atresia with tracheoesophageal fistula (TEF), increased cardio-thoracic ratio, Altman's type I SCT (***Fig.* 1**), and left multicystic kidney. Thoracotomy with staged repair of Vogt type3b was performed. Postoperatively patient developed sclerema and died. Echocardiography to confirm the presence of cardiac anomalies, and tumor markers for teratoma were not possible due to resource constraints. In addition to SCT, we made a diagnosis of VACTERL association owing to presence of three anomalies in our patient.

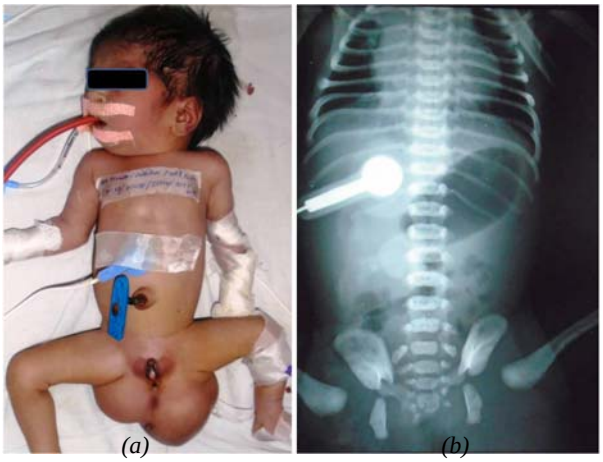

FIG. 1 *(a) Neonate with sacrococcygeal teratoma type 1,small perineum and vestibular fistula, with red rubber catheter not going beyond 10 cms into the esophagus; (b) radiograph showing dilated stomach shadow, soft tissue shadow in the sacrococcygeal region, normal vertebrae and increased cardiothoracic ratio.*

VACTERL association specifically refers to the structural abnormalities derivative of the embryonic mesoderm (disruption in the proliferation, migration and differentiation of mesoderm) [1]. Epiblasts cells migrating from primitive node and proximal part of primitive streak lead to the formation of notochord, paraxial and intermediate plate mesoderm [4]. Failure of some of these epiblasts cells to migrate will lead to remnants at primitive streak which may persist in sacrococcygeal region as a teratoma[4].

We propose that VACTERL association and SCT may be more than a chance association in our patient. Clinicians should have high index of suspicion for VACTERL association in a neonate presenting with sacrococcygeal teratoma and anorectal malformation.

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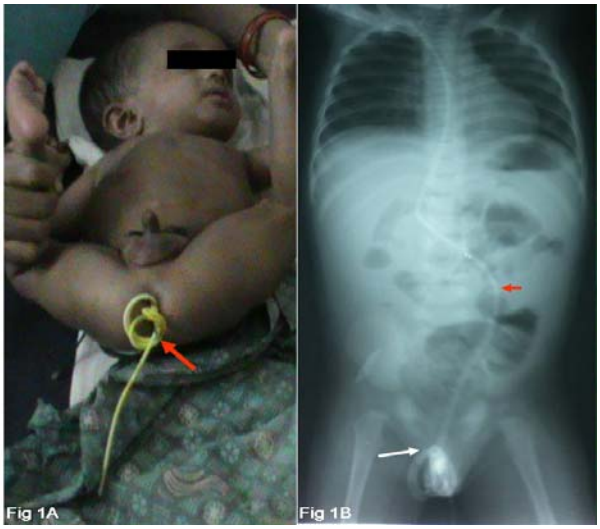
En-masse Protrusion of Ventriculo-peritoneal Shunt Tube Through the Anus

A 7-month-old boy, with a right-sided Ventriculo-peritoneal (VP) shunt *in-situ* for 2 months, presented with shunt tube protruding through anus for 2 hours. The infant was treated for acute diarrhea till 2 days ago. There were no signs of meningitis or peritonitis. Perineum showed a 'bunch of shunt coils' dripping cerebrospinal fluid (***Fig.* 1**). Abdominal *X*-ray showed point of entry of the shunt tube into the sigmoid colon with no pneumoperitoneum (***Fig* 1**). The shunt was divided through small subcostal incision; cranial end was removed and the peritoneal end was pulled out through anal opening.

Besides infection, malfunction, and CSF loculations, the shunt tube can migrate into any visceral organ [1]. Intestinal perforation caused by shunt procedures is rare, and about 50% occur in infants. Anal protrusion of shunt is an extremely rare complication [1,2].

Often, some surgeons keep sufficient length of shunt tube to accommodate for the linear growth of the baby by coiling the peritoneal end and securing the 'bunch of coils' with an absorbable suture in the supra-hepatic space so that shunt does not spread itself all over the peritoneal cavity between the intestinal loops. This decreases the chances of intestinal perforations and spontaneous knotting. Despite this effective technical



(a) *(b)*

***Fig.* 1** *(a) Infant with 'bunch of coils' of shunt tube protruding through the anus with securing sutures in situ (red arrow); (b) Abdominal X-ray showing point of shunt entry into the sigmoid colon (red arrow) and shunt coils lying in the perianal region(white arrow).*

modification to tackle peritoneal complications, anal protrusion still occurred in this child, and the entire 'bunch of intact coils' of shunt protruded *en-masse* through the anus without any peritonitis. Such protrusion should create a big rent in the eroded sigmoid colon or cause peritonitis but strangely there was none, suggesting that shunt erosion is a slow process where erosion and healing by shunt induced adhesions takes place simultaneously to conceal a free perforation. The shunt tip adheres and erodes the bowel by continuous friction, and is then propelled distally by peristalsis to protrude anally [3,4]; diarrhea may further aggravate the process of protrusion.

Mechanisms responsible for silent erosion and anal protrusion are multifactorial. Predisposing factors for anal protrusions are stiff shunt tube, thin bowel wall with strong peristalsis in infants, malnutrition, infection and foreign body reaction. Exaggerated peristalsis in diarrhea can predispose to *en-masse* protrusion of shunt coils. Redundant sigmoid colon is the most favorable site for shunt erosion and subsequent anal protrusion. Abdominal *X*-ray does not show pneumoperitoneum because shunt perforations are usually concealed. Anal protrusion of shunt without peritonitis is treated by percutaneous division and removal of the cranial end, and the peritoneal end is pulled out through the anus [3,5]. The perforation is usually sealed by a chronic fibrous sheath around the shunt track, and laparotomy is usually not required [3,5].

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