Complications of Peritoneal Shunts

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13.1 Introduction

Diversion of CSF fluid for the treatment of hydrocephalus constitutes a standard technique in neurosurgery, and it is one of the most frequent procedures performed in a neurosurgical unit [9, 10, 21, 22, 29, 32, 65]. The peritoneal cavity is the most common site for cerebrospinal fluid (CSF) absorption. It was first used by Ferguson in 1898, through a lumboperitoneal shunt system, and later popularized by authors like Nulsen and Spitz (1952), Holter or Pudenz (1956) [18, 65]. In spite of the spread of endoscopic third ventriculostomies for the treatment of obstructive hydrocephalus, ventriculoperitoneal shunting remains the elective technique for most of the cases of communicating hydrocephalus, which nowadays still stand as the first cause of hydrocephalus for the vast majority of patients. Ventriculoperitoneal shunting usually provide immediate relief of intracranial hypertension, and it is simple to be performed [47]. Unfortunately, the complication rate is relatively high, mostly related to infection and mechanical failure (obstruction, fracture of the distal catheter, or pseudocysts) [4, 9, 10, 21, 45, 66, 69].

Abdominal complications are relatively frequent after VP and lumboperitoneal CSF shunting and account for up to 25 % of noninfectious complications [1, 4, 9, 18, 21, 22, 26, 39, 43, 50]. They can present with a variety of signs and symptoms. Complications of the procedure include ascites, shunt infection, metastases of

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brain tumors, pleural effusion, pneumothorax, intestinal obstruction, perforation of a hollow viscus (such as the bladder and bowel), volvulus, and migration of the distal catheter [2, 4, 10, 12, 13, 15, 18, 24, 27, 39, 45, 59, 66, 70, 77]. Although some of these complications are merely mechanical (disconnection, fracture, migration), many others include an inflammatory and/or infectious etiology, and are potentially hazardous if left undiagnosed [10, 12, 72, 80]. All of them have been related as "nonfunctional abdominal complications of the distal catheter" [47], due to the fact that they share a common pathophysiological origin in the presence of a functioning shunt.

Due to the high variety of possibilities that may complicate an abdominal shunt, awareness about the different pathological entities and prompt recognition will render best results in their treatment and prognosis.

13.2 Epidemiology

In a review of the literature performed by de Aquino et al., they found abdominal complications to be a common problem after ventriculoperitoneal shunting [18]. Prevalence is as high as 47 % (more frequent in males than in females). It may happen at any age, but seems to be more frequent in younger children than in eldest or adults, who are affected only in 10 % of all the cases. The age of the first VP shunt on each patient varied from a few days to 58 years but in 66.7 % of the patients, they were less than 1 year old (usually between the first and tenth month of life).

The lapse of time between the first VP shunt and its complication occurred in the 1st year after VP shunt implantation in 57.5 % of the patients, and only 20 % beyond the 3rd year after shunting [18, 22].

Grosfeld et al. reported an intra-abdominal complication rate of 24 % in a series of 185 children treated by VP shunting procedures [32].

Intra-abdominal fluid collections are also relatively uncommon complications of CSF peritoneal shunts. Abdominal pseudocysts are more frequent among them, and in a study by Salomao et al. 18 cases of abdominal pseudocysts were reported [71]. In their series with positive CSF cultures they found a 44.4 % rate of shunt components infection and positive cultures of the peritoneal end of the catheter in 61 %.

13.3 Physiopathology of Nonfunctional Abdominal Complications of Shunt Catheters

Complication rate after peritoneal shunting is high, mostly related to infection (at CNS, peritoneum, or shunt components) [10, 18, 22, 66], or due to obstruction (of the proximal catheter by choroids plexus, ventricular wall, cerebral parenchyma, blood, and other debris) [2, 9, 11, 34, 52, 83].

Although the silicon elastomer is considered an inert material and designed to remain in the body for very long periods, the fact is that catheters may fracture in its long trajectory at the subcutaneous tissue by mechanical trauma, by a biodegradation process [10, 18, 20, 31], or by the formation of a bacterial biofilm on the catheter surface [26]. Many abnormalities found on this surface can be responsible for bacterial adhesion. There is also evidence that CSF proteins are absorbed at the catheters and act as another responsible factor for biodegradation and bacterial adhesion [18].

Pathophysiology of peritoneal fibrosis has been studied in animal models and humans undergoing continuous abdominal peritoneal dialysis [40]. Ultrafiltration of CSF at the peritoneum follows an osmotic pressure gradient facilitated by hydrostatic pressure mechanisms. Higher pressure of CSF at peritoneum helps the passage of fluid through micropores at the membrane. Molecular studies in chronic peritoneal dialysis models confirm the presence of small pores, which are responsible for the flow of low molecular weight substances such as urea, creatinine, glucose, as well as the presence of large pores, which are responsible for the flow of high molecular weight substances. Larger pores, called "aquapores," would facilitate the passage of substances with higher molecular weight.

Success of the ultrafiltrating process (and thus of the CSF resorption in the abdominal cavity) depends on the functional integrity of the peritoneal membrane and of the vascular mesothelium. Absorption rate of the peritoneal fluid through the lymphatics ranges from 0.5 to 1.5 ml/min, with the peak absorption occurring through diaphragmatic lymphatics, especially at the subdiaphragmatic region, where there are open intracellular channels "stomata." These are conducted to the mediastinal lymphatic and localized before the right lymphatic duct, so it can then reach the right internal jugular and subclavian veins (see the reference paper by de Aquino et al.) [18] Continuous exposure of the peritoneal membrane to the CSF fluid can result in significant changes on its morphology and ultrastructure with a potential risk of peritoneal fibrosis.

Abnormalities in the morphology and ultrastructure of the peritoneal membrane occur especially at the peritoneal mesothelium, with an increased development of the wrinkled endoplasmic reticulum and a decrease in membrane microvilli (responsible for the increase in its surface area), micro-pinocytic vesicles, and changes on the submesothelial layer, leading to a sclerosis effect on the connective tissue (see Krediet and de Aquino et al.) [18, 40]. Glucose present in the CSF fluid could be potentially toxic to the mesothelial cells and be responsible for vascular changes in the peritoneal membrane.

On the other hand, and acting as a summatory mechanism, the presence of foreign bodies inside the peritoneal cavity activates macrophages and monocytes, which stimulate mesothelial cells to produce immunomediators (interleukins or IL) [18]. These initial IL, such as IL-β1, TNF-α, PGE-2, and prostacyclin 2 (PGI-2), activate IL-6 and IL-8, which attract neutrophils to the inflammatory site. It is postulated that IL-β1 yielded by monocytes and macrophages may have an important role in physiopathology of peritoneal fibrosis and can increase collagen synthesis to an elevated level of procollagen in fibroblasts [18]. Peritoneal fibroblasts would respond to inflammatory stimuli by increasing the extracellular matrix compounds, potentially contributing to the development of peritoneal fibrosis. Fibrosis reaction around the distal catheters and structures together with bowel peristaltic movements and increased intra-abdominal pressure would act as a constant source of mechanical pressure that would lead to necrosis and ultimate serosal perforation of viscus at the site of the anchoring [12, 19, 27, 58, 73]. As it will be explained later, the weakness of certain peritoneal areas and the umbilical end of the vitellointestinal duct or the patent processus vaginalis into the scrotum could act as a facilitating mechanism for perforation [6, 49, 58, 81].

13.4 Sterile Ascites

Sterile ascites is a complication rarely reported [2, 9, 16, 17, 32, 43, 75, 83]. In the majority of cases, the pathological collection of CSF in the peritoneum occurs within a pseudocyst, due to infection from a shunt and posterior peritonitis that causes the pathological accumulation of abdominal CSF [9, 26, 83]. In sterile ascites, on the other hand, CSF is not loculated but accumulated in the peritoneal cavity. It is defined by negative Gram stains from the ascitic fluid as well as negative viral and bacterial cultures [16, 75].

Ascites usually results from a concurrent illness such as cirrhosis, congestive heart failure, nephrosis, or disseminated carcinomatosis. To explain the development of sterile ascites after abdominal shunting, different pathological pathways have been recalled.

1. Immune responses from a shunt material breakdown causing inflammatory reaction could lead to fibrosis of the peritoneal layer and abnormal resorption at the serosa level [10, 18, 31].
2. Prior abdominal surgeries or multiple shunt revisions may cause adhesions or preclude resorption due to a malabsorptive peritoneum [9, 32].
3. Elevated CSF protein levels from CNS pathology, such as infection or neoplastic processes, which could increase oncotic forces within the peritoneum [2, 75, 83].
4. CSF overproduction from diffuse villous hyperplasia or papilloma of the choroid plexus [11].

Patients with ascites usually have delayed symptoms of underdrainage on presentation [21, 32].

Physical examination may show abdominal distension and tenderness, usually with little signs of defense. Findings include altered abdominal contour, fluid wave, or dullness to percussion. Abdominal perimeter is high and dilated abdominal wall veins may be seen as a sign of ascites.

Diagnostic imaging consists of abdominal ultrasound, which shows diffuse collection, without loculation or septae and excludes signs of thrombosis in the hepatic vessels. Abdominal CT scans show single, usually large, nonloculated peritoneal fluid collection surrounding the catheter but usually no thickened omentum or peritoneum or other signs of inflammation.

Peritoneal centesis is the diagnostic modality of choice and can provide information regarding the source of the ascites.

In the absence of these potential causative issues, the shunt should be externalized to confirm that the ascites is a result of excessive CSF accumulation. So far, CSF cultures remain the most reliable method to rule out infection [57, 71]. However, growths of microorganisms in the shunt components in the absence of positive cultures in CSF are often attributed to contamination. Extending cultures longer for up to 14 days is recommended to effectively exclude most organisms including slow growing anaerobes such as *Propionibacterium acnes*, before infection is definitively ruled out.

Treatment After diagnosis, the peritoneal catheter is externalized and the patient remains under antibiotics (e.g., vancomycin and ceftazidime) until CSF cultures are proven negative and infection is ruled out. Antibiotics are kept for 24 h after definitive insertion in a new cavity [83]. Due to the high protein accumulation and CSF formation in some pathological entities, atrium is usually the selected one, and ventriculoatrial shunting commonly resolves the problem [2, 11, 51, 78].

Summary Sterile ascites should always be a diagnosis of exclusion. Standard workup must include analysis of the ascitic fluid including cellularity, protein levels, glucose, and cultures as well as cytology to exclude malignancy. Imaging consists of abdominal CT and cranial MRI to rule out associated pathologies. Once malignancy and infection have been ruled out and abdominal pathology resolved, the shunt can be safely converted to a VA shunt.

13.5 Pseudocyst

Pseudocysts are loculated intra-abdominal fluid collections developing around the distal end of the peritoneal catheter [52, 60]. The occurrence of a pseudocyst after peritoneal CSF diversion was first described by Harsh [33]. Since then, a frequency of around 0.7–10 % has been reported in the literature. Pseudocyst can occur any time between several weeks and several years after the initial shunting procedure [21, 22].

The exact cause of cerebrospinal fluid abdominal pseudocysts is not completely understood, but risk factors seem to be related to inflammatory processes. Several predisposing factors have been described: acute shunt infection, a past history of cerebrospinal fluid infection, multiple shunt revisions, previous abdominal surgery, and central nervous system tumors [9, 10, 21, 26, 29, 34, 39, 69]. It is agreed that an inflammatory process, either infectious or sterile, is a frequent predisposing factor. Bacterial infection of the shunt system preceded the pseudocyst formation in as much as 73 % of the reported cases [71], but the incidence of infection could be higher if cultures of CSF were maintained over 14 days or processed for anaerobic germs. The inflammatory process has an important role in pseudocyst formation: inflamed intestinal serosal surfaces, fibrous tissue without epithelial lining, fibrous tissue with acute inflammation, and granulomatous tissue with fibroblasts, collagen bundles, and scattered inflammatory cells have been found in the histological examination [18, 29, 34, 39, 52]. All these figures turn the wall of the pseudocyst into an inflammatory surface that is unable to absorb cerebrospinal fluid.

The time from a shunting procedure to the development of abdominal cerebrospinal fluid pseudocyst ranges from few days to several years.

In a paper from Santos de Oliveira et al., this period extended from 10 days to 15 years [57]. Often, abdominal pseudocysts tend to occur within 6 months of the last intra-abdominal surgical intervention. It has been suggested that smaller pseudocysts tend to be infected, and larger pseudocysts tend to be sterile [61, 71]. However, this could not be proven and one study from Roitberg et al. found no statistically significant link between infection and pseudocyst size [69]. Infection rates varied between 17 and 80 % (average of 42 %) in 128 reviewed cases [52]. *Staphylococcus epidermidis* is the most frequent responsible organism, but other coagulase negative staphylococci may be found such as *S. capitis* or *S. hominis*, as well as Gram negatives like *Propionibacterium acnes* [52, 61, 69].

Symptoms related to the occurrence of an abdominal cyst include vomiting, distension, abdominal pain, fever, and/or abdominal mass. It is often accompanied by signs and symptoms of shunt dysfunction due to the nonreabsorption of CSF loculated inside the cyst such as fontanel bulging, somnolence, headaches, vomiting, or seizures.

Allergic reactions are another potential cause of the sterile inflammation leading to pseudocyst formation [18, 34]. In cases of repetitive pseudocysts in a patient in whom shunt infection has been reasonably ruled out, the possibility of allergic reaction against any of the components of the catheter must be borne in mind. Increase in peripheral eosinophils and serum IgE levels, as well as an eosinophilic infiltrate of the pseudocyst walls may alert about this possibility [18, 39].

There is an open debate about which of the components of the shunt would be responsible for the immunological reaction. Silicone elastomers and gels have been extensively studied due to the widespread use of breast implants. Three different and independent investigation groups have concluded that there is no convincing evidence to support or lend biologic plausibility to an association of silicone breast implants with immune-related human health conditions. They further indicate that there is insufficient or flawed evidence that silicones can elicit an immunotoxic response, trigger a specific immune reaction, or amplify an autoimmune-like disease [39]. Previous reports of specific antisilicone antibodies could have been misdiagnosed due to a different level of circulating albumin and a nonspecific adsorption of the IgG fraction [31].

Other sources of antigenic stimulation have been considered. The ethylene oxide (ETO), a gas used to sterilize medical devices, including VP shunts, is a highly reactive alkylating agent that can react with endogenous proteins to create a neo-antigen. IgE antibodies specifically directed toward ETO protein conjugates, and the presence of an ETO metabolite (ethylene chlorohydrin) in the CSF have been found for as long as 4 months after the last shunt revision in patients with elevated eosinophil counts, who underwent multiple shunt revisions without evidence of an infection.

Another possible antigen source is barium sulfate, the radiopaque agent included in the manufacture of VP shunts to allow for visualization of the shunt by x-rays, latex the material used in the fabrics of surgical gloves, or antibiotics such as vancomycin or gentamycin, frequently used by some surgeons to impregnate catheters during surgery for VP shunting [31].

"Silicone" allergy has been treated with corticosteroids, but reactions recurred upon removal of the medication. Changing the shunt system made of the usual silicone elastomer to one made of polyurethane or made of 'extracted silicone' elastomer usually solves the problem. "Extracted silicone" elastomers are manufactured through an industrial process that retrieves the small percentage of unbounded silicone oil from the catheters leaving only pure solid silicone elastomer, thus making immune reaction less likely to reappear (Medtronic Neurosurgery).

Diagnosis Any patient that carries a VP shunt and complains of abdominal pain or distension is potentially a candidate to suffer a pseudocyst. Diagnostic tools include CSF sampling from the shunt reservoir and aspirate from the cyst to be sent to the laboratory. CSF is evaluated for signs of infection.

Laboratory tests should include Gram stain, culture, glucose, protein, and cell counts.

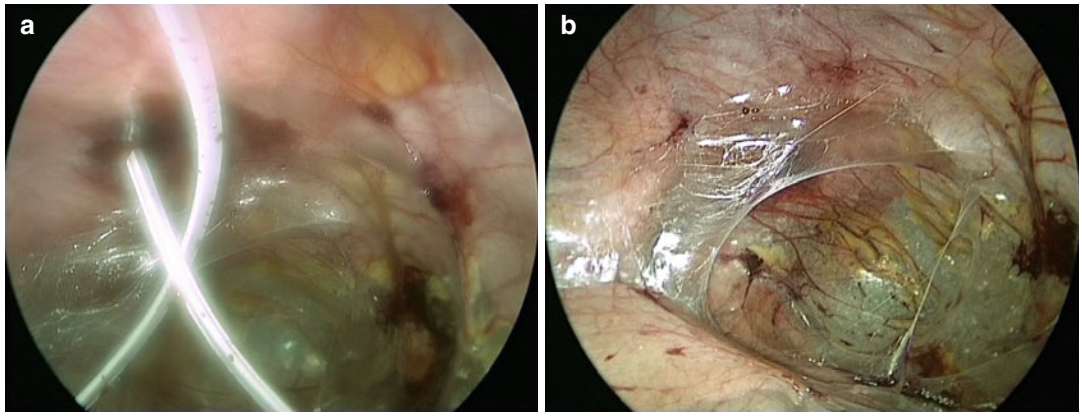

Fig. 13.1 Pseudocyst. Peritoneal catheter is withdrawn from the pseudocyst (**a**) and repositioned in functional peritoneum under laparoscopic assistance (**b**)

Shunt infection is defined after a positive culture of either cerebrospinal fluid or abdominal fluid.

Plain radiographs may show the shunt tube coiled in a soft tissue mass displacing adjacent bowel loops. Abdominal ultrasound or CT

Treatment Several techniques have been used, usually with good results. Pseudocysts have been treated traditionally with surgical shunt externalization, antibiotics for presumed or documented infection, and a second surgical procedure for shunt reinsertion.

The presence or absence of infection must be established before definitive treatment can be carried out. Usually, it is assumed that there is concurrent infection that requires shunt externalization or external ventricular drain. Identification of cerebrospinal fluid infection precludes prompt reinsertion into the peritoneum.

Pseudocysts have traditionally been treated with surgical shunt externalization, connecting the distal catheter to a collecting sterile bag, antibiotics for presumed or documented shunt infection [71], and a second surgical procedure for shunt reinsertion either in the peritoneal cavity or in a new location such as pleura or cardiac atrium through a jugular vein [51]. In the past, ultrasound guided paracentesis followed by radical excision of the cyst walls through exploratory laparotomy and insertion of the distal catheter in a new location inside the peritoneal cavity was the choice elected. Recently, laparoscopic management of the pseudocyst, which involves excision of a portion of the cyst and repositioning the catheter within the peritoneal cavity, is preferred (Fig. 13.1). For some authors repositioning of the peritoneal catheter in the abdomen could lead to the recurrence of abdominal pseudocyst. However, shunt replacement back into the abdomen has been feasible in most cases and in the majority of the patients, contralateral peritoneal cavity is a reliable option. Peritoneal cavity can be used for shunting once the cyst had reabsorbed [1, 9, 29, 37, 41, 42, 44, 71]. To minimize the effects of peritoneal adhesions, the peritoneal catheter may be placed in a retrohepatic subdiaphragmatic position [41, 67, 71]. CSF diversion to ventriculoomental bursa or lesser sac may be considered as another acceptable alternative technique to CSF shunting when the anterior peritoneum loses or decreases its CSF absorption capacity [23, 50] (see later). Only exceptionally, and after failure of the previous techniques, it is a necessary conversion to an atrial shunt [47, 77].

When there is a chance for it, endoscopic third ventriculostomy (ETV) is an excellent approach for selected cases of noncommunicating hydrocephalus. As it has been shown, for those patients that remain shunt dependent or ETV has failed, ventriculoperitoneal shunt can be safely reinserted in the majority of the cases.

13.6 Anal Extrusion. Bowel Perforation

Bowel perforation and anal extrusion of the distal portion of a VP shunt (AEVPS) is a rare mechanic complication of VP shunts. Wilson et al. described the first case reported in 1966 [81]. Since then, more than 100 cases of bowel perforation have been reported and the incidence of this complication is thought to be around 0,1–1 % [15, 19, 27, 49, 58, 63, 72, 81, 82]. In a retrospective review of their series in 2006, Vinchon and colleagues found 19 cases of bowel perforation due to VP shunt: only three of them developed anal extrusion of the catheter.

Pathophysiology and risk factors Pathogenesis and predisposing factors are not completely well understood. By definition, AEVPS involves a bowel perforation that has been produced through different mechanisms. Di Rocco suggested that bowel erosion results from inflammation caused by a preexisting shunt infection [21]. Interaction between mechanic trauma and inflammation following infection can lead to the bowel perforation [18, 19, 27, 43]. In the majority of the reported cases pathogenic agents suggest a peritoneal focus (e.g., *Escherichia coli*), but it is not uncommon to find organisms that are typically related with a perioperative contamination (*S. aureus* or *S. epidermidis*). Different authors suggest that some cases of bowel perforation can be linked to mechanisms of inflammation and rejection of an infected foreign body [18, 19, 34]. Intestinal developmental alterations can be a predisposing factor too: in a paper from Matsuoka et al. bowel perforation was related to a duplication of ileum terminalis that resulted in continuous irritation of a fixed point on the bowel's surface and finally perforation of the sigmoid colon [49]. Encasing fibrosis around the tube has been reported; this fibrosis may have an anchoring effect on the catheter, resulting in pressure and decubitus ulceration, which can lead to a perforation [6, 49]. It has been also reported that the weakness of certain peritoneal areas and the umbilical end of the vitellointestinal duct and the processus vaginalis into the scrotum might remain patent and act as a facilitating mechanism for perforation [59, 81]. Formerly it was believed that spring-coiled catheters were more prone to produce visceral perforations, as this kind of catheter had been implicated in more than the 50 % of the cases, and in cases of gastric and peroral migrations. However, many reports have shown that softer and more flexible silicon catheters can also produce bowel and other viscus perforation [21, 27, 63, 72].

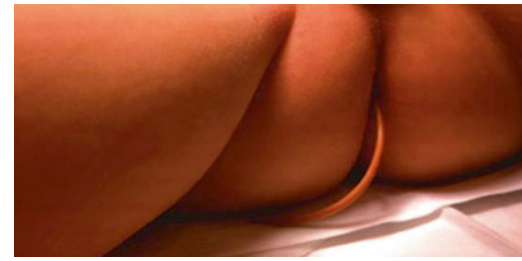

Fig. 13.2 Anal extrusion of peritoneal catheter

No evidence has been found that the peritoneal opening technique (laparotomy versus trochar) when the shunt is positioned could be a risk factor for bowel perforation. Some authors consider that laparotomy is a safer technique; on the other hand, many papers find that trochar technique is at least as safe as laparotomy and it is not a risk factor for bowel perforation [78]. Perforations due to the trochar are described, but usually they are an operative, acute complication, not related to a chronic inflammatory process. Serious vascular damage has been reported during trochar insertion: for this reason, we favor mini-open laparotomy in opposition to trochar-guided insertion.

Other potential risk factors for bowel perforation could be suspected silicone allergy or weakness in the bowel wall resulting from deficient innervation like in children with myelomeningocele or congenital hydrocephalus [4, 12, 34]. It is not clear whether the length of the peritoneal catheter has any implication in hollow viscus perforations.

Diagnosis AEVPS is a pathognomonic sign of bowel perforation (Fig. 13.2), but it is infrequent (15.7 %). The absence of peritoneal signs is

common in cases of bowel perforation due to a VP shunt. Clinical peritonitis is observed in 15–25 % of the cases. As much as 48 % of the cases can develop meningitis and/or ventriculitis. Abdominal radiology can be used when the diagnosis is not obvious. Abdominal CT scan with contrast and ultrasonography may show local inflammation signs and a thickened abdominal wall, but this exploration may result negative. In some cases with a high index of suspicion, it is possible to perform a shuntogram, which consists in the instillation of a contrast medium into the lower portion of the shunt, to demonstrate bowel perforation. Colonoscopy could be used to identify the point of the perforation [27, 49]. Cranial CT scan or MRI must complete the follow-up diagnostic studies. CSF sampling must be obtained from the shunt reservoir, from the tip of the catheter exposed or after externalization of the peritoneal end.

Treatment Bowel perforation has to be managed as a surgical emergency. If the patient does not show any symptom apart from the AEVPS, a conservative management is possible. The shunt can be cut in the abdominal surface, externalizing the proximal end and eliminating the distal end by a trans-anal traction. If the patient presents with clinical (peritoneal and/or meningeal) symptoms, imaging studies are needed to assess the presence of active hydrocephalus and localize the site of the bowel perforation. In this case, laparotomy or laparoscopy should be done in order to repair the intestinal perforation and retire the peritoneal end of the shunt [49, 82]. External ventricular drainage and antibiotic therapy is started until proven that CSF is not contaminated or, in case of CSF infection, that the infection has been defeated. After two or three consecutive negative CSF cultures, it is possible to replace the entire shunt system. Usually, new shunting to the peritoneum is an option, but atrial or pleural diversion is preferred when peritoneal positioning of the catheter is not feasible (e.g., big laparotomy, adhesions, or recurrent perforation).

Prognosis AEVPS is a rare but potentially severe complication of VP shunts. Children with no symptoms of perforation or meningitis show a better evolution. If intestinal perforation leads to a chemical or infectious peritonitis, or the patient suffers meningitis or ventriculitis after Gram-negative infection, then prognosis is bad. The highest mortality rates are shown in patients with abdominal complications [82].

13.7 Bladder Perforation

Bladder perforation by a peritoneal catheter is another rare complication of VPS [6, 15, 35, 53, 63]. This complication was first reported by Grosfeld in two patients, aged 3 months and 1 year, in 1974 [32]. Catheter removal, bladder repair, supra-pubic cystostomy, and antibiotic therapy resulted in recovery in each case. Around ten cases involving normal (nonaugmented) urinary bladder have been reported [6, 15, 35]. It has been also reported more commonly after abdominal repair of urinary bladder, during augmentative procedures for neurogenic bladder [53]. The location of the bladder, which lies in the extraperitoneal compartment, makes it a highly unlikely site of peritoneal catheter perforation, as the catheter must pass through the peritoneum into the extraperitoneal space and subsequently penetrate the bladder.

The mechanisms by which peritoneal catheters perforate hollow organs are not fully understood. An initial local inflammatory reaction around the tip of the catheter initiates its anchoring to the serosa of the hollow viscus [6, 19]. Calcifications occur at the distal tube in barium-impregnated catheters as sign of the fragmentation of the silicon polymers facilitating inflammation and anchoring [10]. Increase intra-abdominal pressure, peristaltic movements of the bowels, and CSF pulsation originates local pressure leading to necrosis and ultimate bladder perforation [6, 19, 63]. After entry into the urethra, final extrusion happens.

Shunt factors, which have been related to perforation, are sharp abdominal tip, long abdominal end of catheter, stiff consistency, barium coating of the catheter, or allergies to the shunt components (like silicon allergy). Initially, a higher tendency of perforation with spring-coiled catheters was reported. However, perforation may occur with any type of catheter.

Perforation of the bladder during trochar insertion or abdominal surgery can be prevented by draining the bladder before surgery. It is our policy to drain the bladder with a Foley catheter at the beginning of every surgery. If the patient is expected to have a long postoperative period (e.g., severe trauma or spinal cord tumor), Foley catheter is left in place. On the other hand, if shunting is a scheduled surgery, Foley is retired immediately after the procedure.

It has been reported that silicone rubber has a slight tendency to stick when it is in a dry state [35]. In distal independent catheters, the system may be flushed with saline. In unishunt systems, at least all components must be moisturized with saline and/or gentamycin dilution.

On examination, patients can be afebrile without neurological deficits or meningeal signs. Abdomen signs may be absent and peritoneal catheter can be seen coming out of the urethra with drops of CSF from the distal end. This can be explained because of a sealing effect of peritoneum around the catheter. If urine enters peritoneal cavity, patients may present with fever, abdominal pain, distension, and erythema of the abdominal wall between the umbilicus and the pubis. Diagnostic imaging must include again abdominal ultrasound and CT scanning.

Treatment In case that a bladder perforation occurs, it must be treated as an emergency. The shunt can be cut proximally and pulled out through the urethra. If no irritative signs are present or abdominal imaging excludes intraperitoneal complication (such as pneumoperitoneum or urinoma) a Foley catheter is placed and one may allow the bladder to heal on its own. However, at the lesser symptom of peritoneal defense, bladder can be approached extraperitoneally, repaired, and cystostomy or transient Foley catheter performed. In both cases, shunt is externalized proximally or external ventricular drainage left in place until CSF cultures are negative or peritoneum is ready to receive again the shunt in the opposite side.

Following a similar pathological pattern, perforation of the scrotum, umbilicus, and vagina has been also reported.

13.8 Other Infrequent Complications

Other rare complications of VP shunt surgery include migration, intestinal volvulus, abdominal wall perforation, or VPS-related abdominal metastases originating from brain tumors. Acute cholecystitis after subphrenic suprahepatic abscess among other bizarre complications is reported occasionally in literature.

13.8.1 Migration

Migration of the distal endings of the shunt components has been reported at different levels [24, 30, 44, 45, 59, 73]. In some cases, the weakness of the wall at anatomical preexisting foramina or their failure to fuse permits the occurrence of this unusual complication. Factors related to the migrations are anchoring of shunt tube to a calcified point, abdominal wall contractions, increased intra-abdominal pressure, flexo-extension movements of head and neck, and also the retained memory of shunt tube [3, 4, 6, 19, 34, 45, 59, 63].

Martin et al. reported migration of the intra-abdominal catheter through the right vertebrocostal trigone (foramen of Bochdalek) into the right hemithorax. This resulted in hydrothorax that resolved after revision of the distal end of the shunt [45]. Symptomatic pleural effusion after VP shunt has been reported, even in the absence of intrathoracic migration [3]. In some cases, this has been caused after hypochondrial compression through diaphragm due to formation of a pseudocyst after placing the shunt catheter in the suprahepatic subphrenic space [30].

Not uncommon, mostly in premature and newborns, is to find the distal end of peritoneal catheter migrating to the scrotal sac. Communication between testicular albuginea and peritoneal cavity together with decubitus and elevated pressure that CSF exerts on the tubular pass, may lead to hydrocele, and entry of the tube in the scrotum.

Upward migration of distal catheter has been also reported to the breast [73], intrathoracic to the pleura [3], intracardiac [70], or even to the

pulmonary artery. Thoracic trauma during placement of a shunt or direct trauma to vessels in the neck are mechanisms related to these complications [30, 46]. Migration of the distal catheter in the heart is a very rare complication of VP shunt that may be lethal, possibly causing pulmonary emboli, arrhythmia, sepsis, or cardiac insufficiency [70]. In all cases reported in literature, the catheter passed through a cervical vein into the jugular vein and ultimately into the heart. Erosion of the vein at the supraclavicular fossa related to a kinking in the catheter may be an alternative mechanism. For some authors, its frequency would be greater in children because of the thinner subcutaneous tissues in the neck. However, it has been hypothesized that it should be less likely in children below the age of 6 years for whom the tunneling devices are typically larger than subcutaneous veins of the neck.

To avoid this complication, tunneling the shunt too medial and too deep in the neck must be avoided. A vein perforated by the shunt passer may be difficult to detect intraoperatively unless profuse venous bleeding is found during subcutaneous tunneling [46].

Diagnosis is easy on simple XRs but CT scanning is advisable to delineate the course of the catheter within the thoracic cavity and heart. Echocardiography may help to discard cardiac perforation, thrombus, or valvular lesions.

Removal of the migrated shunt into the vascular/cardiac flow can be performed: percutaneously under fluoroscopic guidance, by interventional radiology, and in the most complex cases, through open thoracotomy. In those cases where the catheter is completely detached inside the heart or the pulmonary artery, percutaneous transvenous retrieval with a variety of loop snare devices, can be used, and open thoracotomy reserved for the cases of failure of interventional radiology. In these cases, a new VP shunt is indicated.

When the catheter remains connected to the extrathoracic part and the entire distal catheter is in the venous circulation it can be withdrawn percutaneously under fluoroscopic guidance. Care must be taken to ensure that there is no erosion of the heart walls or entanglement with the valves. If echocardiogram or enhanced CT scan shows no injuries to cardiac valves, some authors believe that there is no increased risk of endocarditis in the eventuality of shunt infection [70].

13.8.2 Intestinal Volvulus

Knotting of the distal catheter around the bowel and/or volvulus around the tube is a rare complication that is reported from time to time. Abdominal pain, distension, tenderness and defense, tympanic percussion, and intestinal silence are signs to bear in mind. XRs and ultrasound will make proper diagnosis straight.

Treatment consists of exploratory laparotomy, usually under laparoscopic technique, but sometimes, the entanglement of the tube and the suffering of the intestine may be so severe as to make an open laparotomy necessary. Then, the shunt is externalized and connected to sterile bag until new diversion procedure is needed.

Volvulus is a serious condition that necessitates confirmation of the integrity of the bowel and peritoneum before a new VP shunt is inserted. Otherwise, alternatives to diversion are pleural space, right cardiac atrium, or suprahepatic infradiaphragmatic recess.

13.8.3 Abdominal Wall Perforation

As we have seen, tip of distal catheter may induce a chronic inflammation and localized pressure that will finally result in erosion [4]. The end of the tube together with the continuous effect of the CSF pulsations penetrates the wall and perforates it.

Patients with myelomeningocele are more susceptible to perforation due to a weak musculature and local infective adhesion [4]. Fibrosis around the peritoneal catheter is a risk factor of visceral wall perforation [18]. In premature patients or low-weight newborns, abscess and perforation through umbilicus is another reported complication.

Treatment Infection of the shunt components must be suspected after any perforation of a hollow viscus or abdominal walls. After proper

cultures from CSF or purulent discharges are taken, broad-spectrum antibiotherapy is started; this must include typically gram-positives as well as gram-negative germs, which are not unusual in abdominal pathology related to shunting.

In case that peritonitis or peritoneal abscess is not suspected, distal catheter can be safely removed without laparotomy. The shunt must be removed by cutting the distal tube at the abdominal wall, externalizing the proximal edge and pulling the distal edge from the abdominal wall, without pulling the distal tip proximally, thus preventing the spread of the infection to the externalization site. Management includes shunt removal, external CSF drainage, and assessment for CSF infection followed by a new shunt device within the peritoneum.

13.8.4 Abdominal Metastasis

Extraneural metastases of primary brain tumors are rare and may occur through blood or lymphatic vessels [7, 13]. Spread through VPS can be facilitated by the direct connection established between cerebral ventricles and abdominal. It is also extremely rare, and less than 100 cases have been reported so far in literature [13, 64, 68].

The most frequent histological entities spreading through tube shunting are germinomas and endodermal sinus tumors in older patients (10–18 years of age), while medulloblastomas and astrocytomas are particularly common in the group of younger patients (below the age of 10). There is an overall male prevalence (1.9–1) a feature more pronounced in the older age group [68]. Mean interval between shunt operation and diagnosis of metastases is around one and a half year, but it may extend between 2 months and as late as 5 years after the first procedure. Occasionally, metastases have been diagnosed at autopsy [73].

Ultrasound or CT imaging of the abdomen might be considered as part of the routine follow-up in children with VPS who are being treated from brain tumors.

There are marked differences in prognosis with age and sex, in favor of older children and boys. But main prognostic factor affecting survival is histology of the tumor [7, 68, 74]. Not surprisingly, patients with germinomas and abdominal metastasis after shunting show better prognosis than those with a diagnosis of endodermal sinus tumor or glioblastoma.

Peritoneal metastases appear to respond well to systemic chemotherapy and/or radiation, but they must be considered a severe complication that may affect outcome.

13.9 Options for Catheter Placement After Peritoneal Dysfunction

After multiple distal failures, the peritoneal cavity is often deemed unsuitable for cerebrospinal fluid (CSF) diversion. Probably more than 30 % of patients with VP shunts will experience abdominal complications [1, 9, 18, 22]. Different places have been described when peritoneum is considered inadequate or impaired for CSF diversion, including right atrium of the heart, pleural space, or gall bladder among others. However, because of the benefits of VP over VA shunting (mostly a lower rate of severe complications) [25, 51, 78], many authors defend that every effort should be made to preserve the peritoneum as the place for the definitive distal catheter [1, 23, 37, 41, 62]. Potential complications of ventriculoatrial shunt malfunction are thromboemboli and infection, both life threatening [51, 78]. Thromboembolism occurs in 0.3 % of patients and is nearly always fatal [51]. Shunt nephropathy is also a potential risk long time recognized and reported in the past. The treatment of occluded or sluggish catheters is often anticoagulation that exposes the patient to severe bleeding risks, and revision of these catheters, when needed, is related to more complex procedures that include manipulation of central veins. Revision rates due to catheter outgrowth have been reported to be as high as 66 % [78].

For all these reasons, every effort must be made to find a functional peritoneum after previous shunt failures due either to infection or distal occlusion. Laparoscopy has been used as in the

management of VP shunt complications since the 1970s, providing excellent visualization of the abdomen through a minimally invasive approach [1, 37, 41, 42]. Surgeon can examine the abdomen for adhesions, cysts, loculations, and laparoscopy can be used to clear distal obstructions, excise CSF pseudocysts, or successfully reposition the distal catheter in patients with previous multiple shunt revisions or distorted abdominal anatomy. Laparoscopy is an excellent tool in locating suitable peritoneal pockets of the peritoneum where the distal catheter can be successfully replaced [1, 41, 42]. Open laparotomy remains an alternative for abdominal exploration, in more complex cases where laparoscopy is not indicated or easy to perform due to multiple loculations or dense adhesions [50, 62].

An alternative approach to minimize the effects of peritoneal adhesions is the employment of "reserve pouches" inside the peritoneal cavity [9, 23, 50, 57, 62]. Typically, retrohepatic subdiaphragmatic recess has been successfully used after "lost peritoneum" [57]. Several reports emphasize the advantages: it is away from previous adhesions, which usually affect the anterior peritoneum; and it is the place where the absorption rate of the peritoneal fluid through the lymphatics is higher, due to concentration of lymphatic ducts, and the presence of intracellular channels ("stomata") at the diaphragmatic peritoneum. Rengachary described a transthoracic transdiaphragmatic VP shunt for patients with difficult access to the peritoneal cavity due to diffuse anterior peritoneal adhesion. In this approach, the catheter is inserted directly into the suprahepatic subdiaphragmatic space through the thoracic cavity [67].

Matushita et al. have regained an interesting alternative technique to keep the peritoneal cavity as the main receptacle of CSF absorption [50]. They proposed to insert the distal catheter in the omental bursa (the lesser peritoneal sac), through the foramen of Winslow, jointly with a pediatric surgeon. They denominated this technique of as ventriculo-omental bursa (VOB) shunting. Follow up of three of their patients for over 1 year showed no recurrence of the peritoneal malabsorption. Omental bursa and retroperitoneal space had been used in the past as a receptacle for CSF since 1956, by authors like Picasa [62], Dodge [23], or Kubo [41] (see the beautiful review by Matushita et al.).

The omental bursa, also termed lesser sac, constitutes an extension of the anterior peritoneal space located behind the caudate lobule of the liver and covered by the stomach. It communicates with the anterior peritoneal space, the greater sac, or peritoneal cavity, through a constriction between the liver and duodenum named the epiploic foramen or foramen of Winslow. In its vertical extension, the omental bursa may reach the left iliac fossa and become very compliant in acceptance of fluid absorption, particularly in children. In adults, owing to the adhesions between the layers of the gastrocolic omentum, the vertical extent of the omental bursa is usually more limited [50].

Ventriculopleural diversion is an option that has been popularized in the last years when the VP shunt is not feasible [47]. However, it must be emphasized that pleural absorption may not be effective in children younger than 5 years of age [57]. The coexistence of pulmonary disease may contraindicate this surgery due to the risk of respiratory insufficiency in case of large pleural effusion. Martinez-Lage et al. suggested that the new technology valves may overcome complications related to pleural effusions or hydrothorax after ventriculopleural shunting [47].

For some authors the gallbladder provides also an alternate reservoir for CSF diversion. In 1959, Smith described the gallbladder as a shunt site [76], and since then, it has been used as a "last resource" option after failures of other cavities. The gallbladder is a sterile receptacle, a non-essential organ, reabsorbs water and electrolytes, and the pressure inside the bladder allows for maintenance of intracranial pressure. Bile is believed to provide lytic action, preventing fibrous adhesions and may have the added effect of neutralizing the excess of proteins in CSF [56]. In our personal series, three cases shunted into the gall bladder reached adulthood without any further complication after several failures of shunting procedures to the peritoneum. In two cases, shunt was inserted through an open

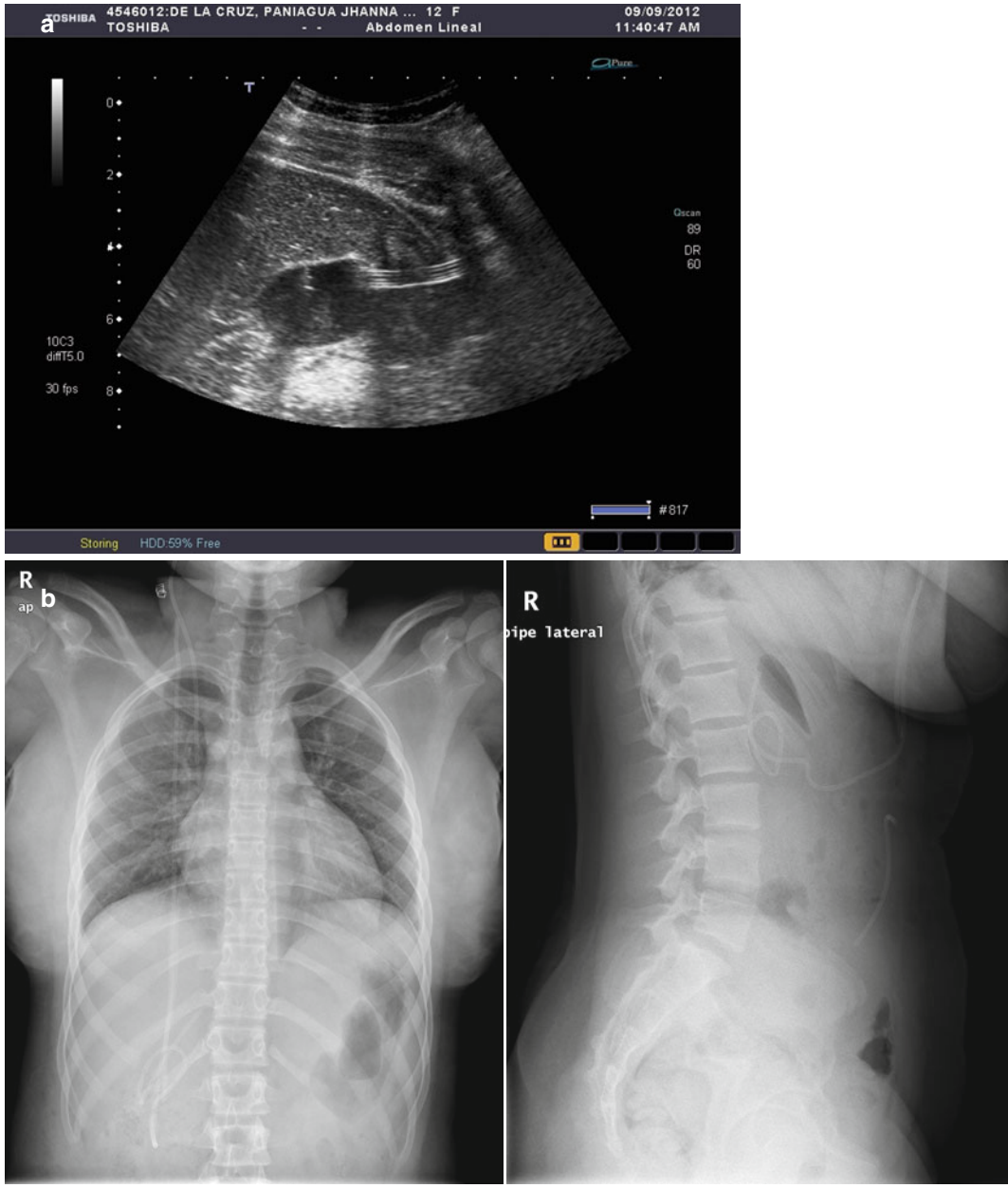

Fig. 13.3 VP shunt, with CSF derived to gall bladder. (**a**) Ultrasound shot showing distal catheter inside the gall bladder. (**b**) XR shows abandoned peritoneal catheter and distal catheter inside the gall bladder

laparotomy (Fig. 13.3), while in the last patient, laparoscopy was sufficient for insertion of the catheter inside the gallbladder and a purse-string suture to secure it to the wall.

Ventriculo-gallbladder shunt is an attractive alternative to peritoneal shunting [36, 48, 55, 75] in children with high protein levels in CSF, like in hydrocephalus secondary to tumors, where the lytic action of bile can potentially break down the high level of proteins present in the CSF [56]. Biliary sphincter tone and relatively high gallbladder pressures (10–20 cm H_2O) would function against the siphon phenomenon, preventing slit ventricle. But this may be also a point for

shunt dysfunction [55, 79]. There have been cases reported for mortality and morbidity associated with meningitis and ventriculitis due to reflux of the bile into the CSF [5, 8]. Olavarria, Tomita et al., remember that the distal slit valve for the gallbladder should be avoided as it may be kinked and opened by the gallbladder contraction [56]. Finally, it must be remembered that, though bile is usually sterile in children, infection rates may be as high as 30 % or higher in adults [48].

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