fortune Journals

Research Article

JOURNAL OF SURGERY AND RESEARCH

ISSN: 2640-1002

Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy

Abu Saleh Mohammad Abu Obaida[1], Sheikh Forhad[2], A.K.M. Khalequzzaman[3], Sayem Al Monsur Faizi[4], Muhammad Fakhrul Islam[5], Abu Naim Wakil Uddin[1], Md. Ruhul Kuddus[1], Md. Mohiuddin Biswas[1], Md. Shamsul Alam[1], Asifur Rahman[*1] ORCID

Abstract

Chiari Malformation 1 (CM1) surgery is as challenging as understanding its intricate pathophysiology. Syringomyelia (SM) which is often present and is closely related to the pathophysiology of CM1 makes surgery formidable. The pathology of CM1 poses challenges at every step in its entire perioperative period. Among many postoperative complications, respiratory arrest or respiratory distress is a relatively uncommon yet devastating one. Several factors may lead to postoperative respiratory problems which can be fatal. Understanding the intricate pathophysiology of the respiratory problem in CM1 is important. Here we discuss the probable pathophysiologies and an effective way to minimize those by being meticulous in the peroperative positioning of the patient.

Keywords: Chiari malformation 1; Respiratory arrest; Respiratory failure; Peroperative positioning

Highlights

1. Surgery of Chiari Malformation 1 (CM1) is challenging.
2. Postoperative respiratory problems are relatively uncommon following CM1 surgery. But that may prove fatal.
3. Understanding the intricate pathophysiology of respiratory problems in CM1 is important.
4. The simple technique of meticulous peroperative positioning of the CM1 patient can prevent postoperative respiratory complications.

Abbreviations list

BAEP: Brainstem auditory evoked potential

CM1: Chiari Malformation 1

CSF: Cerebrospinal fluid

CVJ: Craniovertebral junction

iMRI: Intraoperative magnetic resonance imaging

PFD: Posterior fossa decompression

POD: Postoperative days

SM: Syringomyelia

Affiliation:

[1]Department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University, Kazi Nazrul Islam Avenue, Shahbag, Dhaka 1000, Bangladesh

[2]Department of Orthopedics, Bangabandhu Sheikh Mujib Medical University, Kazi Nazrul Islam Avenue, Shahbag, Dhaka, Bangladesh

[3]Department of Orthopedics, Universal Medical College Hospital, Mohakhali, Dhaka, Bangladesh

[4]Department of General Surgery, Bangabandhu Sheikh Mujib Medical University, Kazi Nazrul Islam Avenue, Shahbag, Dhaka, Bangladesh

[5]Department of Neurology, Dhaka Medical College & Hospital, Dhaka, Bangladesh

***Corresponding author:**
Asifur Rahman, Department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University, Kazi Nazrul Islam Avenue, Shahbag, Dhaka 1000, Bangladesh

E-mail: bijoun007@bsmmu.edu.bd

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

Received: November 18, 2024
Accepted: November 28, 2024
Published: December 30, 2024

Abu Obaida ASM, et al., J Surg Res 2024
DOI:10.26502/jsr.10020409

Introduction

Chiari Malformation 1 (CM1) has remained as enigmatic as ever since its first description and surgery has become more challenging, particularly with the gradual unveiling of newer aspects of its pathology. With a better understanding of the pathology, the outcomes are faring better. However, even with the advent of modern technologies and the adoption of newer techniques, the outcomes are often under par. The primary goals of surgical treatment for CM1 are mainly to restore cerebrospinal fluid (CSF) flow and reestablishment of CSF dynamics. Additionally, surgery also helps in relieving pressure on the brainstem and upper spinal cord at the craniovertebral junction (CVJ). Even with all the available facilities and precautions, unexpected complications may happen at times and can be upsetting for the surgeon. Postoperative respiratory arrest is one such disappointing situation that may jeopardize the life of the patient disappointing the surgeon. Respiratory arrest following posterior fossa decompression (PFD) for CM1 is a rare yet devastating event and most of the depictions in the literature addressing this are mostly case reports.

Generally, without any history of previous respiratory dysfunction, postoperative respiratory problems in CM1 patients are uncommon. However, direct or indirect injury to the brainstem may lead to respiratory problems following CM1 surgery. Other than direct intraoperative handling, postoperative edema or ischemia of the brainstem are other important causes of sudden and immediate worsening of patients from respiratory problems. Secondary injury to the brainstem from compression by postoperative hematoma, new herniation, or cerebellar ptosis may also cause respiratory problems. Postoperative acute obstructive hydrocephalus may also result in respiratory distress.

Many factors may contribute to the development of respiratory problems in CM1 patients which are directly or indirectly associated with CM1. Positioning of the patient during surgery is an important factor that may contribute to postoperative respiratory arrest. The importance of peroperative positioning of CM1 patients regarding respiratory problems, whether the patient has respiratory problems preoperatively or not has been discussed little. The pros and cons of positioning CM1 patients during surgery to prevent postoperative respiratory arrest are discussed here.

Postoperative respiratory problems of CM1 patients

With the advent of modern technologies and early detection, the occurrence of respiratory problems has reduced remarkably. Improvement of spectrums of respiratory problems following surgery for CM1 is well documented in the literature. Many case reports as well as findings of cases in series can be found.

It is well established that many CM1 patients having respiratory problems can benefit from posterior fossa decompression [1]. Several authors have described reports of improvement in sleep apnea following surgical decompression in CM1 patients [2-4]. Improvement of respiratory status has been seen in many patients of CM1 after decompression [5]. Florid respiratory distress has also been seen to improve after PFD in CM1 patients [6]. Even patients presenting with respiratory arrest have been reported to have improved and living an independent life [7,8].

There may be persistence or worsening of respiratory dysfunctions in patients of CM1 following posterior fossa decompression, particularly in those who have preoperative respiratory problems. However, there are descriptions of postoperative worsening of respiratory problems even in those CM1 patients who did not have any symptoms preoperatively. Descriptions are mostly in case reports, and the representations are diverse. Respiratory disturbances are not uncommon in postoperative CM1 patients following the common PFD or some other procedure like Gardner's procedure with Obex plugging. Sudden respiratory arrest at surgery during obex plugging has also been reported [9]. Following PFD for CM1, respiratory depression was found to be the most frequent complication in one series most of which occurred mostly at night within the 5th postoperative days (PODs) where one patient died from sleep apnea following surgery [10]. Reports of sudden death long after surgical decompression for CM1 with a history of respiratory dysfunction and persistent sleep apnea can be found in the literature. Death from aspiration pneumonia following surgery and sudden respiratory arrest with a history of sleep apnea have also been described [8,11-13]. A patient with diurnal and exercise-induced hypercapnia and nocturnal alveolar hypoventilation has been reported [14].

Pathophysiology of respiratory complications associated with CM1

Respiratory dysfunctions in CM1 patients may present in diverse forms and each has its pathophysiological postulation. Generally, automatic and voluntary neural mechanisms regulate respiration. The activities of the medullary respiratory centers, functions of the lower cranial nerves, signals from the chemoreceptors of the carotid and aortic bodies, and actions of the respiratory muscles work in unison to maintain smooth respiration in synchrony. The anatomical and/or functional impairment of any of these alone or in combination poses the risk of respiratory dysfunction in any form. Several of the important mechanisms that play a role, singly or in combinations, in developing respiratory dysfunction in postoperative CM1 patients are considered very briefly below.

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

Abu Obaida ASM, et al., J Surg Res 2024
DOI:10.26502/jsr.10020409

Brainstem compression

Respiratory problems in CM1 patients may ensue from damage to the respiratory centers in the medulla oblongata by compression of the ectopic tonsils, either by direct physical compression or by ischemia from prolonged pressure. This can also result in dysgenesis of neural structures, particularly the reticular activating system [1,7,14-19]. Sudden respiratory deterioration may occur due to direct handling during the surgical procedure. This may result from the loss of respiratory center neurons which are already compromised from congenital deformity or lengthy compression [20,21]. New damage to the brainstem may result from postoperative cerebellar ptosis also. Too large a craniectomy may cause cerebellar ptosis which in turn may lead to further compression on the already sore brainstem and upper cervical cord to cause catastrophic respiratory arrest [22]. Necropsy studies have shown vascular changes as well as ultrastructural malformations in the brainstem of CM1 patients which can be attributed to respiratory dysfunctions in these patients [23]. The overall changes altogether may act in concert to diminish the general responsiveness of the brainstem and ultimately affect the central respiratory drive.

Brainstem clefting

The pressure discrepancy between the cranial and spinal CSF compartments in CM1 tends to disrupt the descending tract of the trigeminal nerve, the nucleus tractus solitarius, and the nucleus ambiguus by creating clefts in the floor of the fourth ventricle. Although the patient may be asymptomatic, the resultant autonomic disturbance may lead to postoperative respiratory depression [24].

Brainstem haemorrhage or infarct

Disordered ventilatory control in CM1 may result from medullary hemorrhages. This may happen from possible venous congestion in the restrained posterior fossa. Occasionally there can be infarction from altered blood supply due to long-standing compression on the medulla compromising its blood flow which may predispose to altered respiratory control [1,10,16,18].

Lower cranial nerve palsy

Dysfunction of the lower cranial nerves, the IX, X, and XI in particular, which maintain the patency of the upper airway makes the CM1 patients vulnerable to obstructive apnea due to stretching of these nerves. Traction of the glossopharyngeal nerve also impairs peripheral chemosensitivity as the afferent input from the carotid bodies is conveyed to the medulla by this nerve and affects respiration. Impairment of lower cranial nerves would increase the risk of aspiration. Consequently, pneumonitis in CM1 patients is another common cause of respiratory distress as the lower cranial nerves are involved in the protective upper airway reflex [1,11,13,16,18,19]. Autopsy findings of damage to the cranial nerve nuclei by compression and stretching of the vagal nerve in CM1 patients also support this [23].

Diminished respiratory reserve

CM1 patients often show very poor respiratory reserve. This can be attributed to the impaired function of the phrenic nerves or the dysfunction of the respiratory centers [11]. The poor tone and weakness of the respiratory muscles resulting from syringomyelia (SM) or scoliosis may also contribute to poor respiratory reserve.

Respiratory muscle weakness

Weak respiratory muscles, another feature related to respiratory problems in CM1 patients, result from the associated SM which mostly compromises the upper cervical spinal cord. The compromised diaphragmatic function and the increased thoraco-abdominal muscle tone play a substantial role in producing respiratory problems in this set of patients [6,11,13,17,25].

Respiratory circuit problem

Dysfunction of the reticular activating system from compression of the brainstem results in the syndrome of afferent respiratory dysfunction in some CM1 patients. The fine adjustments of the respiratory servo mechanisms by the normal facilitatory and modulatory feedback pathways are lacking in these patients causing respiratory problems [11,26].

Loss of chemical influence

Any stretching of the afferent neural pathways of the carotid body chemoreceptors, which act through the IX nerve mainly to carry stimuli from the carotid bodies to the medulla, may lead to the loss of sensitivity of the peripheral chemoreceptors. Ultimately that would manifest as reduced responsiveness to hypoxia or hypercapnia affecting the central respiratory drive to blunt the ventilatory response [14,15,19,27-30]. CSF pressure imbalance may also partially disrupt the nucleus tractus solitarius which acts as the primary relay center to the medulla from the aortic and carotid body chemoreceptors [24].

The syndrome of afferent respiratory dysfunction

Postoperative respiratory deterioration may result from the syndrome of afferent respiratory dysfunction. In this condition, the respiratory servo mechanisms fail to make fine adjustments owing to a lack of normal facilitatory and modulatory feedback pathways. Consequently, despite respiratory dysfunction, preoperatively the gross responses are managed satisfactorily to portray the routine blood gases and vital capacity deceptively within the normal ranges. But, provoking issues like general anesthesia, trauma from surgery, changes in the inspired CO_2 mixture, or narcotics lead to postoperative respiratory deterioration [11].

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

Aspiration pneumonia

Compression and dysfunction of the medulla causing lower cranial nerve palsy leads to loss of pharyngeal sensation and gag reflex. Impairment of the vagally mediated protective upper airway reflex also fails to maintain airway patency, all of which cumulatively aid in aspiration [1,13,25,31]. CM1 patients may also be at risk of pulmonary aspiration leading to aspiration pneumonia from delayed gastric emptying as a result of autonomic dysfunction. Aspiration pneumonia may also result from impaired deglutition due to reduced upper airway reflexes [24].

Alterations in anatomy, physiology, and pathology of the brainstem with changes in the position of the neck

The positional relationship of the brainstem and the tonsils around the CVJ in CM1 is critical throughout the pre, per, and postoperative periods. The herniated tonsils always pose a risk of injury to the brainstem in one way or the other. It seems that the soft tonsils themselves would not harm the brainstem directly much, but the constant pressure that they exert on the brainstem and the disruption of CSF flow and dynamics at the CVJ that they bring about take a toll with time. Additionally, the normal movement of the neck also produces anatomical and physiological changes building a cumulative morbid state endangering the functions of the brainstem.

During surgery of CM1, for the convenience of surgery and adequate access to the CVJ to perform the PFD, the patient is generally placed prone with gentle neck flexion to approximately 1-2 finger breadths between the chin and chest [32-34]. Anatomically, there is no scope for change in the diameter of the foramen magnum in the intact anatomy whatever the patient's position is. However, there are ample possibilities for changes in different parameters of the CVJ with the movement of the neck like in flexion compared to the neutral position. Reductions in the distances between the tip of the dens to the basion, opisthion, and cranial border of the posterior arch of the atlas have been documented during flexion compared to the neutral position. The distances between the basion and both the lower border of the arch of the axis and the most prominent point of the posterior arch of the atlas also lessen during flexion of the neck than in the neutral position [35]. Intraoperative magnetic resonance imaging (iMRI) has demonstrated significant improvement in CSF flow through the foramen magnum dorsal to the tonsils by positioning the patient in the prone position with gentle neck flexion. A mean of 1.0 mm increase of anatomical space dorsal to the cerebellum with CSF flow increase has been recorded by intraoperative cine MRI in flexion, compared to the supine position. However, in the prone position, the anatomical space ventral to the tonsils reduces by a mean of 0.4 mm with a reduction of CSF flow [34]. Brainstem auditory evoked potential (BAEP) can precisely assess the conduction in the dorsolateral regions of the brainstem. With intraoperative BAEP monitoring of the CM1 patients, it has been found that I to V IPLs of BAEP show significantly increased conduction time as the patients are turned prone with gentle neck flexion for surgery. However, significant improvement in conduction through the brainstem is well appreciated in intraoperative BAEP readings just after bone decompression in CM1 patients [32,33]. In addition, respiratory function may often be further jeopardized by the collective effects of laryngeal edema, laryngo-bronchial secretions, and loss of pharyngeal sensation [6,11,13].

Discussion

Most of the respiratory problems in CM1 patients improve following surgery, However, on rare occasions, respiratory arrest following surgery may prove fatal. Therefore, peroperative precautions to take care of the brainstem in CM1 patients during surgery are important in preventing and minimizing respiratory dysfunctions in the postoperative period. In our initial days, we faced the death of 2 patients from respiratory arrest following PFD for CM1. We became very concerned about the respiratory problems and other postoperative complications and tried to find a solution. From our experience, we realized that positioning the patient during surgery, particularly the extent of neck flexion, is a crucial part of the surgery in preventing postoperative respiratory complications.

All the possible pathological mechanisms, singly or in multiple combinations, may result in different kinds of respiratory dysfunctions like central apnea, obstructive apnea, bradypnea, hypopnea, respiratory failure, and even respiratory arrest, both preoperatively and postoperatively. Whatever the pathology behind the spectrums of respiratory problems in CM1 patients is, ultimately all are caused by the dysfunction of the brainstem. The respiratory centers are in the medulla around the CVJ, and CM1 being a pathology around the CVJ has the potential to produce respiratory symptoms in various forms. In CM1, the preexistent jeopardized medulla is constantly exposed to compression of the herniated tonsils and the Valsalva maneuver continually endangers it to repeated hypoxia. If the neck is flexed, even minimally, the already compromised area around the CVJ becomes more vulnerable than in a neutral position as the herniated tonsils compress the brainstem further. The brainstem, harboring the respiratory centers, which is already compromised, is jeopardized more, both mechanically and functionally by positioning the patient in the prone position with the neck flexed, no matter how little that is. The situation worsens further if the flexion is more or is for too long. All these anatomical and physiological alterations play role in unison to impair the function of the already compromised brainstem. These all together hamper the functions of the respiratory

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

centers in the brainstem, particularly the medulla to produce respiratory problems postoperatively if the patient is kept in the prone position with the neck flexed for long during surgery.

Reduction in the lengths of different parameters in combination contributes to the reduction of an overall reduction in the canal diameter of the upper cervical region and around the CVJ in flexion compared to the neutral position, whether be it as part of the natural movement of the neck or be it a part of the positioning of the patient during surgery. The changes in different parameters around the CVJ surely expose the brainstem to extra compression additional to the long-standing posterior compression by the herniated tonsils. Although intraoperative cine MRI demonstrated the increase in CSF flow posterior to the tonsils by positioning the patient prone with flexion of the neck, there is concurrent reduction of the CSF flow anteriorly, ultimately producing compression on the brainstem on the ventral aspect. The bony compression on the brainstem from the front is often ignored but that needs to be considered seriously. Particularly, during flexion of the neck, whether a part of normal movement or a part of the prone position for surgery, the brainstem is kinked against the lower part of the clivus at the anterior margin of the foramen magnum. The brainstem, which is constantly compressed posteriorly by the tonsils is subjected to anterior compression in a prone position exposing it to a double-edged sword whenever there is movement of the neck. The findings of intraoperative BAEP monitoring demonstrate that the brain stem is in a compromised state by the compression of the herniated tonsils at the tight cervico-medullary junction in CM1 patients. The intraoperative BAEP findings confirm that the flexion of the neck during positioning for surgery, further jeopardizes the brainstem during positioning, risking additional neurologic injury. Moreover, the compression on the brainstem exaggerated by positioning the patients in the prone position would always endanger the respiratory centers jeopardizing the respiration of the patient leading to respiratory problems, and even risking death from respiratory arrest. The added compression on the brainstem in the prone position with the neck flexed always would have the risk of ischemia and edema of the brainstem. The ischemia or later edema has the definite possibility of hampering the functions of the respiratory centers. Even if ischemia or edema do not cause immediate respiratory arrest, later they definitely would compromise the functions of the respiratory centers leading to the late development of respiratory problems.

The combined effects of reductions of different anatomical parameters around the CVJ, reduction in space anterior to the brainstem, and reduction in neurophysiological conductivity of the brainstem understandably indicate that there is added compression on the brainstem when the neck is flexed during positioning of the patient for surgery. All these anatomical, physiological, and pathological alterations initiate a 3-pronged orchestrated attack on the already compromised brainstem during flexion. These factors warrant serious attention during the positioning of a patient in the prone position for CM1 surgery. Thus, these can be mitigated or at least reduced to an acceptable and safer range by placing the patient in a neutral position.

For the last few years, considering the anatomical and physiological changes around the CVJ in CM1 patients, we have practiced placing the patients in a neutral position, since we started performing the Stealth cranioplasty which we innovated and described earlier [36-39]. The neutral position, which maintains the neutral anatomy, gives many advantages during and after surgery including minimizing the possibility of postoperative respiratory problems. Over-distraction, over-correction, or torsion of the cervico-medullary junction during positioning the patient for surgery should be carefully avoided to prevent respiratory complications. Endo-tracheal intubations may be challenging and care must be taken not to manipulate the neck much and should be done by the experts with the aid of modern available gadgets. Change in BAEP during flexion of the neck in the prone position is well evidenced. Thus, electrophysiologic monitoring may potentially minimize injury to the cervico-medullary junction during positioning for surgery and moving patients to and from the table perioperatively to reduce respiratory problems effectively. Often extensive PFD is performed. But that does not relieve symptoms always. On the contrary, the extensive PFD may backfire by leading to cerebellar ptosis, which may exaggerate respiratory symptoms from compression on the medulla by the slumped cerebellum. Anticipating any acute worsening of central respiratory control, patients should be considered for observation in an acute care setting postoperatively so that if any emergency arises, that can be managed immediately and effectively. Pre-operative routine screening of respiratory status including sleep apnoea can also reduce the chance of postoperative respiratory complications. Overall awareness among the treating physicians, staff, and caregivers is essential in reducing the occurrences of postoperative respiratory problems.

Conclusion

Chiari malformation 1 with or without syringomyelia is vulnerable to developing respiratory distress, both pre and postoperatively. Respiratory disorders in CM1 patients may result from diverse pathologies. A preoperative meticulous evaluation and peroperative proper positioning of the patients can prevent postoperative morbidity and fatality significantly.

Disclosure of conflict of interest: None

Acknowledgment

We are grateful to our nursing and other staff and the patients.

Funding: None

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

Credit author statement

Abu Saleh Mohammad Abu Obaida: Conceptualization, Data curation, Formal analysis, Writing - original draft, Writing - review & editing

Sheikh Forhad: Data curation, Formal analysis, Writing - review & editing

A.K.M. Khalequzzaman: Formal analysis, Writing - review & editing

Sayem Al Monsur Faizi: Formal analysis, Writing - review & editing

Muhammad Fakhrul Islam: Formal analysis, Writing - review & editing

Abu Naim Wakil Uddin: Data curation, Formal analysis, Writing - review & editing

Md. Ruhul Kuddus: Data curation, Formal analysis, Writing - review & editing

Md. Mohiuddin Biswas: Data curation, Formal analysis, Writing - review & editing

Md. Shamsul Alam: Data curation, Formal analysis, Writing - review & editing

Asifur Rahman: Conceptualization, Data curation, Formal analysis, Writing - original draft, Writing - review & editing

References

1. Yoshimi A, Nomura K, Furune S. Sleep apnea syndrome associated with a type I Chiari malformation. Brain and Development 24 (2002): 49-51.
2. Keefover R, Sam M, Bodensteiner J, et al. Hypersomnolence and pure central sleep apnea associated with the Chiari I malformation. Journal of child neurology 10 (1995): 65-67.
3. Levitt P, Cohn MA. Sleep apnea and the Chiari I malformation: case report. Neurosurgery 23 (1988): 508-510.
4. Ruff ME, Oakes WJ, Fisher SR, et al. Sleep apnea and vocal cord paralysis secondary to type I Chiari malformation. Pediatrics 80 (1987): 231-234.
5. Bullock R, Todd N, Easton J, et al. Isolated central respiratory failure due to syringomyelia and Arnold-Chiari malformation. BMJ (Clinical research ed) 297 (1988): 1448-1449.
6. Gentry JB, Gonzalez JM, Blacklock JB. Respiratory failure caused by Chiari I malformation with associated syringomyelia. Clinical neurology and neurosurgery 103 (2001): 43-45.
7. Fish D, Howard R, Wiles C, et al. Respiratory arrest: a complication of cerebellar ectopia in adults. Journal of Neurology, Neurosurgery & Psychiatry 51 (1988): 714-716.
8. Omer S, Al-Kawi MZ, Bohlega S, et al. Respiratory arrest: a complication of Arnold-Chiari malformation in adults. European neurology 36 (1996): 36-38.
9. Matsumoto T, Symon L. Surgical management of syringomyelia- current results. Surgical neurology 32 (1989): 258-265.
10. Paul KS, Lye RH, Strang FA, et al. Arnold-Chiari malformation: review of 71 cases. Journal of neurosurgery 58 (1983): 183-187.
11. Arora P, Behari S, Banerji D, et al. Factors influencing the outcome in symptomatic Chiari I malformation. Neurology India 52 (2004): 470-474.
12. Chaouch N, Meraï S, Rouhou SC, et al. Insuffisance respiratoire aiguë isolée révélant une malformation d'Arnold-Chiari: À propos de deux observations. Revue de Pneumologie Clinique 63 (2007): 319-322.
13. Doherty M, Spence D, Young C, et al. Obstructive sleep apnoea with Arnold-Chiari malformation. Thorax 50 (1995): 690-697.
14. Smith K, Gomez-Rubio AM, Harris TS, et al. Unusual Ventilatory Response to Exercise in Patient with Arnold-Chiari Type 1 Malformation after Posterior Fossa Decompression. Case reports in pediatrics 16 (2016): 8359838.
15. Cai C, Oakes W. Hindbrain herniation syndromes: the Chiari malformations (I and II). Seminars in Pediatric Neurology (1997).
16. Gagnadoux F, Meslier N, Svab I, et al. Sleep-disordered breathing in patients with Chiari malformation: improvement after surgery. Neurology 66 (2006): 136-138.
17. Hamal D, Fernandes A, Ghimire P, et al. Acute Respiratory Failure Requiring Invasive Ventilation in Adults With Congenital Syringomyelia/Arnold-Chiari Malformations: A Systematic Review. Cureus 16 (2024): e70109.
18. Leu RM. Sleep-related breathing disorders and the Chiari 1 malformation. Chest 148 (2015): 1346-1352.
19. Tran K, Hukins CA. Obstructive and central sleep apnoea in Arnold–Chiari malformation: resolution following surgical decompression. Sleep and Breathing 15 (2011): 611-613.
20. Ono S, Kanda F, Takahashi K, et al. Neuronal cell loss in the dorsal raphe nucleus and the superior central nucleus in myotonic dystrophy: a clinicopathological correlation. Acta neuropathologica 89 (1995): 122-125.

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.

21. Sherman JL, Barkovich A, Citrin CM. The MR appearance of syringomyelia: new observations. American journal of neuroradiology 7 (1986): 985-995.

22. Di X, Luciano MG, Benzel EC. Acute respiratory arrest following partial suboccipital cranioplasty for cerebellar ptosis from Chiari malformation decompression: Report of 2 cases. Neurosurgical focus 25 (2008): E12.

23. Portier F, Marianowski R, Morisseau-Durand M, et al. Respiratory obstruction as a sign of brainstem dysfunction in infants with Chiari malformations. International journal of pediatric otorhinolaryngology 57 (2001): 195-202.

24. Williams DL, Umedaly H, Martin IL, et al. Chiari type I malformation and postoperative respiratory failure. Canadian Journal of Anesthesia 47 (2000): 1220-1223.

25. Quebada PB, Duhaime AC. Chiari malformation Type I and a dolichoodontoid process responsible for sudden cardiorespiratory arrest: case report. Journal of Neurosurgery: Pediatrics 103 (2005): 567-570.

26. Cocito D, Peci E, Garbossa D, et al. Neurophysiological Correlates in Patients with Syringomyelia and Chiari Malformation: The Cortico-Diaphragmatic Involvement. Journal of Clinical Medicine 11 (2022): 50-80.

27. Bauerle L, Rogowski B, Shingala A, et al. Protracted respiratory failure in a case of global spinal syringomyelia and Chiari malformation following administration of diazepam: illustrative case. Journal of Neurosurgery: Case Lessons 6 (2023): 21.

28. Khatib MY, Elshafei MS, Shabana AM, et al. Arnold-Chiari malformation type 1 as an unusual cause of acute respiratory failure: A case report. Clinical Case Reports 8 (2020): 1943-1946.

29. Martinot A, Hue V, Leclerc F, et al. Sudden death revealing Chiari type 1 malformation in two children. Intensive care medicine 19 (1993): 73-74.

30. Zolty P, Sanders M, Pollack I. Chiari malformation and sleep-disordered breathing: a review of diagnostic and management issues. Sleep 23 (2000): 637-643.

31. Stephany JD, Garavaglia JC, Pearl GS. Sudden death in a 27-year-old man with Chiari I malformation. The American journal of forensic medicine and pathology 29 (2008): 249-250.

32. Anderson RC, Dowling KC, Feldstein NA, et al. Chiari I malformation: potential role for intraoperative electrophysiologic monitoring. Journal of clinical neurophysiology 20 (2003): 65-72.

33. Anderson RC, Emerson RG, Dowling KC, et al. Improvement in brainstem auditory evoked potentials after suboccipital decompression in patients with Chiari I malformations. Journal of neurosurgery 98 (2003): 459-464.

34. Bond AE, Jane JA, Liu KC, et al. Changes in cerebrospinal fluid flow assessed using intraoperative MRI during posterior fossa decompression for Chiari malformation. Journal of neurosurgery 122 (2015): 1068-1075.

35. Marinković S, Milić I, Djorić I, et al. Morphometric multislice computed tomography examination of the craniovertebral junction in neck flexion and extension. Folia Morphologica 76 (2017): 100-109.

36. Rahman A. "Stealth Cranioplasty" for Adult Chiari Malformation Type 1: A Philosophical Journey of Innovation, Adaptation, and Evolution. Neurosurgical Procedures-Innovative Approaches: IntechOpen (2019).

37. Rahman A, Ahmed N, Obaida ASMA, et al. Clinical Outcome of Stealth Cranioplasty for Adult Chiari Malformation 1: An Encouraging Experience. Journal of Surgery and Research 7 (2024): 87-95.

38. Rahman A, Rahman MA, Alam MS, et al. Stealth cranioplasty in symptomatic adult chiari 1 malformations: Experience at Bangabandhu Sheikh Mujib Medical University. Bangabandhu Sheikh Mujib Medical University Journal 16 (2023): 8-16.

39. Rahman A, Rana MS, Bhandari PB, et al. "Stealth cranioplasty:" A novel endeavor for symptomatic adult Chiari I patients with syringomyelia: Technical note, appraisal, and philosophical considerations. Journal of Craniovertebral Junction and Spine 8 (2017): 243-252.

Citation: Abu Saleh Mohammad Abu Obaida, Sheikh Forhad, A.K.M. Khalequzzaman, Sayem Al Monsur Faizi, Muhammad Fakhrul Islam, Abu Naim Wakil Uddin, Md. Ruhul Kuddus, Md. Mohiuddin Biswas, Md. Shamsul Alam, Asifur Rahman. Preventing Postoperative Respiratory Problems in Chiari Malformation 1 and the Crucial Role of Peroperative Positioning: Technical Nuances and Philosophy. Journal of Surgery and Research. 7 (2024): 546-552.