management and Follow-up

Patients were mobilized on postoperative day one with a soft cervical collar for 5–7 days. Neurological status and vertebrobasilar symptoms were assessed daily during hospitalization. Control Doppler ultrasonography was performed on days 3 and 7 post-operation to verify the restoration of arterial blood flow.

Long-term follow-up evaluations at 3, 6, and 12 months included neurological examination, Doppler studies with functional tests, and assessment of quality of life and work capacity. Surgical outcomes were classified as complete regression, partial improvement, or no change in symptoms.

Results and Discussion

Clinical Outcomes

A total of **23 patients** (12 women and 11 men, aged 20–58 years, mean age 41.6 ± 7.9) with symptomatic Kimmerle anomaly were treated between 2018 and 2024. All patients presented with symptoms of vertebrobasilar insufficiency (VBI), including occipital headache (100%), dizziness (91%), tinnitus (65%), transient visual disturbances (48%), and syncopal episodes (26%). Duration of symptoms ranged from 6 months to 3 years.

Conservative therapy resulted in partial regression of symptoms in 9 patients (39%). However, recurrence occurred in 6 of these patients within 4–6 months, necessitating further intervention. Fourteen patients (61%) with persistent or progressive manifestations underwent surgical decompression of the vertebral artery.

Radiological and Hemodynamic Findings

Preoperative Doppler ultrasonography demonstrated a 45–70% reduction in blood flow velocity within the affected vertebral artery during head rotation or extension. Multislice CT angiography confirmed extravascular compression at the level of the posterior arch of C1 caused by a complete or incomplete bony bridge (ponticulus posticus). In four cases, bilateral anomalies were identified.

After decompression, intraoperative ultrasonography showed immediate restoration of laminar blood flow and elimination of the turbulence pattern. On postoperative day 7, Doppler studies confirmed normalization of flow velocity and symmetry between the vertebral arteries in all operated patients.

Early and Long-Term Results

No intraoperative vascular injury, neurological deterioration, or cerebrospinal fluid leakage occurred. Postoperative pain was mild and managed with standard analgesics. Hospitalization time averaged 6.2 ± 1.4 days.

During follow-up (mean 12 months, range 6–24 months), the majority of patients demonstrated stable neurological and hemodynamic improvement:

Outcome measure	n (%) of surgical patients

Complete regression of symptoms	11 (78.6%)
Partial improvement	3 (21.4%)
Recurrence or persistent VBI	0 (0%)
Normalization of vertebral artery flow on Doppler	13 (93%)
Return to previous work capacity	12 (85.7%)

These findings are consistent with previously reported results. A.K. Chetrkov et al. (2005) observed regression of general cerebral symptoms in 90%, disappearance of dizziness and imbalance in 87%, and complete recovery from syncopal episodes in 100% of patients after decompression surgery. Similarly, V.V. Krylov et al. (2017) described six operated patients (mean age 42.7 years) in whom pain and VBI symptoms regressed completely in three cases and significantly improved in the remaining two, confirming the high clinical efficacy of the intervention.

Discussion

The management of Kimmerle anomaly remains controversial due to its frequent asymptomatic presentation and variable clinical significance. The anomaly itself is often regarded as an incidental radiological finding; however, in patients with characteristic symptoms and documented hemodynamic impairment, it becomes a surgically correctable cause of vertebrobasilar insufficiency.

Conservative management remains the first-line approach, especially in patients with mild or transient symptoms. Pharmacotherapy (vasoactive agents, antioxidants, muscle relaxants, and neuroprotectors) combined with physiotherapy and cervical immobilization may reduce symptoms temporarily. Nevertheless, the recurrence rate of 50–60% observed both in our series and in the literature highlights the limitations of symptomatic treatment when structural compression persists.

Surgical decompression of the vertebral artery represents a rational solution in refractory cases. The goal of surgery is to remove the osseous bridge (ponticulus posticus) compressing the V3 segment of the vertebral artery, thereby restoring normal blood flow through the vertebrobasilar system. Our results confirm that decompression yields rapid and sustained improvement of neurological and vestibular symptoms, with normalization of arterial hemodynamics.

Minimally invasive and neuronavigated approaches offer significant advantages over the classical midline technique. The paravertebral intermuscular access using tubular retractors minimizes muscular trauma, blood loss, and postoperative pain, enabling earlier rehabilitation. These results agree with modern literature, where minimally invasive decompression achieved comparable outcomes with fewer complications and shorter hospital stays (Zhang et al., 2021; Liu et al., 2022).

The absence of intraoperative complications and the high rate of functional recovery in our cohort further confirm the safety and reliability of the described technique. Long-term follow-up demonstrated persistent regression of symptoms and restoration of work capacity in the majority of patients.

Limitations

The study is limited by the relatively small sample size and the absence of a control group of conservatively treated patients matched by severity and duration of symptoms. Future multicenter studies with larger cohorts and quantitative perfusion imaging could provide a more objective assessment of surgical efficacy.

Conclusion.

Kimmerle anomaly represents a clinically significant cause of vertebrobasilar insufficiency in patients with unexplained vestibular and neurological symptoms. Although often considered an anatomical variant, the presence of a bony bridge over the vertebral artery may lead to hemodynamic compromise, especially during head rotation or extension.

Conservative therapy remains the first-line treatment and can achieve temporary symptomatic relief in many patients. However, its long-term effectiveness is limited, with frequent symptom recurrence due to persistent mechanical compression.

Surgical decompression of the vertebral artery provides definitive resolution in patients with progressive or refractory symptoms and documented flow impairment. The paravertebral intermuscular and minimally invasive approaches offer safe and effective alternatives to traditional midline exposure, reducing operative trauma, postoperative pain, and hospitalization time while ensuring full decompression of the vertebral artery.

Our clinical experience, supported by published data, demonstrates that surgical treatment achieves complete or substantial regression of symptoms in the vast majority of patients, normalization of vertebrobasilar hemodynamics, and restoration of working capacity.

Further prospective studies with larger cohorts and long-term follow-up are required to optimize patient selection criteria and establish standardized treatment algorithms for symptomatic Kimmerle anomaly.

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