



Review Article

An Overview of Ondine's Curse and its Diagnostic Options in the Medical Field

Margret Chandira Rajappa, Palanisamy Pethappa Chetty,
Devi Sowndarya Nagendra Boopathi, Lokeshwaran Sekar, Praveen Kumar Arumugam

Vinayaka Missions College of Pharmacy, Vinayaka Missions Research Foundation (DU), Yercard Adivaram, Saiem, Tamil Nadu, India.

DOI: <https://doi.org/10.24321/0019.5138.202276>

I N F O

Corresponding Author:

Margret Chandira Rajappa, Vinayaka Missions College of Pharmacy, Vinayaka Missions Research Foundation (DU), Yercard Adivaram, Saiem, Tamil Nadu, India.

E-mail Id:

mchandira172@gmail.com

Orcid Id:

<https://orcid.org/0000-0002-6553-0825>

How to cite this article:

Rajappa MC, Chetty PP, Boopathi DSN, Sekar L, Arumugam PK. An Overview of Ondine's Curse and its Diagnostic Options in the Medical Field. *J Commun Dis.* 2022;54(2):89-94.

Date of Submission: 2022-05-27

Date of Acceptance: 2022-06-23

A B S T R A C T

One of the most captivating mythical tales in the realm of medicine is Ondine's curse. Ondine was an eternal water spirit who became human after falling in love, marrying, and having a child with a man. When she saw her husband sleeping with another woman, she cursed him to stay awake to control his breathing, according to one version of the story. The unusual illness marked by loss of autonomic breath control while voluntary breathing remains intact was cleverly called "Ondine's curse" during the nineteenth century. Nowadays, Ondine's curse is most commonly connected with congenital central hypoventilation syndrome; nevertheless, it is also used to describe a variety of respiratory illnesses in the medical literature.

Keywords: Sleep Apnea Disorders, Hypoventilation, Central Sleep Apnea

Introduction

Ondine was a young nymph who fell in love with a mortal man, according to mythology. When she discovered he had been unfaithful to her, she felt outraged and cursed him to stay awake, so that he could regulate his breathing. This ancient myth has become real in the present world and the sharing of the story among people has led to a misunderstanding of this story among medical professionals. The uncommon illness, known as "Ondine's curse", was aptly called so during the 19th century. It is defined by the lack of autonomic breath control while voluntary respiration is unaffected. Congenital central hypoventilation syndrome is now most frequently connected with the phrase "Ondine's curse," although in medical literature, it also refers to some

respiratory conditions like neurological disorder that causes complete brain injury and failure of automatic ventilation. Here, we review the myth with an emphasis on history, arts, and medicine.^{1,2}

Causes of Ondine's Curse

A rare inherited disorder of the autonomic nervous system (ANS) and respiratory regulation is known as congenital central hypoventilation syndrome (CCHS). This condition, once referred to as Ondine's curse is brought on by a PHOX2B gene mutation that impairs neural crest cell growth. When the disease is present from birth, it presents as swallowing issues. Intestinal conditions like Hirschsprung's disease, or cancers like neuroblastoma might also be present in addition to it.³⁻⁶



Epidemiology

According to estimates, there is only one case of congenital central hypoventilation syndrome (CCHS) for every 200,000 live births. The introduction of more thorough screening procedures for the discovery of PHOX2B mutations has nonetheless shown that CCHS is not as uncommon as was first believed.⁷⁻⁹

International statistics say that congenital central hypoventilation syndrome with a PHOX2B mutation affects approximately a thousand children worldwide (CCHS). However, other people think that it is probably too low. There are no racial differences in the prevalence of CCHS. It seems that both genders are equally affected by the diseases.

CCHS is apparent from birth, although the diagnosis may take longer due to differences in the symptoms' severity or a lack of knowledge among doctors, especially in milder cases. Late-onset CCHS may manifest as an aberrant ventilatory response to a severe infection or may follow the administration of an anaesthetic or CNS depressant during a surgical procedure among age groups varying from school-age youngsters to adults.

Treatment

Its treatment is entirely supportive and it is based on the assessment of respiratory impairment, cardiac dysfunction, and gastrointestinal dysfunction, as well as surveillance for underlying oncologic manifestation. There is no cure or gene therapy for CCHS.

CCHS is a condition that mandates lifelong care of children and other people with supportive mechanical measures. The fundamentals of Ondine's curse treatment continue to be respiratory assistance, including mechanical ventilation and occasional tracheostomy.¹⁰⁻¹⁵

Diagnosis

Genetic investigations that show a mutation in the PHOX2B gene and the exclusion of alternative causes of sleep-related hypoventilation are required for the diagnosis of CCHS.

- Genetic testing for a mutation in the PHOX2B gene
- Polysomnography
- Brain magnetic resonance imaging (MRI)
- Chest radiography and computed tomography (CT) scanning
- Diaphragm fluoroscopy, ultrasonography, or both
- Echocardiography
- Comprehensive testing for neuromuscular disorders and inborn errors of metabolism
- Serial monitoring of complete cell blood (CBC) counts
- Blood gas analysis

These are the ancillary techniques for the diagnosis of CCHS.¹⁵⁻²⁰

Study Related to the Disease

Based on the medical literature and papers that were available, as well as the expertise of the Department of Forensic Medicine of the Medical University in Wroclaw, Poland, between 2002 and 2010, a study was conducted with an objective to identify the current Ondine's curse scenarios and definitions circulating among medical professionals and patients. The analysis of archived information made it possible to identify instances in which expert teams recognised the probability of providing patients with severe hypoventilation-related respiratory insufficiency and sleep or awareness issues with the wrong medical care. Current medical knowledge was used to analyse and interpret the selected cases, paying special attention to the most recent definitions of genetic hypoventilation syndromes, iatrogenic illnesses, and intoxication-related ventilation anomalies.²⁰⁻²⁵

Abnormal Breathing Patterns Related to Neurological Dysfunction

The phrase Ondine's curse is used in the neurological literature to describe respiratory abnormalities that arise in individuals with central nervous system dysfunctions. They are largely caused by structural lesions in parts of the brainstem that govern respiratory regulation. This word is frequently used by drug users to describe scenarios in which the consumed chemicals induce respiratory arrest and death. For many years, cigarette lighters were renowned as easy weapons of misuse, although the most prevalent mode of poisoning was ingesting rather than inhaling the fuel. The volatile hydrocarbons that make up lighter fluid, such as benzene, propane-butane, hexamine, lycopene, and naphtha, have recently been discovered to be widely misused, particularly by teenagers. Abusers can inhale them directly from the containers or indirectly via a polythene bag-made tent to attain a high state common to many drugs of abuse. The volatile hydrocarbons can also be extracted from cigarette lighter re-fuelling canisters, which are lawful to carry even by minors, or "compressed air" containers. The latter was meant to remove dust from electronic equipment, but the pressured gas within it isn't pure air; instead, it's a high concentration of different hydrocarbons combined with other inert gases, such as R134a or R152a. It has been observed that in situations of lethal intoxication, seemingly healthy people go into cardiac arrest and die soon after inhalation, whereas habitual abusers may not be affected. In such circumstances, autopsies reveal no distinctive macro or microscopic symptoms, and the cause of death may only be determined by toxicological examination. The inquiry should be based on information gleaned from the patient's medical records. Brain and fat tissue

samples, which are not routinely analysed, are particularly useful in such circumstances. When the circumstances of a death are unclear and a legal or official investigation is underway, such specimens are gathered and kept for further investigations.²⁶⁻³⁴

Interest of Forensic Medicinal Members in the Ondine's Curse for the Cure³⁵⁻⁴⁰

The study of pre- and postoperative anaesthesiology issues has piqued forensic medicine's interest in Ondine's curse. Many medicines used during general anaesthesia, as well as changed gas proportions, are known to impact central respiratory chemosensors and ventilatory chemoreflex. In the practice of forensic medicine, diseases or functional disruptions of respiratory tract organs are frequently blamed for a patient's death, although Ondine's curse instances do not fit well into this restrictive framework. A panel of specialists from the Department of Forensic Medicine of the Medical University in Wrocław, Poland, investigated the cases of several medical mistakes that were chosen to be described in this article. In the years 2002 and 2003, they released two such cases in a row.

The patient, a 28-year-old pregnant lady, arrived at the gynaecology and obstetrics hospital's admittance department in the early stages of a correctly scheduled natural birth, according to the case files. Her last pregnancy had ended due to a scheduled Caesarean section, and this was the major reason for the upcoming cesarean surgery. The patient insisted on being put under general anaesthesia throughout the treatment and refused to accept the choice of subarachnoid or subdural anaesthesia, which an anaesthesiologist on duty strongly recommended as the best alternative. Her calm attitude, weight, and weak chin were all considered barriers to effective general anaesthesia by the anaesthesiologist. Indeed, small issues arose during the placement of the endotracheal tube due to challenges with laryngeal visualisation, which were remedied by employing a Bougie lead. The ensuing caesarean section went off without a hitch. There were no medical difficulties in the operating room, and the life-monitoring gadgets revealed no anomalies. However, due to a previous history of "difficult intubation," the endotracheal tube was not withdrawn immediately after surgery.

After the procedure, the patient was able to wake up for a brief period. She was transported in the horizontal position to a post-operative room, which was only 3 meters away, escorted by the anaesthesiologist, one of the operating room nurses, and a midwife. The anaesthesiologist dropped the patient off at the post-operative room's door and handed the patient's health status card to the midwife, along with the dispositions that needed to be completed in the post-operative room. The helping nurse and midwife

transferred the patient from the trolley to the hospital bed, and the nurse then returned to the operation room. The only member of staff assigned to the post-operative room was the midwife. The alarm went out as soon as she connected the monitoring devices to the patient's body, triggered by a hypoxic arterial blood oxygen saturation of 78%. The midwife attempted to "wake the patient up to make her breathe" by calling her by name and moving her, but she was unable to do so. As directed by the anaesthesiologist, the midwife began passive oxygen treatment by connecting an oxygen line to the endotracheal tube through a connection and adjusting the gas flow rate to 3-4 l per minute.

When the midwife returned her gaze to the patient after switching an empty intravenous drip and checked the type of fluid that should be supplied, she noted that she had become purple and "was swelling rapidly." The midwife disconnected the oxygen line, attached the AMBU bag, and began ventilation right away. The midwife maintained AMBU ventilation because the on-call anaesthesiologist was unavailable at the time, but the patient's health deteriorated, with the abdomen protruding more and more and spotty brown-violet discolourations emerging on the face and upper limbs. The patient was transported to the local intensive care unit (ICU) 3 minutes after experiencing the first symptom of cardiorespiratory collapse. She was diagnosed with subcutaneous emphysema of the chest and abdomen, neck, head, and both upper limbs, as well as massive bilateral pneumothorax and pneumoperitoneum. The air was successfully emptied from both pleural cavities, but the patient remained profoundly comatose, with asymmetric pupils, pathologic neurological reflexes in the lower limbs, and decreased reflexes and muscular tension throughout the body. Brain oedema and multifocal ischemia lesions of the frontal lobes were seen on computed tomography. The patient's life was saved; she was able to recover consciousness and live. She, on the other hand, never entirely healed. Despite months of therapy, she remained physically and psychologically disabled, unable to do even the most basic daily duties and suffering from severe personality and emotional problems.

A result of surgical respiratory insufficiency and acute hypoxia, the above-mentioned case is of life-threatening central hypoventilation. In some ways, this exemplifies the significance of Ondine's curse in clinical and forensic medicine. The first expert opinion in the case stated that an unintentional tracheal rupture was the only cause of the later problems that led to the development of subcutaneous emphysema; the tracheal injury was presumably caused by difficult intubation. This explanation, along with three other similar opinions, did not persuade the prosecutor in charge of the case, since the tests found no tracheal rupture and the case's characteristics remained unexplainable. In

the end, the forensic team was asked to provide further expertise.

The in-depth examination allowed the ultimate interpretation of the case situation to be redefined, as well as the medical staff's obligation as a result. The patient never regained complete consciousness in the operating room after the procedure, and the issues were attributed to the occurrence of Ondine's curse, which occurs when general anaesthesia is used. After a brief time of unassisted breathing, the patient relapsed into unconsciousness with rapidly worsening hypoventilation. Because of the proximity of the two rooms, arterial blood oxygen desaturation was not monitored during the transfer from the surgery to the post-operation room, which took significantly longer than necessary. As a result, the patient arrived in the post-operative room with severe hypoxia, setting off a cascade of events that culminated in the fatal end. Furthermore, further interrogation by the prosecution showed that the midwife had no prior experience at her present job and had just recently returned from protracted maternity leave when the issue happened. She replied in a fairly rational manner, quickly connecting the oxygen line to the endotracheal tube. Unfortunately, in her haste, she made a deadly blunder by connecting the high-pressure oxygen line to the endotracheal tube using a Y-shaped connection, the second Y-arm of which was obstructed by a stopper.

The patient was then inflated with oxygen, resulting in extensive subcutaneous emphysema, bilateral pneumothorax, pneumoperitoneum, and many gas emboli in the brain, resulting in several ischemia foci, particularly in the frontal lobes and brainstem. This connection, on the other hand, was poorly stored in the post-operative area, with no respirator connected. The Y-type connections are commonly used to link endotracheal tubes to respirators, with a side branch used to evacuate the fluid that accumulates in the bronchial tree on occasion, and so the side branch is blocked most of the time. During the installation of one-directional oxygen flow, the patient should have had a T-type connection fitted, with a side branch exposed to the environment to allow the surplus gas to escape. As a result, there were several errors in this case.

The anaesthesiologist broke medical protocol by abandoning a patient who was known to be at high risk of respiratory insufficiency or a return of respiratory depression following surgery. The risk stemmed from the possibility of delayed anaesthetic compound release from the patient's fat tissue, as well as a discrepancy between the anaesthetic elimination rate, which typically has long-lasting effects, and the anaesthetic antidotes administered after surgery to wake the patient up, whose effects wear out much faster. Until the patient regains consciousness, the anaesthesiologist is the primary caregiver. Other co-responsible members

of the medical team, however, were also implicated in the multi-layer systemic medical blunders. The midwife who incorrectly hooked the oxygen line, the chief of nursing staff who failed to teach the midwife at her new job, and the chief of the ward who was aware of defective equipment in use but did not replace it were all among them. All of these individuals should have been better aware of their responsibilities as medical practitioners.

Before the caesarean section, there was no history of apnea or any other type of breathing impairment in the patient mentioned above. After being briefed about the potential complications with intubation, the patient insisted unequivocally on and granted signed approval for, general anaesthesia during labour. It is necessary to give written consent to anaesthesia-related operations for the anaesthesiologist's actions to be lawful.

Symptoms of Ondine's curse, characterised as unexplained hypoxia with sleep disorders, are frequent throughout pregnancy and after delivery. Hypoventilation has been linked to sleep difficulties in a 34-year-old woman in the third trimester of her second pregnancy.²⁵ The pregnancy was hampered by widespread oedema and elevated blood pressure, the same as the previous. These symptoms were followed by sporadic short bouts of apnea, lasting up to 1 minute, beginning in the 25th week of pregnancy. When her spouse detected a protracted spell of breathlessness, he would raise her up. After being awoken, she showed no pathological indications. Apneic episodes got more severe after the 29th week, and she eventually required breathing assistance and an emergency caesarean surgery. Following surgery, the patient was unable to breathe spontaneously for two weeks, following which the breathing issues progressively faded. The patient's initial vaginal delivery had likewise been linked to apnea two years prior. Breathing issues arose shortly after birth, and the patient was intubated; however, the breathing difficulties recovered spontaneously, and the endotracheal tube was withdrawn within 4 hours.

Conclusion

The term Ondine's curse has been attributed to a variety of respiratory dysfunction diseases in medical literature, and its awareness enables early diagnosis and treatment, particularly to avoid unexpected death among infants. During forensic examinations of suspicious causes of death in the postoperative period, the likelihood of spontaneous or iatrogenic problems leading to hypoventilation should be considered. It may be challenging to diagnose CCHS as the cause of death in previously untreated individuals. As a result, cases of unexplained death preceded by breathing disturbances should be thoroughly investigated, including a thorough review of the patient's medical history and all available medical records, a full autopsy, extensive

toxicology screening, and possibly DNA testing for the Phox2b mutation. To rule out medical mistakes, all hypoxia cases resulting from central hypoventilation after surgery conducted under a general anaesthetic should be investigated by forensic medicine.

Acknowledgement

We wish to place on record our heartfelt thanks to our university and our principal, Dr Venkateshwarlu, MPharm (PhD) for having rendered their noble patronage.

Conflict of Interest: None

References

1. Auer RN, Rowlands CG, Perry SF, Remmers JE. Multiple sclerosis with medullary plaques and fatal sleep apnea (Ondine's curse). *Clin Neuropathol.* 1996 Mar;15(2):101-5. [PubMed] [Google Scholar]
2. Barone DA, Chokroverty S. Neurologic diseases and sleep. *Sleep Med Clin.* 2017 Mar;12(1):73-85. [PubMed] [Google Scholar]
3. Chen ML, Turkel SB, Jacobson JR, Keens TG. Alcohol use in congenital central hypoventilation syndrome. *PediatrPulmonol.* 2006 Mar;41(3):283-5. [PubMed] [Google Scholar]
4. Chen ML, Tablizo MA, Kun S, Keens TG. Diaphragm pacers as a treatment for congenital central hypoventilation syndrome. *Expert Rev Med Devices.* 2005;2(5):577-85. [PubMed] [Google Scholar]
5. Valika T, Chin AC, Thompson DM, Kabre R, Lavin JM, Neault SH, Ballard HA, Kenny AS, Weese-Mayer DE. Airway obstruction during sleep due to diaphragm pacing precludes decannulation in young children with CCHS. *Respiration.* 2019;98(3):263-7. [PubMed] [Google Scholar]
6. Fujisawa H, Yoshida Y, Niida Y, Hasegawa M, Yamashita J. Cyanotic breath-holding spell: a life-threatening complication after radical resection of a cervicomedullary ganglioglioma. *PediatrNeurosurg.* 2005;41(2):93-7. [PubMed] [Google Scholar]
7. Gaultier C, Amiel J, Dauger S, Trang H, Lyonnet S, Gallego J, Simonneau M. Genetics and early disturbances of breathing control: the genetics of childhood disease and development: a series of review articles. *Pediatr Res.* 2004 May;55(5):729-33. [Google Scholar]
8. Gaultier C, Trang H, Dauger S, Gallego J. Pediatric disorders with autonomic dysfunction: what role for PHOX2B? *Pediatr Res.* 2005;58(1):1-6. [PubMed] [Google Scholar]
9. Gozal D. Congenital central hypoventilation syndrome: an update. *PediatrPulmonol.* 1998 Oct;26(4):273-82. [PubMed] [Google Scholar]
10. Gross A, Kłys M. [Suicide by propane-butane inhalation: a case report and literature review]. *Arch Med SadowejKryminol.* 2002 Jan;52(1):37-42. Polish. [PubMed] [Google Scholar]
11. Gupta R, Oh U, Spessot AL, Anderson RC, Kaiser MG, Fitzsimmons BF. Resolution of Ondine's curse after suboccipital decompression in a 72-year-old woman. *Neurology.* 2003;61(2):275-6. [PubMed] [Google Scholar]
12. Hamilton J, Bodurtha JN. Congenital central hypoventilation syndrome and Hirschsprung's disease in half sibs. *J Med Genet.* 1989;26(4):272. [PubMed] [Google Scholar]
13. Ishibashi H, Umezawa K, Hayashi S, Shibutani K. Anesthetic management of a child with congenital central hypoventilation syndrome (CCHS, Ondine's curse) for dental treatment. *Anesth Prog.* 2004;51(3):102. [PubMed] [Google Scholar]
14. Juan G, Ramón M, Cisca, MA, García B, Lloret T, Cervelló MA, Marín J. Insuficiencia respiratoria agudacomoprimeramanifestacion de lesiones del tronco cerebral. *Arch Bronconeumol.* 1999;35:560-3. Spanish. [Google Scholar]
15. Kapnadak SG, Mikolaenko I, Enfield K, Gress DR, Nathan BR. Ondine's curse with accompanying trigeminal and glossopharyngeal neuralgia secondary to medullary telangiectasia. *Neurocrit Care.* 2010;12(3):395-9. [PubMed] [Google Scholar]
16. Ios C, Estournet-Mathiaud B, Pinard JM, Cheliout-Hérait F. Sleep disorders caused by brainstem tumor: case report. *J Child Neurol.* 2001;16(10):767-70. [PubMed] [Google Scholar]
17. Matsuyama M, Nakazawa K, Katou M, Ota K, Masuko H, Iizuka T, Mori T, Hayashi H, Hayashihara K, Saito T, Satoh M, Hizawa N. Central alveolar hypoventilation syndrome due to surgical resection for bulbar hemangioblastoma. *Intern Med.* 2009;48(11):925-30. [PubMed] [Google Scholar]
18. Matschke J, Laas R. Sudden death due to central alveolar hypoventilation syndrome (Ondine's curse) in a 39-year-old woman with heterotopia of the inferior olive. *Am J Forensic Med Pathol.* 2007 Jun;28(2):141-4. [PubMed] [Google Scholar]
19. Manning HL, Leiter JC. Respiratory control and respiratory sensation in a patient with a ganglioglioma within the dorsocaudal brain stem. *Am J Respir Crit Care Med.* 2000;161(6):2100-6. [PubMed] [Google Scholar]
20. Marczak M, Pokorski M. Oxygen breathing and ventilation. *J PhysiolPharmacol.* 2003;55(1):127-34. [Google Scholar]
21. Mirkin DB. Benzene and related aromatic hydrocarbons. In: Shannon MW, Borron SW, Burns MJ, editors. Haddad and Winchester's clinical management of poisoning and drug overdose. 4th ed. WB Saunders; 2007. p. 1363-76.
22. Nakajima M, Katsura K, Hashimoto Y, Terasaki T,

- Uchino M. [A case of Ondine curse associated with a medullary tumor]. *RinshoShinkeigaku*. 2000;40(8):811-5. Japanese. [PubMed] [Google Scholar]
23. Niazi AU, Mocon A, Varadi RG, Chan VW, Okrainec A. Ondine's curse: anesthesia for laparoscopic implantation of a diaphragm pacing stimulation system. *Can J Anaesth*. 2011;58(11):1034-8. [PubMed] [Google Scholar]
24. Nowicka J, Kulikowska J, Korczyńska M, Celiński R, Chowaniec C. [Toxicological and medico-legal analyses of sudden deaths resulting from butane inhalation]. *Arch Med SadowejKryminol*. 2011 Jan;61(1):43-6. Polish. [PubMed] [Google Scholar]
25. Ochoa-Sepulveda JJ, Ochoa-Amor JJ. Ondine's curse during pregnancy. *J Neurol Neurosurg Psychiatry*. 2005;76(2):294. [PubMed] [Google Scholar]
26. Pokorski M, Grieb P, Wideman J. Opiate system influences central respiratory chemosensors. *Brain Res*. 1981;211(1):221-6. [PubMed] [Google Scholar]
27. Pokorski M, Morikawa T, Takaishi S, Masuda A, Ahn B, Honda Y. Paralysis of respiratory muscles and hypoxic ventilatory chemoreflex. *Biomed Biochim Acta*. 1989;48(5-6):S573-7. [PubMed] [Google Scholar]
28. Rao GS, Ramesh VJ, Lalla RK. Ventilatory management and weaning in a patient with central hypoventilation caused by a brainstem cavernoma. *Acta Anaesthesiol Scand*. 2005;49(8):1214-7. [PubMed] [Google Scholar]
29. Raslan AM. Percutaneous computed tomography-guided transdiscal low cervical cordotomy for cancer pain as a method to avoid sleep apnea. *StereotactFunctNeurosurg*. 2005;83(4):159-64. [PubMed] [Google Scholar]
30. Severinghaus JE. Ondine's curse: failure of respiratory center automaticity while awake. *Clin Res*. 1962;10:122. [Google Scholar]
31. Sarnoff SJ, Whittenberger JL, Affeldt JE. Hypoventilation syndrome in bulbar poliomyelitis. *J Am Med Assoc*. 1951;147(1):30-4. [PubMed] [Google Scholar]
32. Schläfke ME, Pokorski M, See WR, Prill RK, Loeschcke HH. Chemosensitive neurons on the ventral medullary surface. *Bull Physiopathol Respir*. 1975;11(2):277-84. [PubMed] [Google Scholar]
33. Siewiera J, Trnka J, Kübler A. do zabieguplanowego. *AnestezjologiaIntensywna Terapia*. 2013;45:48-53. Polish. [Google Scholar]
34. Susło R, Trnka J, Siewiera J, Drobnik J. Ondine's curse genetic and iatrogenic central hypoventilation as diagnostic options in forensic medicine. In: Crusio WE, Dong H, Radeke HH, Rezaei N, Steinlein O, Xiao J, editors. *Advances in experimental medicine and biology*. Springer; 2015. p. 65-73. [PubMed] [Google Scholar]
35. Tanaka K, Kanamaru H, Morikawa A, Kawaguchi K. Central hypoventilation syndrome complicated with lateral medullary infarction after endovascular treatment of the vertebral artery dissecting aneurysm: a case report. *NMC Case Rep J*. 2016;3(4):133-6. [PubMed] [Google Scholar]
36. Tomczak E, Wiergowski M, Jankowski Z, Wilmanowska JA. [Dextromethorphan (DXM): new methods of intoxications among teenagers--a case description]. *Arch Med SadowejKryminol*. 2012;62(3):197-202. Polish. [PubMed] [Google Scholar]
37. Trnka J, Gesicki M, Suslo R, Siuta J, Drobnik J, Pirogowicz I. Death as a result of violent asphyxia in autopsy reports. *Adv Exp Med Biol*. 2013;788:413-6. [PubMed] [Google Scholar]
38. Udwiadia ZF, Athale S, Misra VP, Wadia NH. Radiation necrosis causing failure of automatic ventilation during sleep with central sleep apnea. *Chest*. 1987;92(3):567-9. [PubMed] [Google Scholar]
39. Valente S, De Rosa M, Culla G, Corbo GM, Ciappi G. An uncommon case of brainstem tumor with selective involvement of the respiratory centers. *Chest*. 1993;103(6):1909-10. [PubMed] [Google Scholar]
40. Wiesel S, Fox GS. Anaesthesia for a patient with central alveolar hypoventilation syndrome (Ondine's Curse). *Can J Anaesth*. 1990;37(1):122-6. [PubMed] [Google Scholar]