AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 3, Issue 10, 2025 ISSN (E): 2993-2149

Surgical Treatment of Kimmerle's Anomaly

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Introduction. Patients with vertebrobasilar insufficiency and pain syndrome, which are frequently encountered in the practice of a neurologist and neurosurgeon, require a comprehensive examination to clarify the etiologic factor in the development of vertebrobasilar insufficiency and conduct differential diagnostics [1,2]. One of the possible causes of this condition is the Kimmerle anomaly - a variant of the structure of the first cervical vertebra (atlas), in which an additional bony arch is formed, extending from the posterolateral part of the lateral mass of the atlas above the groove of the vertebral artery and connecting with its posterior arch, and ossification of a portion of the atlanto-occipital ligament is also possible. The diagnostic complex, in addition to a clinical and neurological examination, includes spondylography, computed tomography and magnetic resonance imaging of the cervical spine, Doppler ultrasound and direct angiography. A dependence of the frequency and severity of neurological syndromes on the severity of the anomaly and the age of the patients has been established. An algorithm for the comprehensive diagnosis and treatment of this pathology has been developed [2]. Along with Arnold-Chiari malformations, 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 [3,4]. AC occurs in 18.8% of cases according to sectional findings, in 17.2% according to the analysis of multispiral computed tomography (MSCT), and in 16.6% according to spondylography [5,6]. The disease occurs equally often in both men and women (15.8% and 14.6%, respectively). According to N. A. Shchikunov (2014), in a study of 305 incidents of congenital AC, a bilateral symmetrical variant was observed in 42% of cases. In this case, 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; of these, a completely closed canal on the left and an incompletely closed one on the right occurred in 12% of cases, and a completely closed canal on the right and an incomplete one on the left occurred in 7%. Unilateral AC was determined in 39% of cases, of which a left-sided anomaly was noted more often (30%), and a right-sided anomaly less often (9%). In the case of a left-sided canal of the PA, it was incomplete in 16% of cases and complete in 14% [7,8].

Keywords: kimmerle anomaly, vertebrobasillary insufficiency, pain syndrome, vertebral artery, vertebro-vascular conflict, decompression of the vertebral artery, craniovertebral anomaly

Purpose of the study— optimization of the diagnosis of cerebrospinal fluid flow disorders in children based on comparative clinical and neuroimaging analysis using neurosonography (NSG) and magnetic resonance imaging (MRI) in order to improve the accuracy of early detection, severity assessment and outcome prediction in central nervous system pathologies in newborns and older children.

Materials and methods: Clinical algorithm for the treatment of pulmonary artery disease [3]: Stage 1 (st.) — determination of the atomic variant of pulmonary artery disease; Stage 2 — verification of the group of clinical manifestations, depending on the severity and presence of specific complaints; Stage 3 — identification of the diagnostic minimum of additional examinations and the tactical group. Rules for drug treatment of pulmonary artery disease syndrome [9]: 1. Drugs acting on venous outflow: semi-synthetic diosmin (Detralex, Phlebodia), troxerutin, ginkgo biloba. 2. Non-steroidal anti-inflammatory drugs: nimesulide, lornoxicam, celecoxib, celebrex. 3. Drugs that normalize blood flow in the pulmonary artery: pentoxifylline, vincamine, vinpocetine, cinnarizine, nimodipine, nicergoline, sermion, instenon. 4. Cholinergic drugs: citicoline, ceraxon, recognan, gliatilin. 5. Complex neuroprotective drugs: cytoflavin, actovegin, cerebrolysin, piracetam, mexidol. 6. Metabolic therapy: cytoflavin, mildronate, thiotriazoline, trimetazidine. 7. Symptomatic therapy - muscle relaxants: tolperisone, antispasmodics: drotaverine, histamine-like: betahistine, antimigraine: sumatriptan. Modern conservative treatment includes: physiotherapy (intrastic electrical stimulation, acupuncture, gentle traction of the cervical spine, local administration of botulinum toxin type A, PA blockade, ozone therapy), massage of the cervical collar zone, immobilization of the cervical spine [10]. The results of the examined inpatients of the Neurosurgery Department of the Multidisciplinary Clinic of the Samarkand State Medical University in the period 2024-2025 were analyzed.

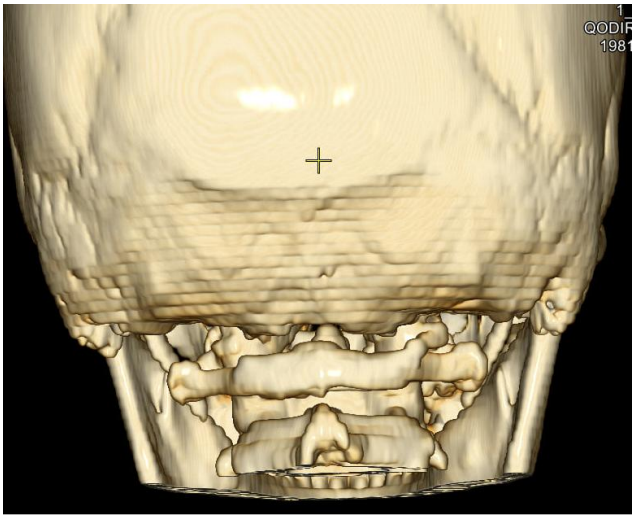
More than 25 years after the introduction of fetal MRI for diagnosing central nervous system (CNS) anomalies, it has become clear that this method provides important additional information to clarify diagnoses and improve counseling for patients with cerebrospinal fluid flow disorders. However, ultrasound, including neurosonography, remains the primary method for screening and primary diagnosis due to its availability, safety, and ability to be performed at the patient's bedside [1].

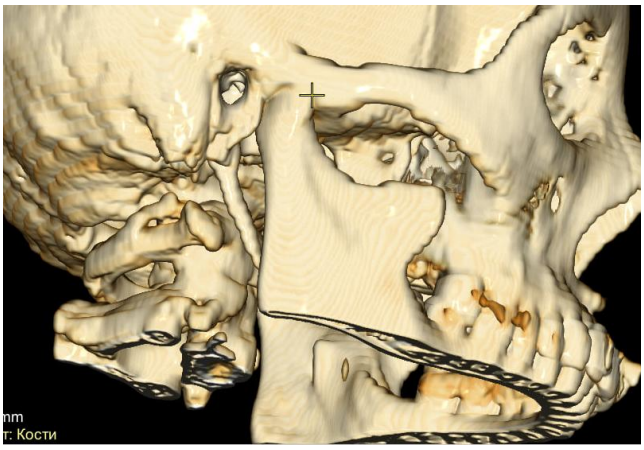
Before surgical correction of VBI, all other causes that could lead to the clinical picture must be objectively excluded. Surgical treatment is the definitive treatment method. Only a few studies have been devoted to surgical treatment of AK. Absolute indications for this procedure include progressive symptoms of AK, ineffective conservative therapy for 6 months, and positive functional tests. Indications for surgical treatment of AK: 1. Severe clinical manifestations of autonomic dysfunction and VBI.

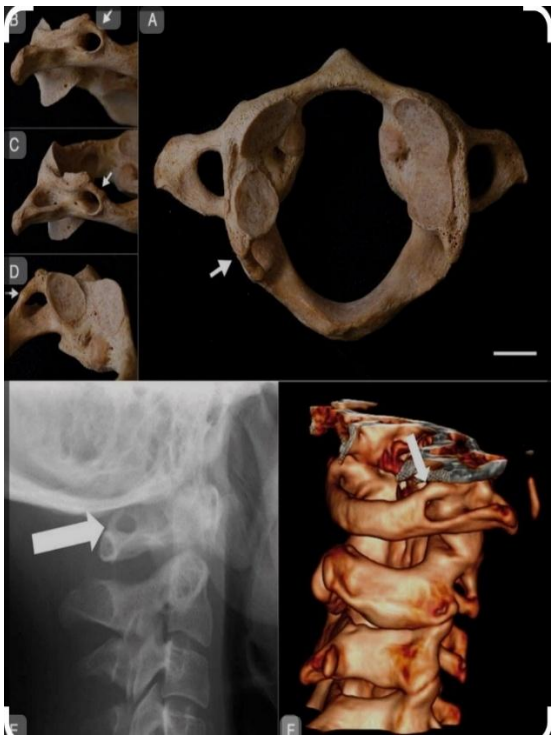
Patients spend most of the time in a supine position, and an exacerbation develops with head rotation (3 points or more according to Rankin and 7 points or less according to Rivermead) 2. Moderate disease course. Relapses of vegetative syndrome and VBI several times a year, while the intensity of attacks increases. Rankin = 1-2 points, Rivermead = 8-13 points. 3. Mild course. Minimal manifestations of vegetative syndrome and VBI, accompanied by drug-resistant radiculopathy. Rankin = 1 point, Rivermead = 14-15 points. Surgical technique. Decompression of the VA is performed through the posterior median or paravertebral intermuscular approach. The patient is positioned prone, the head is fixed in a Mayfield clamp. During the classic procedure, a skin incision is made along the posterior midline from the occipital protuberance to the spinous process of the C3 vertebra. After dissecting the skin and subcutaneous fat, the occipital bone, C1 tubercle, and C2 spinous process are exposed through the nuchal ligament. To adequately visualize the C1 arch and the bony rings to be resected, a sufficiently wide skeletonization of the craniovertebral junction bone structures is required: the occipital bone should be at least 3 cm lateral to the posterior midline, and the C1 arch should extend up to the C2-3 joint. The main disadvantage of this approach is its traumatic nature, as it utilizes a posterior midline approach (along the posterior midline of the neck) with exposure of the occipital bone and the posterior structures of the C1-2 vertebrae. In addition to a large skin incision with a cosmetic defect, with such approaches during skeletonization of the posterior vertebral structures, severe trauma is inflicted on the muscular apparatus of the cervical spine, which causes pain in the postoperative period in such patients, limitation of mobility due to muscle-tonic syndrome

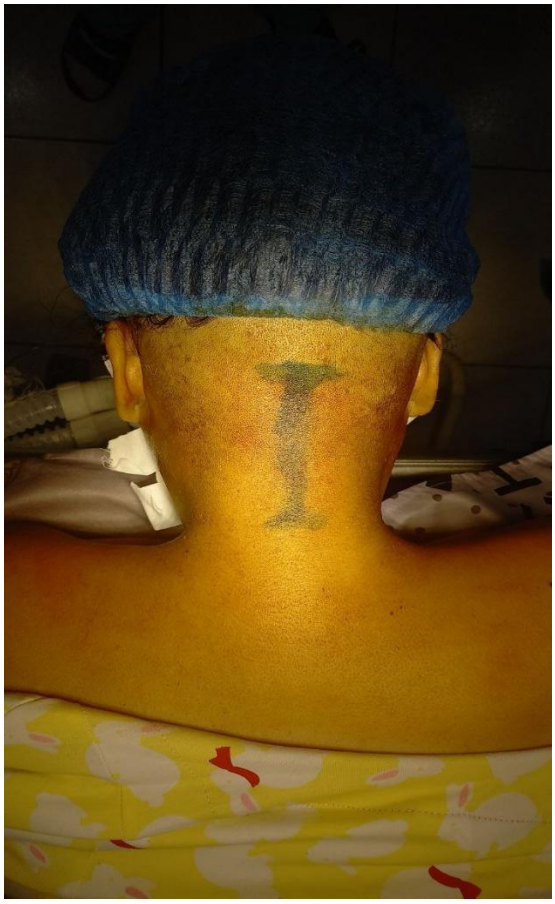
Research results:

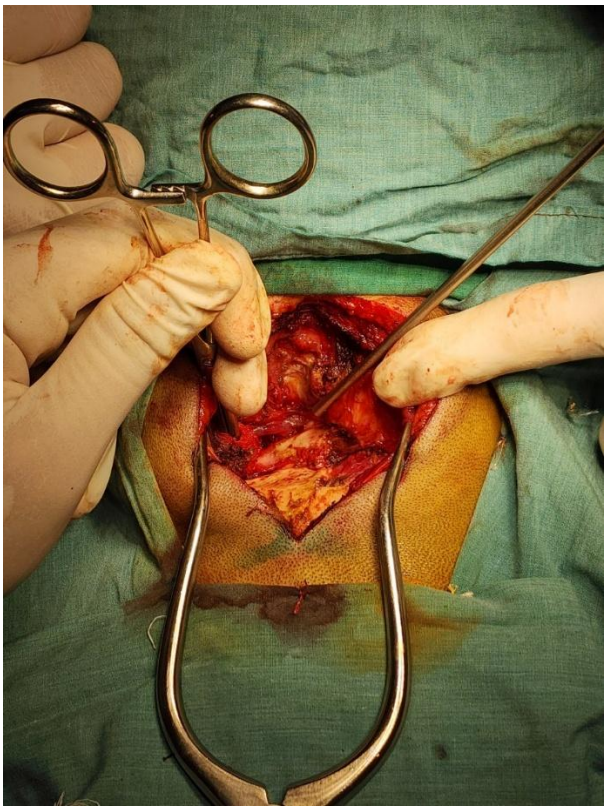
Pic 1. Anomaly Kimmerle on the right side

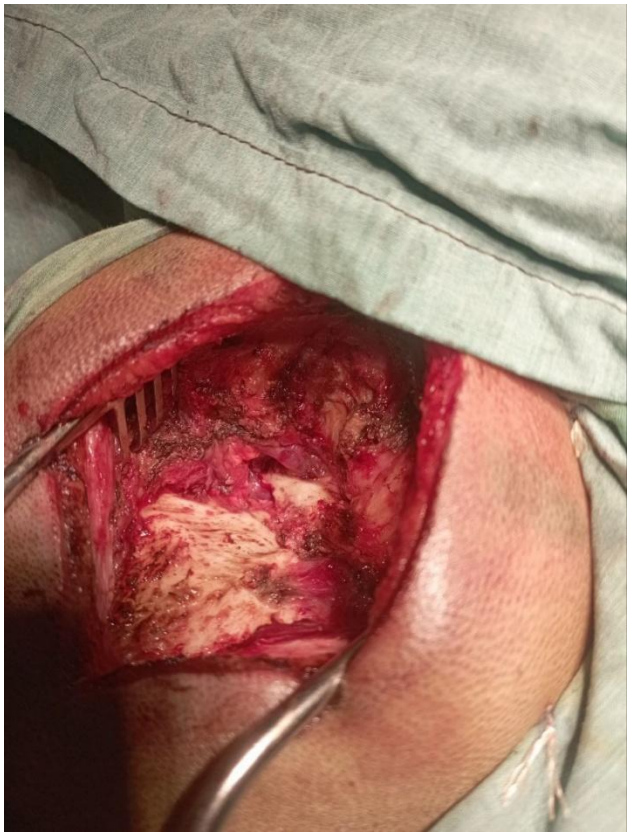


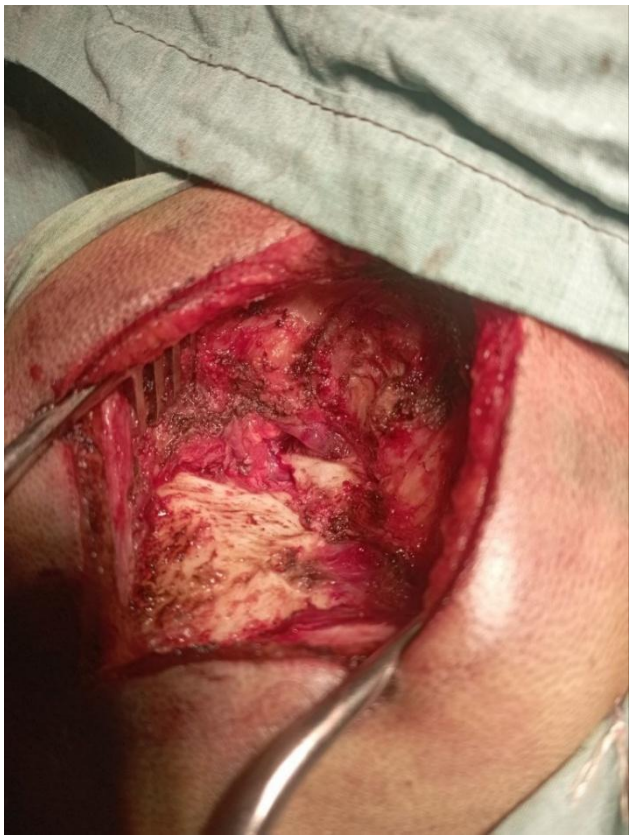


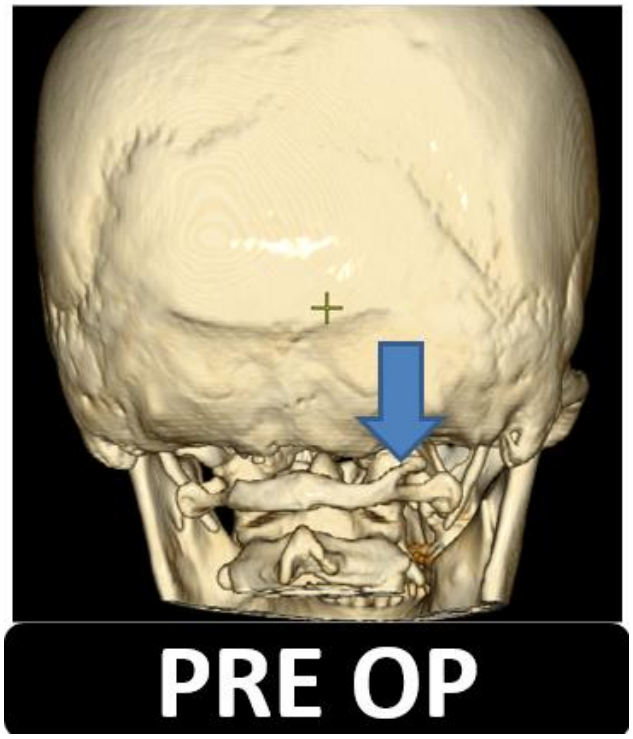








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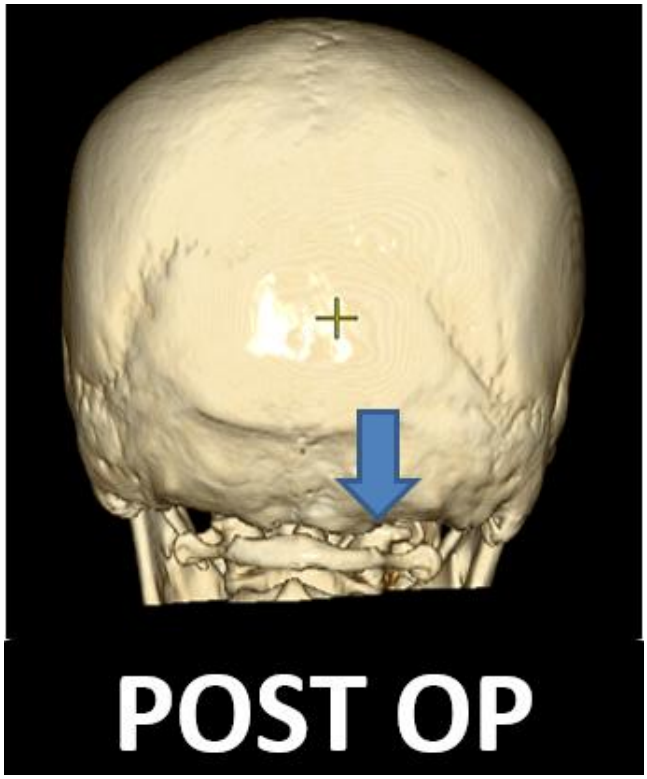
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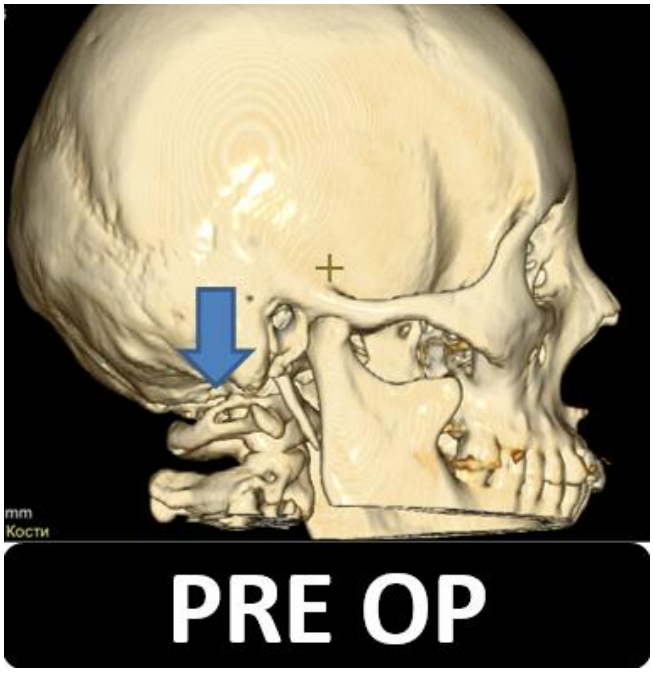

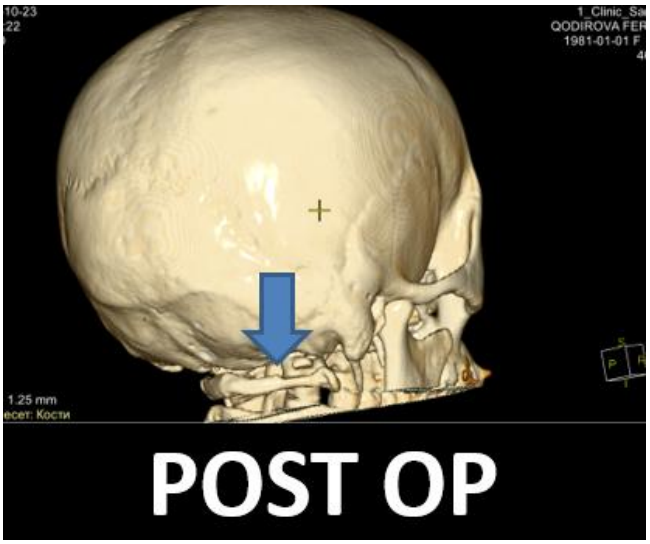

Conclusion: The main pathogenetic factor in the development of disease symptoms is extravasal compression of the VA, prolonged trauma to the vessel adventitia, irritation of the paravasal sympathetic fibers and branches of the occipital nerve. Surgical treatment of VBI in patients with AK is preferred in the absence of positive dynamics against the background of conservative therapy and the progression of neurological deficit with an increasing frequency of paroxysms. Reconstructive vascular surgery for VBI syndrome in AK should be performed only for therapeutic purposes, and their timely implementation allows for sustainable clinical improvement in most patients. Performing surgery through paravertebral intermuscular approaches is an alternative to traditional intervention through the posterior median approach. This minimally invasive approach provides complete visualization of the compressed area of the V3 segment of the VA and helps minimize postoperative pain.

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