

Severe respiratory failure following ventriculopleural shunt

Shahzad Alam, Nagaraju M. Manjunath

Abstract

Cerebrospinal fluid (CSF) diversion procedure has been used for long to treat hydrocephalus in children. The principle of shunting is to establish a communication between the CSF and a drainage cavity (peritoneum, right atrium, and pleura). Ventriculoperitoneal shunt is used most commonly, followed secondly by ventriculopleural shunt (VPLS). Hydrothorax due to excessive CSF accumulation is a rare complication following both the type of shunts and is more frequently seen with VPLS. We report a case of a 6-year-old female child presenting with massive CSF hydrothorax with respiratory failure following VPLS. The aim of the article is to highlight early recognition of this rare and life-threatening condition, which could easily be missed if proper history is not available.

Keywords: Hydrothorax, ventriculoperitoneal shunt, ventriculopleural shunt

Access this article online

Website: www.ijccm.org

DOI: 10.4103/0972-5229.169359

Quick Response Code:



Introduction

Extracranial shunts are routinely used to divert cerebrospinal fluid (CSF) into the extravascular compartment for the symptomatic relief from hydrocephalus. Ventriculoperitoneal shunts (VPSs) are the most widely used methods for this indication. Though rarely used, today ventriculopleural shunt (VPLS) is considered in patients who have failed VPS or are unsuitable either due to adhesions, infection, thrombosis, or obliteration. Studies have shown VPLS to be effective alternative for draining CSF both in children and adults.^[1,2] We report a case of a 6-year-old female child presenting with massive CSF hydrothorax with respiratory failure following VPLS.

Case Report

A 6-year-old child presented with progressively increasing breathing difficulty for 3 days, there was no fever, cough, or cold. She was being treated with oral antibiotics and salbutamol nebulization by a general

practitioner for 3 days. On examination, the patient had tachypnea with severe respiratory distress, oxygen saturation was 70% which increased to around 90% with oxygen, and air entry was diminished on left side with dullness on percussion. The child had tachycardia with normal blood pressure and perfusion. The child was irritable due to hypoxia, but showed no signs of raised intracranial hypertension. The child was intubated due to hypoxemia and put on mechanical ventilator requiring high positive end-expiratory pressure up to 12 cm H₂O and 100% oxygen initially for maintaining normal saturations. Chest X-ray [Figure 1] showed massive left side pleural effusion with a shunt located on the same side, which was confirmed by ultrasonography. On further enquiry about the history, the child was found to be a patient of congenital aqueductal stenosis who underwent VPS, which was later converted into VPLS due to VPS malfunction. Intercostal drainage tube was inserted immediately, and 1 L of clear colorless fluid

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

From:

Department of Pediatrics, Pediatric Intensive Care Unit, Narayana Hrudayalaya, Bangalore, Karnataka, India

Correspondence:

Dr. Shahzad Alam, Department of Pediatrics, Pediatric Intensive Care Unit, Narayana Hrudayalaya, Bangalore, Karnataka, India.
E-mail: aaalishahzad@gmail.com

How to cite this article: Alam S, Manjunath NM. Severe respiratory failure following ventriculopleural shunt. *Indian J Crit Care Med* 2015;19:690-2.

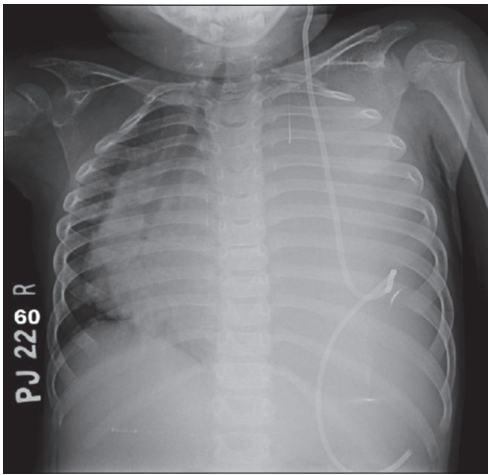


Figure 1: Chest X-ray showing massive left-sided hydrothorax with shunt end in pleural cavity

was drained slowly over the next 48 h. Pleural fluid analysis showed the following results: Cell count-2 (100% lymphocytes), protein-0.2 gm/dl, albumin-0.1 gm/dl, lactate dehydrogenase-70 U/L, and glucose-141 mg/dl. Cytology, gram stain, and culture of fluid were negative proving transudative nature of the fluid. Other causes for transudative effusion were excluded, as serum albumin, liver function, urine examination, and renal function were normal. The child improved following drainage, and repeat chest X-ray showed expanded lungs. Neurosurgery consultation was taken, and imaging of brain was done which showed bilateral VPS with collapse of occipital horns of bilateral lateral ventricle and crowding of posterior fossa structures suggestive of intracranial hypotension secondary to CSF overshunting. The child was extubated successfully on the 2nd day as ventilator requirements decreased and sensorium improved. VPLS was later exteriorized at the distal end as external ventricular drainage and intercostal drain were clamped and removed. A new VPS was inserted on the same side disconnecting the old one at previous incision site, and the old VPLS was removed. The child was monitored for any complications post surgery for 3 days and was finally discharged on 3rd postoperative day.

Discussion

In patients with hydrocephalus, CSF can be shunted in any body cavity capable of absorbing CSF (peritoneum, pleura, and atrium). VPSs are preferred over others due to the large absorption surface of the peritoneal lining, ease of insertion, and the low complication rate, but there are some instances where other modalities such as ventriculopleural or ventriculoatrial shunts have been preferred, especially in patients with active inflammation of peritoneum, adhesions due to past surgery, ascites,

peritoneal dialysis, and failure of prior VPS.^[3] In our case, the reason to insert a VPLS was the failure of previous VPSs. VPLSs are commonly associated with pleural effusion, but these are usually small and does not require any interventions and are treated conservatively.^[4] A small asymptomatic pleural effusion is typically visible on the chest X-ray, indicating that the VPLS is in action, but does not imply that it is dysfunctional.^[3] The ability of pleural surfaces to absorb any accumulated fluid within the pleural cavity partially determines the occurrence and the degree of pleural fluid accumulation. Fluid accumulates if the rate of fluid formation exceeds the rate of absorption. Large hydrothorax due to excess CSF accumulation in the pleural cavity has been reported although rarely.^[3,5,6] Two hypotheses can explain the hydrothorax complicating the VPLS: (a) Impaired pleural absorptive capacity, due to pleural damage secondary to prior infection and/or chronic exposure to CSF and (b) excessive drainage of CSF into the pleural space.^[3,7]

Shunt over drainage is seen in 10–12% of patients with VPS and may manifest as slit ventricle syndrome, intracranial hypotension syndrome, subdural fluid collections, craniosynostosis, ventricular compartmentalization, and cerebellar tonsillar herniation.^[8] The symptoms of over drainage can be very similar to those of underdrainage, though there are important differences. Headaches, dizziness, and fainting occur and are often worse after getting up from lying down, whereas the headaches caused by high CSF pressure are often worse on waking, before rising in the morning. Intracranial hypotension syndrome is usually a late complication reported 5–17 years after the shunt placement and commonly affects adolescents and older patients and presents with triad of postural headache, diffuse pachymeningeal gadolinium enhancement, and low CSF opening pressure.^[9,10] We hypothesized that our patient might have VPLS malfunction leading to CSF over drainage as suggested by the magnetic resonance imaging brain although our patient did not show any signs of intracranial hypotension or it may be a consequence of decreased pleural absorptive surface, which lead to gradual accumulation of CSF. Rapid drainage of hydrothorax could also lead to sudden drop of CSF pressure in brain and cause intracranial hypotension syndrome, but this complication was avoided by slow drainage of CSF, in our patient, over 48 h and later VPLS was exteriorized at significant height to avoid intracranial hypotension.

In our case, there was a delay in diagnosis by the practitioner, which led to slow accumulation of CSF

in pleural space leading to severe life-threatening massive hydrothorax. Diagnosis at our center was also delayed initially due to lack of proper history and also as the initial emphasis was on the management of severe life-threatening episode and chest X-ray could only be done after intubation, which was inevitable at the situation. The child was later successfully treated with drainage of CSF and later conversion of VPLS to VPS. The message conveyed by this case report was to emphasize getting early chest X-ray with the first sign of respiratory distress in these patients with VPLS to avoid such severe life-threatening episodes as a complication of shunt malfunction.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Jones RF, Currie BG, Kwok BC. Ventriculopleural shunts for hydrocephalus: A useful alternative. *Neurosurgery* 1988;23:753-5.
2. Piatt JH Jr. How effective are ventriculopleural shunts? *Pediatr Neurosurg* 1994;21:66-70.
3. Küpeli E, Yılmaz C, Akçay S. Pleural effusion following ventriculopleural shunt: Case reports and review of the literature. *Ann Thorac Med* 2010;5:166-70.
4. Hoffman HJ, Hendrick EB, Humphreys RP. Experience with ventriculo-pleural shunts. *Childs Brain* 1983;10:404-13.
5. Beach C, Mantley DE. Tension hydrothorax due to ventriculopleural shunting. *J Emerg Med* 1998;16:33-6.
6. Irani F, Elkambergy H, Okoli K, Abou DS. Recurrent symptomatic pleural effusion due to a ventriculopleural shunt. *Respir Care* 2009;54:1112-4.
7. Grunberg J, Rébori A, Verocay MC, Ramela V, Alberti R, Cordoba A. Hydrothorax due to ventriculopleural shunting in a child with spina bifida on chronic dialysis: Third ventriculostomy as an alternative of cerebrospinal diversion. *Int Urol Nephrol* 2005;37:571-4.
8. Weprin B, Swift D. Complications of ventricular shunts. *Tech Neurosurg* 2002;7:224-42.
9. Paldino M, Mogilner AY, Tenner MS. Intracranial hypotension syndrome: A comprehensive review. *Neurosurg Focus* 2003;15:ECP2.
10. Park ES, Kim E. Spontaneous intracranial hypotension: Clinical presentation, imaging features and treatment. *J Korean Neurosurg Soc* 2009;45:1-4.