ISSN: 2684-5997 Open Access

# Anesthetic Modality-Related Intracranial Hypotension: Some Insights

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### Introduction

Despite being a rare condition, spontaneous intracranial hypotension (SIH) is very incapacitating and has a wide range of presentation and prognosis. The most typical symptom is acute orthostatic headache, however there can also be additional clinical presentations such neck discomfort or stiffness, nausea, tinnitus, or photophobia. Despite its rarity, more people are becoming aware of it thanks to improved access to and advancements in neurological imaging tests like computed tomographic myelography and spine MRI. SIH is believed to be brought on by a cerebrospinal fluid (CSF) leak or a low CSF pressure, despite the fact that the precise aetiology is uncertain, which has given rise to a variety of myths [1-3].

In patients with well-defined SIH symptoms and typical cranial imaging abnormalities, there may occasionally be no evidence of CSF leak despite a full and focused diagnostic workup. In these situations, a pathological cranial-to-spinal shift without a visible CSF leak has been proposed. This might be as a result of increased spinal compliance, which results in clinical signs of SIH and downward displacement of cranial structures. The mainstay of treatment is an epidural blood patch (EBP) with an anesthesiologist if conservative therapy is unable to relieve the patient's symptoms.

## **Description**

In some circumstances, such as those involving dural rips, ruptured meningeal diverticula, or CSF-to-venous fistulas, CSF leaks may be treatable surgically. Due to a lack of understanding or consideration of the diagnosis, SIH is frequently misdiagnosed. We provide a case series of three patients with different clinical characteristics of SIH; they complained of both intolerable dizziness and headaches, the classic and most typical symptoms of the illness. The MRI results supported the preliminary SIH diagnosis. After that, the patients received an EBP with success and continued to be symptom-free during follow-up.

The goal of this case series is to demonstrate how difficult it is to diagnose SIH, which can manifest with a wide range of clinical symptoms. The diagnosis is made primarily based on the severity of the patient's clinical features, and magnetic resonance imaging of the brain and spine rounds out the process. It can result in subdural hematoma (SDH), and if conservative therapy is unsuccessful, an EBP is still the basis of treatment.

SIH is an uncommon syndrome with a 5-per-100,000 annual incidence that peaks in the fourth or fifth decade of life and is marginally more prevalent in women [4,5]. The debilitating occipital or frontal positional diffuse headache

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Received: 05 September, 2022, Manuscript No. japre-23-85764; Editor Assigned: 06 September, 2022, PreQC No. P-85764; QC No. Q-85764; Reviewed: 12 September, 2022; Revised: 19 September, 2022, Manuscript No. R-85764; Published: 27 September, 2022. DOI: 10.37421/ 2684-5997.2022.05.156.

symptoms that worsen with resuming upright posture, better with recumbent posture, and disappear within 15 to 30 minutes of lying down or performing other actions that raise intra-abdominal pressure define the clinical presentation. Orthostatic headache does not always exist, though.

Other signs and symptoms may include vomiting, nausea, soreness or stiffness in the posterior neck, diplopia, blurred vision, photophobia, tinnitus, other subjective hearing problems, and, in rare instances, a comatose state resembling encephalopathy. Most headaches start suddenly, and patients often recall the exact moment when they first felt the symptoms. There are two potential pathways for how headaches develop in SIH: First, due to the drooping of the brain and CSF leakage from the dural rip, pain-sensitive sensory cranial nerve fibres are stretched and stimulated.

### Conclusion

Therefore, any loss in CSF volume should be accompanied by an increase in cerebral blood volume to make up for it, even if intracranial volume remains same. This mechanism is furthered by the advantageous effects of vasoconstrictor drugs like coffee and theophylline. Intense dizziness and a headache were the two main complaints of our patients, which is a unique clinical presentation of the illness. It is likely that a rip in the dura matter causes CSF leak and subsequent intradural CSF hypovolemia in the majority of cases, even though the exact pathogenesis is unknown. Theories about connective tissue problems, starvation, short stature, or female predominance as a result of hormones are unfounded, and symptoms can manifest without a definite reason (prior trauma, history of central neuraxial block, iatrogenic causes). Although low CSF pressure was originally considered to be a major contributing factor to the syndrome, it is now assumed that in most cases, the pressure is normal, suggesting that inadequate CSF volume, rather than low CSF pressure, is the actual cause of the condition.

# **Acknowledgement**

None.

## **Conflict of Interest**

There are no conflicts of interest by author.

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**How to cite this article:** Cera, Elisa. "Anesthetic Modality-Related Intracranial Hypotension: Some Insights." J Anesthesiol Pain Res 5 (2022): 156.