Complications Specific to Pleural Type of CSF Shunt

15

Marcelo Galarza and Patricia Martínez

Contents

Abbreviations

CSF	Cerebrospinal fluid
VPS	Ventriculoperitoneal shunt
VAS	Ventriculoatrial shunt
VPL	Ventriculopleural shunt

15.1 Introduction

When ventriculoperitoneal shunt (VPS) or ventriculoatrial shunt (VAS) is no longer suitable or is not an initial valid alternative for cerebrospinal fluid (CSF) diversion, VPL is usually the next option to consider. VPL drainage is seldom considered as an alternative route for CSF drainage owing to the feared complications of pleural effusion [2, 6, 7]. However, some series document the feasibility and safety of VPL and subdural-pleural shunting [1, 16, 21, 34, 39, 40, 45]. A few authors have even used VPL shunting as the first option for treating hydrocephalus [39]. The most frequent complication of pleural shunts is symptomatic tension hydrothorax causing respiratory distress [1, 2, 6, 43]. Some reports argue that pleural effusion is an age-related event [46]; accordingly the use of VPL shunting has been discouraged especially

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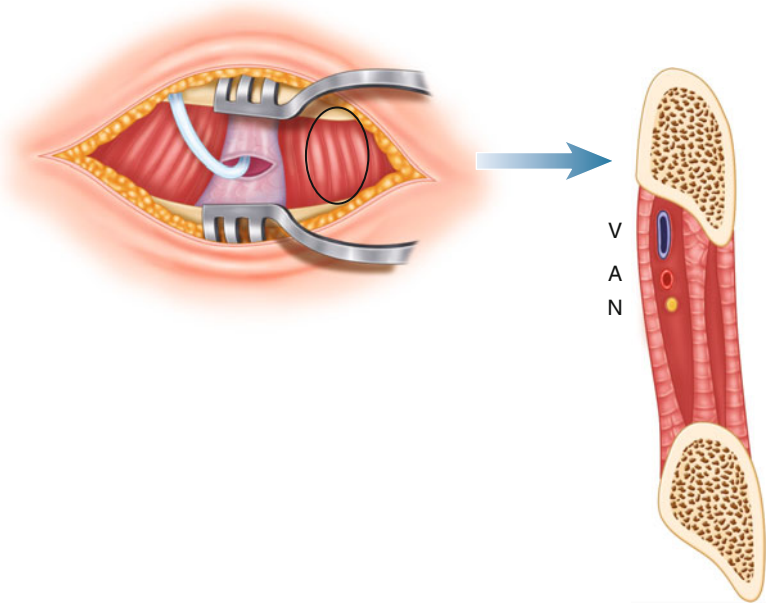

Fig. 15.1 Drawing showing the pleural step of a VPL shunt technique. A small transverse skin incision is made in the third to the fifth anterior intercostal space, and the distal catheter of the shunt is passed to the thoracic incision through the subcutaneous tissue. The pectoralis major and the intercostal muscles are split to expose the pleura. Care is taken to avoid the costal nerve, according to the rib anatomy (*n* nerve, *a* artery, *v* vein)

in children [33]. In contrast, other authors have demonstrated that the diversion of CSF to the pleural cavity constitutes a valid alternative in children as young as 3 years old [21, 34].

Insertion technique of VPL is essential to avoid initial complications and to prevent near future malfunctions. Our technique is not different to what is previously reported [31]. All children are operated on under general anesthesia and with orotracheal intubation. The cranial part of the surgical procedure did not differ from the techniques commonly used for peritoneal shunting. The ventricular catheter is introduced posterior parietal-occipital, inferior and posterior to the parietal boss, and well away from the sensorimotor cortex, with the tip being directed toward the frontal horn. After shaving, the patient is placed with the neck extended and the head turned away from the side to be shunted. Shunts are usually placed on the right side to avoid the dominant hemisphere areas. The scalp incision is made through the skin and galea, and a C-shaped flap is centered on the chosen burr hole site. The scalp is held open by a self-retaining retractor. A tunneling device is used to create a subcutaneous tunnel between the cranial and the thoracic incisions. The chosen shunt is passed into the subcutaneous tunnel, and the tunneler is removed. A small transverse skin incision is made in the third to the fifth anterior intercostal space. The distal catheter of the valve is passed down from the cranial to the thoracic incision through the subcutaneous tissue. Care is taken to avoid the costal nerve, according to the rib anatomy (Fig. 15.1). The costal nerve, if injured, can later develop chronic neuritis. The pectoralis major and the intercostal muscles are split to expose the pleura. The anesthetist deflates the lungs, and the parietal pleura is incised for a length of approximately 3 mm. At this point, the ventricular catheter is inserted, and a good flow of CSF confirms appropriate placement. The CSF pressure is checked by a manometer connected to the ventricular catheter if necessary. The CSF is collected for cell count, Gram's stain, protein, and glucose. The ventricular catheter is cut to an appropriate length and connected to the distal valve and tubing. The CSF flow is checked at the distal end. The catheter is then finally introduced into the pleural cavity. The lungs are inflated, the muscles approximated, and the skin closed. A chest radiograph was taken at the end of the operation. The patient is observed in the pediatric intensive care unit for 6 h and then returned to the neurosurgical ward. Children are discharged home on the fifth to seventh day. A repeat chest

radiograph and a computerized tomography scan are performed before discharge.

Alternatively, the transdiaphragmatic route was reported by Rengachary in [42], as a substitute path to the peritoneum through the pleural cavity. We do not have experience with this route.

From a historical perspective, the VPL was initially described by Heile in [15]. An initial attempt to drain CSF into the thoracic duct and pleural cavity was reported by Ingraham and Sears [18]. Later, a series of VPL by Ransohoff in 1954 [40] and 1963 [41], Fein and Rovit [8], Venes (1979) [45] and Hoffmann (1983) [19] gave support to this option. Nonetheless, some reports on complications of VPL shunting have obscured the benefits of its use in daily clinical practice.

Although reported early, the first up to standard series of VPL shunting in the management of hydrocephalus was reported by Ransohoff in 1954. He pointed out that the procedure was relatively simple and that the pleural surfaces tended to absorb CSF well. Pleural effusions were not a significant problem in the initial six patients that he reported.

Nixon in 1962 [37] indicated that many surgeons had encountered problems, particularly pleural effusions, with VPL shunts and recommended the use of valves to avoid that complication, with good results in three patients.

The series of Jones et al. included, from 1969 to 1979, an initial series of 29 children, and later, from 1979 to 1982, a further series of 52 other patients received VPL shunts.

In 1979, Venes and Shaw described their technique for insertion of a pleural shunt, using a trocar to pass the tube to the pleural cavity. They mentioned a 10–20 % risk of a pneumothorax following VPL shunt insertion.

Further serial cases were reported by Hoffman et al. [16], giving support to this option, although not exempted from complications.

Megison and Benzel [33] have carried out a retrospective study of 88 pleural shunting procedures. There was a 7 % complication rate related to the use of the pleural space as the shunt finishing point. Complications at the pleural end included shunt obstruction, either functional or structural; pleural effusion; pneumothorax; and other technical problems. There were no deaths associated with shunt dysfunction or other complications. VPL shunting for hydrocephalus, when used with appropriate precautions and with careful patient selection, is a viable alternative for the treatment of adult hydrocephalus. Although the complications that are unique to this procedure are pneumothorax and pleural effusion, they were encountered infrequently in this series. The authors concluded that VPL shunting may be indicated when other routes are not available.

Piatt [39] reported that the survival of simple ventriculopleural shunts in his series was not significantly different from that of simple ventriculoperitoneal shunts in patients of comparable age with a comparable recent shunt revision history.

Table 15.1 shows the listed cases of reported complications in the literature, including cases from our personal series.

From here, we will discuss the possible complications that we may find with this technique.

Table 15.1

Associated complications in the pleura related to hydrocephalus and to pleural shunts	Reference
Tension pneumothorax	[16]
Tension hydrothorax	[2]
Tension hydrothorax related to subdural-pleural shunt	[6]
Recurrent pleural effusion	[33]
Empyema	[19]
Disconnection	[39]
Migration	[20]
Dislodged/coiling	[34]
Pleural adhesions	[45]
Peritoneal effusion	[47]
Costal neuritis	Personal series
Cerebrospinal fluid galactorrhea	[35]
Hydrothorax related to ventriculoperitoneal shunt	[13]
Pleurisy with clear liquid due to ventriculoatrial shunt	[10]
Infarction pneumonia due to ventriculoatrial shunt	[44]
Cardiac tamponade and heart failure	[50]
Fibrothorax child	[49]
Glial tumor metastases	[46]
Fibrothorax adult	[22]

15.2 Pneumothorax and Subcutaneous Emphysema

A pneumothorax (pl. pneumothoraces) is an abnormal collection of air or gas in the pleural space that separates the lung from the chest wall and which may interfere with normal breathing. Pneumothorax is also defined as the presence of any air inside the chest, and it can be made during the procedure of inserting the catheter through the thorax wall to the pleural cavity (Fig. 15.2). Subcutaneous emphysema develops when the air migrates into the subcutaneous space. It may also be found in the physical examination of patients with serious pulmonary disease.

Usually the surgical technique employs either a thoracoscopic approach [26] or, usually, an intercostal incision to introduce the catheter inside the thorax. This latter technique may predispose for the development of a pneumothorax.

15.2.1 Prevention

Prevention is related to anesthesia events. Continuous feedback within the surgical and anesthetist team is essential. General anesthesia with orotracheal intubation, with no attempts to spontaneous breathing in order to avoid cough, avoiding nitrous oxide and providing hand ventilation to deflate the lung when the surgeon enters the parietal pleura are anesthetic management details for preventing complications such as pneumothorax and subcutaneous emphysema [14].

A positive pressure sustained ventilation administered by the anesthesiologist is used during approximation of the previously spread muscles with a single absorbable stitch. This minimizes the retention of air in the pleural space [33].

In general, no purse-string suture is needed at the pleural entry site [31, 38].

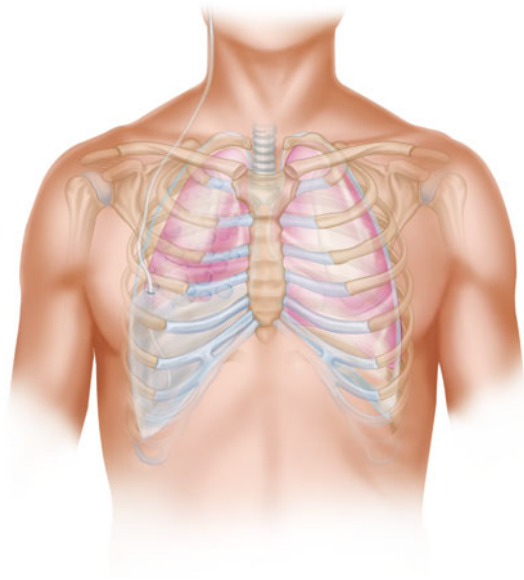

Fig. 15.2 Schematic drawing showing a pneumothorax or presence of any air inside the chest

15.2.2 Identification

Physical exploration reveals crepitus at palpation, if there is associated subcutaneous emphysema, and thoracic auscultation shows decreased breath sounds. Chest radiograph is the standard procedure for the diagnosis of pneumothorax. It should be upright and preferably in the posteroanterior projection. If the patient cannot be upright, a lateral decubitus view with the suspect side positioned up may be helpful. Radiographs obtained in exhalation may accentuate the pneumothorax, but most of the thoracic surgeons have not found this technique useful enough in most clinical situations to warrant the double radiographic exposure. In general, the percentage of collapse is underestimated with the X-ray chest. It is feasible to find air in the subcutaneous space at X-ray when subcutaneous emphysema appears.

Computed tomography of the lungs gives an excellent evaluation of pneumothorax, but the cost-effectiveness of such a procedure must be questioned [28].

The physiologic consequences of a pneumothorax range from little, such as 10 % of collapse in a young person, to life-threatening, such as tension pneumothorax in an older patient with an already compromised cardiopulmonary function aggravated by mediastinal shift and compression of the contralateral lung [9].

15.2.3 Management

A small pneumothorax in a healthy patient can be observed and followed until its reabsorption. Supplying extra oxygen to such patients theoretically hastens the resolution of the pneumothorax, but the true cost-effectiveness of such treatment can be questioned. Kircher and Swartzel [23] estimated that 1.5 % of the air is reabsorbed over each 24-h period.

Simple aspiration is particularly useful in a smaller pneumothorax with a delayed diagnosis, when the passage of time suggests that the process will be self-limited.

Thoracocentesis is indicated when a wait and see conduct is not an option. A tube thoracostomy should be carried out for pneumothoraces over 30 % to hasten recovery or in cases of lesser degrees of lung collapse for those patients with symptoms of associated disorders, such as heart or chronic pulmonary disease. We prefer to insert tube thoracostomy of 24–28-F catheter directed toward the apex. Rarely, this iatrogenic pneumothorax requires a more aggressive surgical option, like videothoracoscopic approach or even thoracotomy. The complications of thoracocentesis include aggravation of pneumothorax, which may be seen in 3–20 % of patients, hemothorax, pulmonary edema, intrapulmonary hemorrhage, and hemoptysis. Other rare events are vagal inhibition, air embolism, subcutaneous emphysema, bronchopleural fistula, empyema, seeding of a needle tract with malignant cells, and puncture of the liver or spleen.

Subcutaneous emphysema is directly related to the pneumothorax and usually resolves itself when pneumothorax is resolved [9]. There is no need for a specific approach.

15.3 Pleural Effusions, Empyema, and Fibrothorax

The presence of a pleural collection of fluid observed on chest radiographs of patients with VPL shunting (Fig. 15.3a) should be of no concern in the absence of respiratory symptoms [21]. In our opinion [31], the finding of small hydrothoraces in otherwise asymptomatic patients indicates that the shunt is functioning. However, we consider that patients with VPL shunts must be regularly followed up in view of the reports on

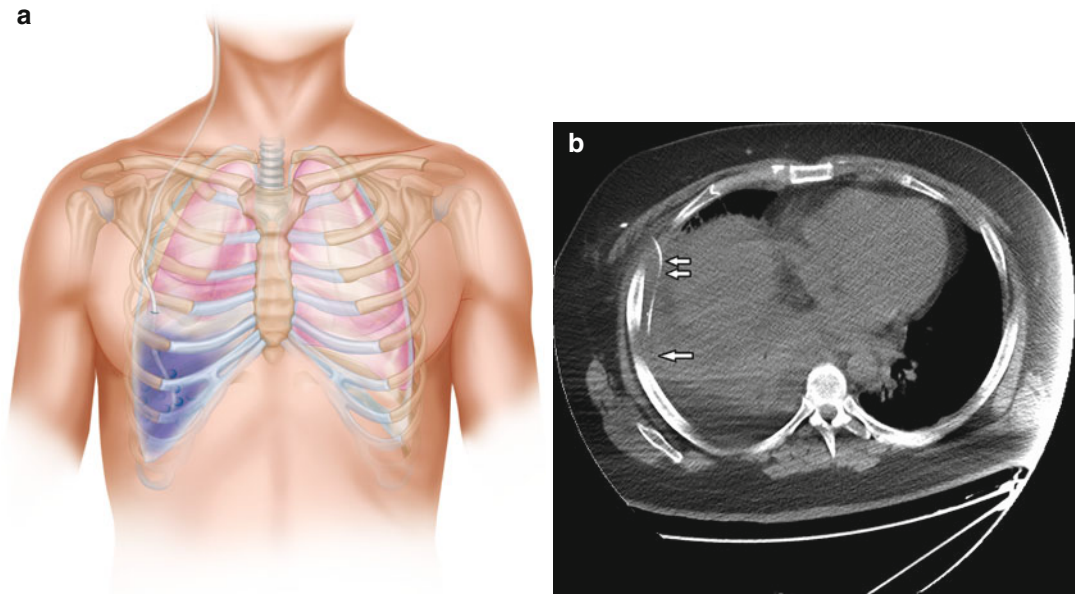

Fig. 15.3 (**a**, **b**) Pleural collection of fluid observed on the chest radiographs of patients with VPL shunting is shown in this schematic drawing. Fluid collection (*single arrow*) secondary to CSF drainage of a VPL shunt in the pleural space depicted in this thorax computed tomography. The distal catheter is in close relationship (*double arrow*)

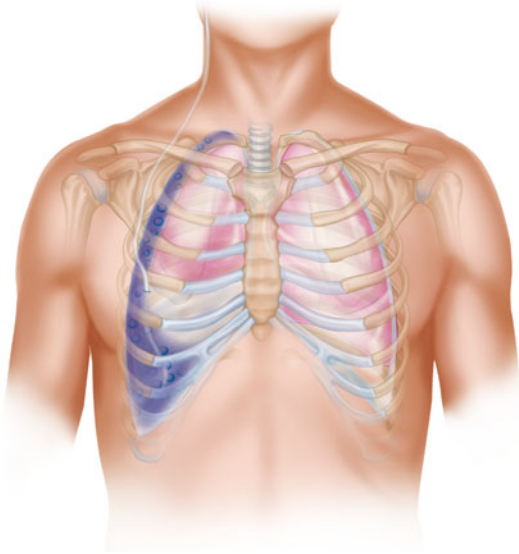

Fig. 15.4 Schematic drawing showing a symptomatic tension hydrothorax causing respiratory distress

tension pleural effusions that can appear at any time, as a result of changes in the valve pressure or in the absorption capability of the pleural cavity [21, 25]. Still, the most frequent blunt complication of pleural shunts is symptomatic tension hydrothorax causing respiratory distress [2] (Fig. 15.4).

When pleural effusions are not resolved, fibrotic changes may develop. However, severe fibrotic change in the pleural cavity is an unusual complication of ventriculopleural shunts, and only two cases of a fibrothorax have been described [22, 49]. Although these cases do not warrant abandonment of the pleura as a potential site for CSF drainage, they raise awareness about this complication.

The development of fibrotic changes in the pleura is believed to be related to the chemical composition of CSF, an immune-related mechanism, or on the other hand, related to a low-grade infection. The length of time required to produce these severe changes appears to be quite variable. It is not clear whether the antisiphon devices will be able to prevent this complication, and a long-term follow-up of patients with these devices may help detect the development of fibrosis.

15.3.1 Prevention

Megison and Benzel [33] warned about the use of VPL shunting in adults with pulmonary disease. The ventilator reserve must be considered with particular care especially in meningomyelocele patients with kyphoscoliosis and in cases of Chiari malformation. In all these cases the burden of a pleural fluid effusion in an already-restricted ventilatory capacity might precipitate a frank respiratory failure [39].

Jones et al. [21] have achieved the use of an antisiphon device connected with the valve, with the aim of preventing the formation of clinically significant CSF pleural effusions. The authors reported that only 1 of 52 children developed a symptomatic hydrothorax that required conversion to a ventriculoperitoneal shunt

Nevertheless, the use of antisiphon devices in children frequently induces symptoms of underdrainage, owing to the narrow margins that these devices achieve for effectively controlling raised intracranial pressure [31].

Ventriculopleural shunts can be associated with debilitating scenarios, and caution should be exerted in their use in debilitated or immunocompromised patients, to avoid the occurrence of severe fibrotic changes in the pleural cavity with lung entrapment [22].

The general management consists of evaluation of pleural fluid and its drainage, supplemental oxygen, intravenous volume replacement, and the consideration of empiric antibiotics to cover coexistent infections. A comprehensive shunt evaluation with careful observation or revision of the ventriculopleural shunt is recommended. Removing the intrapleural shunt catheter must be considered, only if an infection has been demonstrated.

15.3.2 Identification and Specific Management

15.3.2.1 Pleural Effusion

Fluid collection in the pleural space decreases lung volume, increases intrathoracic pressure, and leads to irritation and chest pain (Fig. 15.3b). On

Table 15.2 Mechanisms of pleural effusions

Increased systemic hydrostatic pressure
Decreased oncotic pressure in the microcirculation
Increased pleural microcirculation permeability
Increased pulmonary interstitial fluid
Obstruction of lymphatic drainage
Flow of liquid from walls or other sources: peritoneum, retroperitoneum, cerebrospinal space, external catheters
Reduction of the negative pressure in the pleural space
Thoracic vascular rupture
Rupture of the thoracic duct

examination, patients may demonstrate tachypnea, hypoxia, hypotension, dullness to percussion of the involved hemithorax, decreased chest wall expansion, and jugular venous distention.

Pleural fluid accumulates relatively slowly, allowing for the body to compensate with alterations in intravascular volume and reflex tachycardia [25].

Once a critical volume and pressure has been reached within the hemithorax, decreased venous return and hypotension ensues [48].

Pleural effusions develop due to the alterations in dynamics of net pleural fluid production and absorption. There are many possible mechanisms involved (Table 15.2). The mechanisms for accumulation of pleural effusions with ventriculopleural shunts remain speculative. The presence of a shunt catheter in the pleural space may produce local irritant effects, inducing a chronic subclinical inflammatory response, as supported by the predominant lymphocytosis in the pleural fluid. Inflammation leads to increased pleural fluid production and impaired lymphatic flow, causing pleural fluid accumulation and lung collapse. This further reduces the pleural surface area, resulting in a decrease in the net absorption of the pleural fluid. Continuous addition of cerebrospinal fluid compounds the problem, leading to rapid accumulation of large pleural effusions [2].

Beta-2-transferrin level has been used as a novel diagnostic strategy. A sample of fluid, drained via tube thoracostomy, was sent for beta-2-transferrin level. This desialated isoform of transferrin is almost exclusively found in the CSF with only minimal amounts present in cochlear perilymph and in the aqueous and vitreous humor of the eye. Multiple studies have validated the use of beta-2-transferrin as a specific marker for CSF leakage with sensitivity and specificity approaching 100 and 95 %, respectively. Furthermore, Huggins and Sahn [17] report the use of beta-2-transferrin to identify the presence of a CSF pleural effusion in an elderly patient with a duropleural fistula. According to the authors, beta-2- transferrin has never been previously utilized to identity CSF hydrothorax in children with VP shunts.

Increased densities on chest radiography are frequently attributed to parenchymal infiltrates when they actually represent pleural fluid. Free pleural fluid is best demonstrated with lateral decubitus chest radiography, ultrasonography, or computed tomographic scans. The presence of loculated pleural fluid is best demonstrated with ultrasonography [9].

Thoracocentesis with a needle aspiration of the pleural fluid under local anesthesia may be indicated. If the fluid appears benign, is not loculated, and can be removed totally or nearly so, thoracocentesis may be all that is necessary to control the disease process. Leukocyte count, Gram stain, cultures, cytological studies, and glucose and LDH levels of the fluid should be carried out. If negative, resolution usually occurs. If the fluid is positive by biochemical evaluation, staining, or culture, pleural effusion has turned to an empyema, and close chest tube drainage is required [30].

Biochemical and microbiological analyses of pleural fluid can demonstrate that there is an intrapleural complication such as empyema. The appearance of pleural fluid and the levels of glucose, LDH, pH, and proteins (see Light's criteria [29]) with white blood cell count and the presence of microorganisms in the pleural fluid (Gram's stain) or in specific growth culture could indicate a complicated exudative pleural effusion that requires to be drained with a tube of thoracostomy.

An uncomplicated effusion is nonpurulent, has a negative Gram's stain result for bacteria, has negative results by culture, and is generally free flowing. Using biochemical parameters, Light and associates noted a pH greater than 7.30, a normal glucose level, and an LDH concentration less than 1,000 IU/L.

In case of loculated pleural effusion, the administration of intrapleural fibrinolytic agents such as urokinase may be required. Although chest tube drainage combined with enzymatic debridement is effective, some authors [24, 27, 29] have reported better results with the videothoracoscopic approach, reporting a higher success rate, shorter hospital stay, and less overall economic costs.

In our experience, a video-assisted thoracoscopy procedure for treating pleural effusion related with VPL shunts is rarely needed.

Rarely, VP shunts may develop pleural effusions. The tip of a VP shunt may be located intraabdominally, usually in the suprahepatic area or the pelvis. One case has been reported of a suprahepatic shunt perforating the diaphragm with a CSF collection in the pleural cavity. Still, according to some authors, VPL shunts are rarely used because of frequent pleural effusions, which may cause a decrease in the effectiveness of CSF shunts and respiratory compromise [12].

Third ventriculostomy was used as an alternative of cerebrospinal diversion in a case of hydrothorax due to ventriculopleural shunting in a child with spina bifida on chronic dialysis [11].

Acetazolamide was used to decrease cerebrospinal fluid production in chronically ventilated patients with ventriculopleural shunts [4].

Tonn et al. [44] reported a rare but life-threatening complication of ventriculoatrial shunt as infarction pneumonia and pleural effusion. They presented the case of a female patient with ventriculoatrial shunt insertion as long-term treatment for aqueductal stenosis who presented with recurrent episodes of dyspnea, chest pain, and unilateral pleural effusion. Diagnostic evaluation revealed a positive D-dimer test, bilateral basal infiltrates, and pleural effusion. Transesophageal echocardiography established the diagnosis of a thrombus in the right atrium. Laboratory testing for thrombophilia revealed a homozygous factor V Leiden mutation. A shunt revision was performed and resolved the malfunction.

A similar case was reported by Gerbeaux et al. in 1977 [10].

Specific management of pleural effusion that consists of a needle thoracocentesis is needed when a clinical infection is suspected, or it could be performed only if the pleural effusion measures more than 10 mm on lateral decubitus chest radiography [32].

15.3.2.2 Tension Hydrothorax

Tension hydrothorax is an emergency condition that requires prompt recognition and treatment to maximize chances for survival. The presentation of a hydrothorax is often subacute due to the slow accumulation of fluid in the hemithorax (Fig. 15.4). Hypotension, hypoxia, and tachycardia are signs of clinical decompensation and indicate that the collection of fluid in the pleural space has impaired cardiopulmonary function.

Davidson and Zito [6] reported also an acute tension hydrothorax in a patient with subdural-pleural shunting. He reported a 3-year-old child with a bilateral chronic subdural hematoma treated successfully with a subdural-pleural shunt. Two months later, the patient returned with severe respiratory embarrassment due to cerebrospinal fluid hydrothorax. By removing the shunt the hydrothorax was resolved.

Hadzikaric et al. [13] reported a case of cerebrospinal fluid tension hydrothorax as a rare complication of ventriculoperitoneal shunt. The case was a 16-month-old boy known to have congenital hydrocephalus and a Dandy-Walker cyst who presented with serious respiratory distress. Examination revealed right pleural effusion and congested throat. Thoracocentesis with drainage of the pleural cavity for 10 days failed to free the patient from pleural effusion. Following an intraperitoneal injection of Omnipaque, a chest X-ray was done, and samples of pleural fluid taken before and after the injection were compared on X-ray, revealing the presence of contrast in the postinjection pleural effusion. Changing the VP shunt for a ventriculoatrial shunt resulted in immediate complete disappearance of the pleural effusion and of the patient's chest symptoms. This was a rare occurrence.

Specific management consists when a tension hydrothorax must be treated immediately with a tube thoracostomy when clinical decompensation is noted. Emergency physicians should be aware of this potential complication and consider

it in their differential diagnosis for cardiopulmonary distress in patients with a ventriculopleural shunt [48].

15.3.2.3 Thoracic Empyema and Fibrothorax

A thoracic empyema occurs when bacteria invade the normally sterile pleural space. The process was described by Andrews in 1962 (cited by Light [29]) as a continuum of three stages. Stage 1 is characterized by the presence of an exudative effusion from increased permeability of the inflamed and swollen pleural surfaces. This stage corresponds to the uncomplicated effusion and is initially sterile.

With bacterial invasion, the process blends into fibrinopurulent stage 2, true empyema or Light's (Light 1980 [30]) complicated pleural effusion; it is composed of white blood cells greater than 500 cells per μL, protein level greater than 2.5 g/dL, pH less than 7.2, and LDH levels greater than 1,000 IU/L. Heavy fibrin deposition takes places on both pleural surfaces, particularly the parietal pleura. The effusion becomes purulent, with a white blood cell count above 15,000 cells per μL. Biochemically, the pH decreases to levels below 7.0, the glucose decreases to less than 50 mg/dL, and the LDH increases to greater than 1,000 IU/L.

Stage 3 begins as early as 1 week after infection with collagen organization and deposition on both pleural surfaces and entrapment of the underlying lung. This process is mature in 3–4 weeks, and the organized collagen on the pleural surface is termed a peel [32].

Usually, thoracic empyema is a cause of a generalized and systemic infection (Fig. 15.5). It could aggravate the patient clinical situation and later can develop a sepsis with lethal consequences, if it is not promptly and correctly treated.

As shunt infection is a known complication of ventriculopleural shunting, a high index of suspicion must be maintained for infection in both the CSF and any associated pleural effusion when these patients present with fevers without another obvious source. Early sampling of the CSF and pleural fluid will direct prompt institution of antimicrobial therapy and shunt exteriorization, which may prevent the development of frank empyema [19].

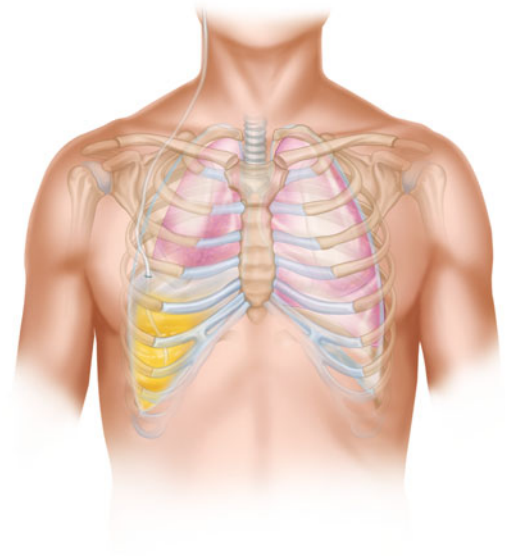

Fig. 15.5 Schematic design of a case of thoracic empyema secondary to VPL shunt. Usually it is a cause of a generalized and systemic infection

For the development of fibrothorax, chronic condition is essential and is characterized by dense fibrosis, contraction and trapping of the lung, atelectasis, and reduction of the size of the hemithorax. Fibrothorax with invasion of the chest wall and narrowing of the intercostal spaces may be thought of as the end stage of this process [32, 49].

The specific management of empyema and fibrothorax consists basically of two techniques. Video-assisted thoracoscopy (VATS) allows adhesiolysis and debridement of pleural cavity. Nowadays, it is considered to be the primary modality for treating complicated empyema after initial therapy, with or without chest tube drainage, in many institutions.

Although VATS has the advantages of better exposure and patient tolerance, open thoracostomy with debridement is an effective approach to treat empyema and fibrothorax. Empyemectomy is rarely performed. Extrapleural dissection of the parietal surface and the sac from the lung, just as in decortication of a chronically collapsed and trapped lung, requires undesirable and unnecessary lung resection very often [32].

As this complication is rarely seen, only two cases were reported. Yellin et al. in 1992 [49] reported open thoracostomy and decortication. In this case, the shunt had to be removed, and decortication had to be performed to alleviate severe respiratory symptoms in the child. Another similar case was reported by Khan and Kahlil in 2008 [22].

15.4 Overdrainage

An interesting issue is that of the changes induced in the intracranial pressure by the dynamics of a valve that drains into the pleura. Intrapleural pressures are subatmospheric throughout the respiratory cycle, which probably induces a continuous sucking effect of the CSF that could decrease the intracranial pressure. At least theoretically, VPL shunting should produce symptoms and signs of CSF overdrainage in all patients treated by this technique. In 1998, Munshi recorded the intraventricular pressures with a telemetric sensor in patients with VPL shunts. Four patients with ventriculopleural shunts were monitored telemetrically while supine and at increments of head elevation to 90°. Their findings indicated that the negative intrapleural pressures generated during the respiratory cycle tend to generate intraventricular pressures that are consistently lower than those observed with peritoneal shunting. This observation suggests that VPL shunts may be appropriate for patients requiring very low intraventricular pressures in order to resolve their hydrocephalic symptoms [36]. This event was partially reflected by a similar case reported by Chiang et al. [5].

Using an antisiphon device attached to the valve probably prevents this untoward effect. In fact, several authors have stressed the importance of placing such a device in cases with CSF drainage into the pleural cavity [21, 36].

Antisiphon devices are believed to prevent this complication by preventing overdrainage. Short-term follow-up of patients with VPL shunts for as long as 2.5 years has shown good results from these devices in preventing the occurrence of pleural effusions [31].

The use of antisiphon devices in children frequently induces symptoms of underdrainage [21], owing to the narrow margins that these devices achieve for effectively controlling raised intracranial pressure. The good results obtained in the cases reported by Martínez-Lage et al. [31] could well be attributed to the fact that their valves were not of the standard differential-pressure type.

It would be reasonable to expect that the use of an externally adjustable shunt, by regulating the pressure settings of the valve to the patient's needs, will be capable of avoiding both the siphoning effects on the ventricles and the formation of significant collections of CSF within the pleura [31].

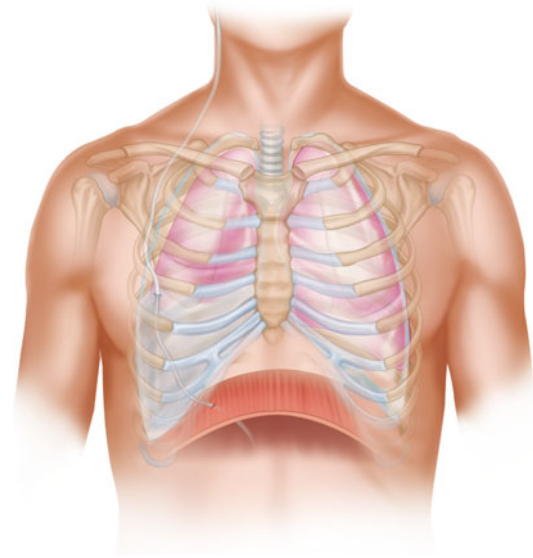

Fig. 15.6 Schematic design of a case of a transdiaphragmatic migration of a VPL shunt

15.5 Migration

Migration of ventriculoperitoneal shunts into the pleural cavity has also been noted in association with the growth of the patient, with resultant pleural effusion. Johnson and Maxwell [20] reported a delayed case, while Pearson et al. [38] reported a migration of the distal catheter between the ribs and pleura with subsequent dysfunction.

We have seen a case of a transdiaphragmatic migration of a VPL shunt (Fig. 15.6) as well as a

rare migration of the distal catheter from the pleural space to the cardiac atrium through the pulmonary vein (Fig. 15.7). Another migration reported is from the pleural space to the mediastinum with ensuing pericardiac effusion. These two later cases are extremely rare occurrences.

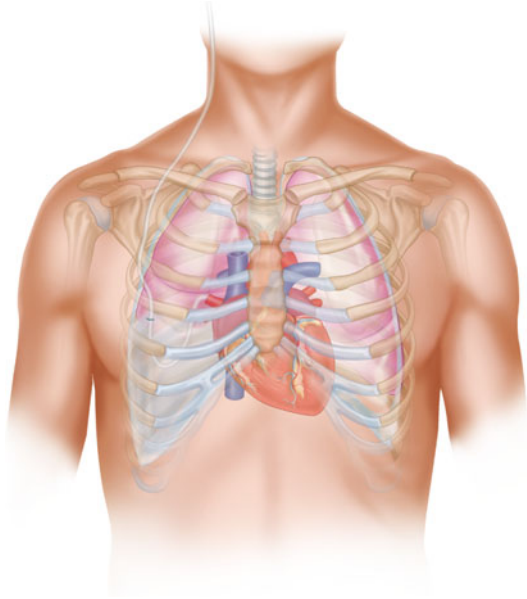

Fig. 15.7 Schematic drawing showing a rare migration of the distal catheter from the pleural space to the cardiac atrium through the pulmonary vein

15.6 Proximal and Distal Obstructions

Pleural inflammatory process associated to pleural effusion and fibrin deposits over the pleural surfaces can produce distal catheter obstruction. This complication is associated with a catheter malfunctioning and pleural effusion, with or without an infection.

Management consists of removing the catheter inside the pleura and replacing it if necessary.

15.7 Disconnection and Coiling

Disconnection is a fairly common complication, directly related to the surgical technique. Their management consists in reconnecting both ends surgically.

We have seen two cases of coiling of the distal catheter in the pleural space. An excessive length of the distal catheter may have contributed to this complication (Fig. 15.8a, b).

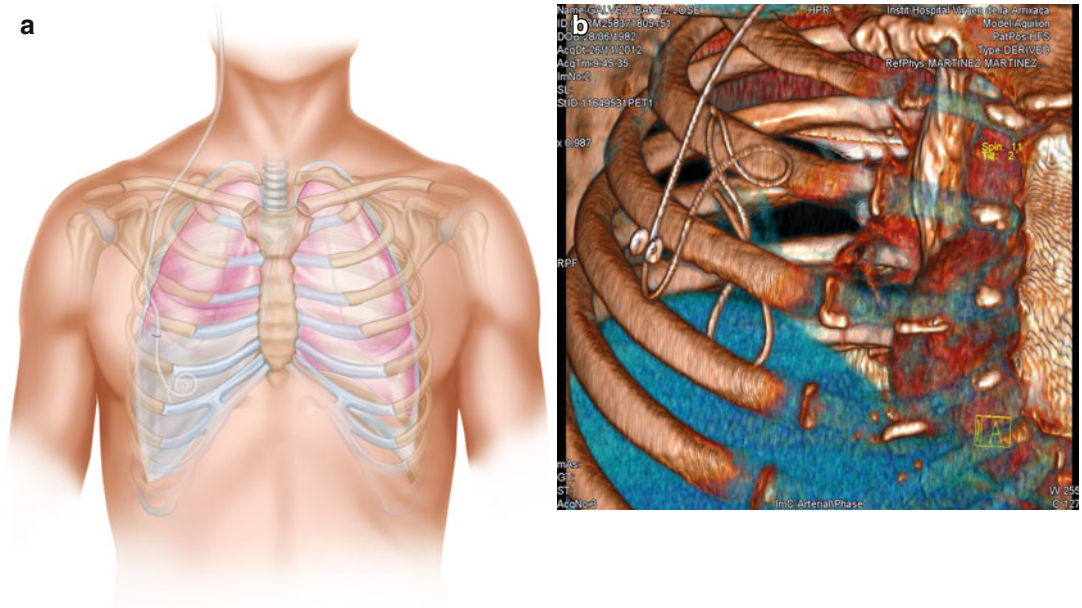

Fig. 15.8 (**a**, **b**) Schematic design showing coiling of the distal catheter in the pleural space. An excessive length of the distal catheter may have contributed to this complication as we have seen in this 3-D thorax computed tomography

15.8 Cerebrospinal Fluid Galactorrhea

When a migration of catheter takes place or even in association with pleural effusion, the cerebrospinal fluid could spread around nearer tissues such as the breast. Continued drainage of the cerebrospinal fluid into the subcutaneous and breast tissue led to the development of breast enlargement and drainage via the nipple. Also, it is collected inside until drained spontaneously through the nipple or is surgically drained. This extremely rare complication is related to the pleural end of the catheter retracted out of the pleural cavity [35].

Regarding our experience, we have recently treated a case that simulated abscess mastitis in a young male treated with ventriculopleural shunt due to a ventriculoperitoneal shunt failure. He presented an asymptomatic loculated pleural effusion with initial fibrotic changes that stopped the normal CSF reabsorption, so the CSF continued flowing to the issues around (adipose tissue of the breast) simulating an abscess mastitis (Fig. 15.9).

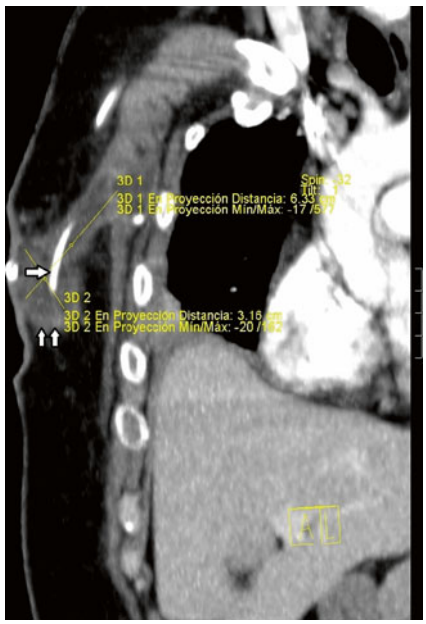

Fig. 15.9 Thorax computed tomography showing an abscess mastitis (*double arrow*) flowing to the adipose tissue of the breast from the distal catheter of a VPL shunt (*single arrow*)

It was surgically drained, microbiological cultures were taken, and the shunt catheter was externalized.

Management included CSF microbiological cultures, systemics antibiotics, and removing the shunt catheter with surgical drainage of the damaged area. A third ventriculostomy was performed to alleviate his hydrocephalic symptoms.

15.9 Tumor Spread Through the Shunt Tubing

Shunts as an artificial anastomosis can provide the means for the spreading of tumor cells by the cerebrospinal fluid. The first spreading of tumor cells via a shunt was reported in 1954 by Ransohoff, and interestingly, it was a ventriculopleural shunt [40]. Most cases from there include seeding of posterior fossa medulloblastoma or other highly malignant intracranial tumor spreading through a ventriculoperitoneal shunt. Further nearly 100 cases of the world literature with shunt-associated metastasizing of brain tumors were reported. The extraneural spreading of tumor cells through shunt tubes must be considered as a possible complication of the shunting procedure.

Other authors have described the spreading of tumor cells from ventricles to the pleural space [46].

Brust et al. [3] reported a case of glial tumor metastases through a ventriculopleural shunt resulting in massive pleural effusion.

Specific management consists of chemotherapy and/or radiotherapy, if they are indicated.

If a tension hydrothorax occurs, thoracocentesis or even chest tube drainage is recommended.

15.10 Cardiac Tamponade

Zaman et al. [50] reported an unusual case of a patient with a ventriculopleural shunt presenting with signs and symptoms of heart failure due to massive pericardial effusion. Imaging revealed the distal shunt catheter end within the middle mediastinum to have migrated from the pleural

space. The patient underwent a shunt revision procedure resulting in complete resolution of the presenting pathology. This complication is extremely rare, and we have only seen a similar case once.

In sum, VPL is an accepted shunt technique in neurosurgical scenarios. Although technically feasible and relatively simple, it is not exempted from specific complications. It can be used when the peritoneum is not feasible. The avoidance of complications is directly related to the shunting insertion technique, including anesthesiologist facts and proper selection of the patient. Prevention of malfunction is also related to the abovementioned factors and to intrinsic factors of the shunt components. Nonetheless, as in any shunt procedure, there is still too much to gain knowledge of.

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