

A Review of Ventriculoperitoneal Shunt Migration

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Ventriculoperitoneal shunt migration is contextualized alongside the broader subjects of shunt obstruction and shunt infection. These three concepts are so interlinked that discussion of either is deficient without a discussion of the other two. Despite understanding this fundamental umbrella under which shunt migration finds its place, this review is limited to shunt migration in its own right. As an anatomic overview distal shunt migration may either be (1) internal, where the wall of any hollow viscus in the thoracic or abdominopelvic cavities is penetrated by the distal end of the shunt tubing, (2) external, where the shunt tubing penetrates and protrudes through any part of the abdominal wall and presents to the environment, or (3) compound, where after migration into a hollow viscus the shunt secondarily presents to the environment through an anatomical orifice [1]. While this anatomical understanding is succinct in understanding distal shunt tip migration, what must be added is proximal ventriculoperitoneal shunt migration which is an established entity in its own right [2,3,4].

Considering distal shunt tip migration the overall incidence as a percentage of shunt complications is reported to be as high as 10% [5]. Due to the close association between distal shunt migration and shunt infection, the most common presentation is hence meningitis and ventriculitis [1;5].

Understanding the pathophysiology of distal tip shunt migration is specific for the age, gender and anatomical site at which the distal tip shunt migration occurs. In the male neonatal and infant populations a critical consideration is the incidence of inguinal hernias that occur post ventriculoperitoneal shunt insertion. This has been reported to be as high as 17% [6]. In this age group the most common site for distal shunt migration in males is hence into the scrotum and the pathophysiology explaining this is the increased intra-abdominal pressure due to cerebrospinal fluid ascites causing either the inguinal hernia itself by reopening the processus vaginalis or by preventing its natural closure. Even in a term male neonate the processus vaginalis is open in 90% of patients. At 1 year of age the processus vaginalis remains open as a potential route for scrotal migration in 50% of male infants. Surprisingly several studies have shown that in up to 30% percent of adult males the processus vaginalis remains patent, and as such these individuals have a natural corridor for scrotal distal shunt tip migration [6,7,8]. Despite the high incidence of inguinal hernias post ventriculoperitoneal shunting a recent review, which considered 437 cases of congenital hydrocephalus that underwent ventriculoperitoneal shunting, the incidence of scrotal migration of the distal shunt tip occurred in only 4 (0.9%) of cases. This is indicative of the rarity of this presentation [9]. In terms of the management of these cases, the accepted principles involve repositioning of the distal shunt tubing into the abdominal cavity and operative closure of the patent processus vaginalis which can be achieved either laparoscopically or by open inguinal surgery. Fortunately shunt revision is seldom required and shunt infection in these cases is a rarity [7, 10, 11].

Whilst scrotal migration seems relatively free of accompanying shunt infection a case report of scrotal migration with subsequent scrotal perforation has been reported and hence the attending neurosurgeon should be aware that these cases are not always benign [12].

In terms of the other naturally occurring hiatuses in the abdominal cavity, mention must be made of the transdiaphragmatic hiatuses of Bochladek and Morgagni. Here the negative intra-thoracic pressure and positive intra-abdominal pressure post ventriculoperitoneal fluid shunting, along with the natural flow and absorption of peritoneal fluid into the thoracic lymphatics, are postulated as the pathophysiology by which shunt migration into the mediastinal or pleural cavity may be explained [13].

Besides ventriculoperitoneal shunt migration occurring through naturally occurring defects in the abdominal wall, as discussed, a plethora of literature exists, mostly in the form of isolated case reports, of shunt migration through adhesion and erosion into abdominopelvic viscera, or onto and within or through the abdominal wall [10]. In these cases a completely different pathophysiology explains this phenomenon.

What is accepted in this regard is that firstly the intra-abdominal catheter becomes adhered to the visceral or parietal peritoneum through a localized inflammatory but soon thereafter fibrotic reaction [10]. At the interface between the shunt tubing and the respective visceral surface/abdominal wall an intense foreign body immune response occurs with neutrophilic degranulation and lymphocytic infiltration resulting in proteolysis and further fibrosis of the visceral/abdominal wall interface. As this process proceeds the visceral surface is weakened and is further augmented by the constant pressure exacerbated by the shunt tubing. Ultimately there is a full thickness erosion and, with regards any muscular organ, the subsequent muscular contraction pulls the shunt tubing into its luminal cavity [14]. Support of this pathophysiology is provided by the observation that since the introduction of more flexible tubing compared to the older more rigid tubing, such as the Raimondi spring loaded catheter [26], there has been a noticeable decrease in visceral and abdominal wall ventriculoperitoneal shunt erosion and subsequent shunt migration [15,16]. An additional factor taken from the literature, although the authors concerned do admit this to be their hypothesis and as such is to date unproven, is the length of intra-abdominal shunt tubing which they hypothesize to be an independent risk factor needing investigation in the pathophysiology of shunt migration [17].

Whilst this pathophysiology may explain the mechanism through which shunt erosion occurs into thoracic, abdominopelvic viscera or the abdominal wall, what this review of the Pubmed English literature found using the search words “ventriculoperitoneal shunt migration” or synonyms thereof, was a myriad of sites and presentations through which shunt migration may present [1;5;6;7;9;10;13;14;15;17;18;19;23;24].

Considering the abdominal wall itself comparing erosion and presentation through a potential anatomical weakness, for example the umbilicus,

Versus erosion and presentation through an intact abdominal wall, a recent comparative study considering the literature published over the last 20 years evaluated 31 cases of abdominal wall shunt migration. In this study the authors conclude there to be no statistically significant difference ($P>0.05$) in the incidence between shunt migration through a potential weakness in the abdominal wall and shunt migration through an intact abdominal wall [17]. A trend was however seen where 17 (55%) of cases had shunt extrusion through a potential weakness in the abdominal wall, dominated by the umbilicus, versus 14 (45%) of cases where the shunt eroded through and extruded through an intact abdominal wall [17]. In another case report detailing a case of shunt erosion and presentation through the umbilicus, possible causative factors are put forward to explain this occurrence and include firstly that put forward in the previous study namely a potential weakness of the abdominal wall with peristalsis driving the shunt tubing towards this site [17]. In addition an umbilical abscess, embryological remnants namely a persistent umbilical vein, and incomplete involution of the urachus, as well as omental inflammation are postulated to be additional predisposing factors [18]. While the umbilicus dominates as the principle site of shunt migration through the abdominal wall, isolated case reports do report additional sites. One such case report involved shunt tubing that presented through a patients back at the site of previous lumbar spinal fusion surgery [19].

Considering intra-abdominal shunt migration into hollow viscus's nothing exists in the Pubmed literature that specifically gives the site of shunt migration in decreasing order of frequency of occurrence. As such the various sites cannot be compared in order of frequency.

Intrathoracic migration through adherence and erosion through the diaphragm into the pleural space is recognized in several case reports as an established occurrence [20,21,22]. The occurrence of a hydrothorax in a patient with a ventriculoperitoneal shunt does not however dictate that this has occurred. The peculiarity of this association is due to the fact that a cerebrospinal fluid pleural effusion can also occur with a distal shunt appropriately placed and confirmed to be in the abdominal cavity [23]. A Pubmed review analyzing this exact issue reveals 21 case reports describing hydrothorax occurring in a patient with a ventriculoperitoneal shunt. Of these 60% describe the cerebrospinal fluid hydrothorax as occurring secondary to intrathoracic migration of the distal shunt tip, and, 40% describe hydrothorax occurring with a ventriculoperitoneal shunt appropriately positioned in the abdomen [22].

Considering bowel perforation and shunt migration by peristalsis ultimately protruding through the anus, the reported incidence is 0.1-0.7% of cases [24;25]. As opposed to other forms of ventriculoperitoneal shunt migration this form is perhaps the most serious and has a reported mortality of 15% due to the possible subsequent development of meningitis and brain abscess's [26]. Occurring almost exclusively in children, the thin bowel wall is recognized as the probable risk factor for the occurrence of ventriculoperitoneal shunt adherence and subsequent bowel wall perforation [28]. Albeit a rare occurrence, the seriousness of this specific complication has resulted in a proposal in the literature that, in children specifically, the distal shunt tip should be surgically anchored to the parietal peritoneum with non-absorbable suture. As a precautionary measure this intervention was noted to add little to the operative time and completely prevented ventriculoperitoneal shunt catheter migration from occurring in the study group [29]. At the other end of the gastrointestinal tract rare case reports also exist of ventriculoperitoneal shunt tubing adhering to the stomach or proximal small intestine and being vomited out of, and presenting in the mouth. This occurrence is very rare when compared to the numerous case reports of shunt tubing presenting through the anus and as per a review of the Pubmed literature this rare complication has only been reported 8 times [30].

Another very rare occurrence is ventriculoperitoneal shunt migration into the bladder lumen after adhering to and eroding through the intraperitoneal part of the bladder dome.

To date only 10 case reports of this occurrence have been reported as per a Pubmed review of the English literature [31,32,33,34,35,36,37]. Once within the bladder lumen the shunt tubing may present through the urethra [31,32,33,34,37], be associated with the development of a bladder calculus [38], or even rarer is a single case report of a ventriculo-peritoneal shunt tip perforating into the bladder lumen, knotting intra-luminally and subsequently presenting through the urethra [39].

Management of the various forms of distal shunt migration are dictated by the cornerstones of whether or not the shunt is obstructed and or infected. Other presentations include disconnection, pseudocyst formation, scrotal hydroceles, hydrothorax presenting as dyspnea and poor effort tolerance, all of which may need management in their own right [7;9;23].

Our experience

At our institution once a diagnosis of shunt migration is made we uniformly administer appropriate prophylactic antibiotics. We obtain a cerebrospinal fluid sample at presentation that is taken from the bulb of the ventriculoperitoneal shunt and sent for microscopy culture and sensitivity. Immediately thereafter we administer prophylactic antibiotic therapy. An antibiotic with gram positive cover namely cloxacillin 500mg administered intravenously 6 hourly, and gram negative cover namely intravenous ceftriaxone 1g administered 12 hourly are used. We add anaerobic cover in the form of metronidazole 500mg administered 8 hourly intravenously. Our local institutional susceptibility studies have shown an extremely low prevalence of methicillin resistance staphylococcus and hence we only administer vancomycin if methicillin resistant staphylococcus has been confirmed as the infecting organism on culture and susceptibility testing.

In a clean setting such as scrotal or thoracic migration, where the patient has no clinical nor hematological septic markers nor does he/she have raised polymorphs on the cerebrospinal fluid chemistry with a normal cerebrospinal fluid glucose, we simply revise the shunt by returning the migrated end into the abdominal cavity and for example closing the inguinal hernia at the same setting. We also inspect the contralateral side and will close this opening prophylactically if open. We consider a pseudocyst a marker of ventriculoperitoneal shunt sepsis and manage this accordingly.

In a contaminated or dirty setting, where the shunt tubing is exposed to the environment, we opt for removal of the whole shunt tubing including the ventricular catheter with or without placement of an external ventricular drain depending on whether or not the patient has increased pressure hydrocephalus. As a rule it is important not to pull the contaminated tubing back through a sterile peritoneal cavity and we transect and pull this part out distally. At our institution we do this through a mini laparotomy. The shunt tubing is sent to microbiology and the antibiotic therapy is continued. Our shunt tubing culture results are often polymicrobial and if the patient is responding well to the empiric antibiotic regimen, namely improving clinically with a falling daily c-reactive protein, we continue the empiric regimen. Any organism isolated on culture of the cerebrospinal fluid is treated immediately with directed antibiotic therapy. We repeat the CT scan at 3 day intervals and will place an external ventricular drain or re-insert a ventriculoperitoneal shunt once 3 negative daily cerebrospinal fluid cultures are demonstrated.

Overall these patients do well unless they develop ventriculitis and by adhering to a strict protocol for the management of shunt migration it is our experience that these patients do return to the community. Post discharge they are followed up closely in our out-patient department.

In conclusion shunt migration is a rare but interesting subject. Astute diagnosis and prompt management of the various forms by which this problem presents are cornerstones in the successful management of this rare complication.

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