

MANAGEMENT OF INFECTED VP SHUNT IN CONGENITAL HYDROCEPHALUS, LOCAL EXPERIENCE

*¹Waseem Yousif Ali, ²Mahamed Natheer Khasro and ³Dr. Mohammed Ayad Almeran

¹M.B.CH. B/CABMS-Neurosurgery/Ibn-Sina Teaching Hospital/Mosul-Iraq.

²M.B.CH. B/CABMS-Neurosurgery/Ibn-Sina Teaching Hospital/Mosul-Iraq.

³M.B.CH. B/CABMS-Neurosurgery/University of Mosul-College of Medicine/Iraq.

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Corresponding Author: Waseem Yousif Ali

M.B.CH. B/CABMS-Neurosurgery/Ibn-Sina Teaching Hospital/Mosul-Iraq.

ABSTRACT

Objectives: The goal of current study was to look into the etiology, clinical characteristics, pathogens, mortality, morbidity, and treatment options for ventriculo-peritoneal (VP) shunt infections in people with congenital hydrocephalus. **Patients and methods:** Prospective case series descriptive study was accomplished in the Neurosurgery department at Mosul teaching hospital, from January 2011 to January 2013, the patients with VP shunts infection was studied Once infection was suspected, a cerebrospinal fluid (CSF) sample was taken and once infection confirmed. **Results:** The incidence of age: 69.44% below 1year. and 30.6% were between the age of 1-2 years. Time between the surgery and the shunt infection: 61.11% were presented within 6 months of operation and 38.88% presented between 6 months to 2 years. Death occurs in 13.8%. **Conclusions:** Gram-positive organisms were the commonest cause of shunt infections, (staphylococcus aureus and epidermidis). The timely usage of empirical antibiotic at the time of admission and appropriate antibiotics according to antimicrobial susceptibility testing afterwards, are essential for successful treatment.

KEYWORDS: Ventriculoperitoneal, Shunt, Infection, External ventricular drain.

INTRODUCTION

The Greek expressions "hydro" and "cephalus" imply "water" and "head," respectively. It's a situation distinguished by an excess of fluid that located in the brain, as the term means. The "water" in hydrocephalus is really cerebrospinal fluid (CSF), a transparent fluid that surrounds the brain as well as spinal cord. An inappropriate enlargement of the cerebral ventricles is result from an excessive buildup of CSF. This enlargement puts strain on the brain's tissues, which could be detrimental.^[1] The CSF channels and ventricles were discovered by Key and Retzius in 1875.^[2]

CSF production within the ventricles by choroid plexus that in turn split hydrocephalus into communicating were demonstrated by Dandy and Blackfan in 1914.^[2,3] Hydrocephalus can be caused by abnormalities in the inherited genetic, such as the aqueductal stenosis gene, or developmental problems, such as spina bifida in addition to encephalocele, which are both related with neural tube defects. Intraventricular hemorrhage, disorders such as meningitis, malignancies, subarachnoid hemorrhage, or traumatic head injuries all possible

causes.^[4] As a result, hydrocephalus can be either acquired or congenital.

Congenital hydrocephalus is a condition that appears at birth and is caused by events or effects that occur during fetal development, as well as genetic abnormalities. Acquired hydrocephalus can develop at any moment after birth or at a later date. There are two types of hydrocephalus: communicative and non-communicating.^[5]

Headache, nausea, papilledema, blurred vision, disturbed level of consciousness, problems with balance, poor coordination, gait disturbance, urinary incontinence, disturbance in the developmental progression, irritability, drowsiness, lethargy, or other personality changes or cognition difficulty, including memory loss, are all possible symptoms. Clinical neurological examination and cranial imaging techniques for instance, ultrasonography in addition to computed tomography (CT) and magnetic resonance imaging (MRI). Moreover, pressure-monitoring techniques are used to diagnose hydrocephalus.^[6]

For decades, many approaches for treating hydrocephalus have involved directing CSF from the sinus of brain to other body cavities for permanent drainage and absorption. Patients with hydrocephalus now have a better prognosis thanks to VP shunting, with many of them living normal lives and achieving normal intellect. In 1968, Nulson and Spitz invented a “one-way pressure-regulating valve” that they implanted in the atrium via the jugular vein, ushering in the contemporary shunting period. John Holter operated on the early development and improvement of the shunt valve as the archer of a hydrocephalic patient. Becker and Nulson established a new norm in hydrocephalus treatment and laid the road for today's standard, ventriculoperitoneal shunts, as a result of improved biomaterials such as silicone.^[7]

Complications can be encountered either in the immediate peroperative or in postoperative follow-up period. Complications associated with the VP shunt can occur somewhere along its path, from the ventricle at base of the skull to the peritoneal cavity caudally. Commonly encountered complications include: mechanical obstruction of distal peritoneal catheter by omentum or other structures leading to shunt malfunction, shunt infection, formation of abdominal pseudo cyst, spontaneous bowel perforation, intestinal obstruction, inguinal hernia and development of liver abscess, over shunting which may cause subdural hematoma, extrusion of components of shunt apparatus. Rare complications consist of movement of the peritoneal catheter into the stomach mucosa, liver, gallbladder, bowel, colon, diaphragm, urinary bladder, vagina, and scrotum.^[8]

Shunt systems, on the other hand, are not always flawless technologies. Infections, blockages, and the requirement to prolong or substitute the catheter are all possible complications. Shunt systems, in general, necessitate regular medical monitoring and follow-up. When issues arise, the shunt system will almost always need to be revised.^[9]

Although the early symptoms of shunt infection in children: fever, irritability are similar to many childhood illnesses; we must determine the symptoms associated with shunt failure in a particular individual. If we suspect there is a problem with the shunt, it is wise to have it checked rather than ignore it. It is preferable to have a false alarm examined than to ignore it. Although shunt problems can be life-threatening, they can nearly always be treated effectively if caught early. Although there is some association between the precise etiology of hydrocephalus and the result, the prognosis for persons diagnosed with hydrocephalus is difficult to predict. The presence of simultaneous illnesses, the appropriateness of diagnosis, in addition to the efficacy of treatment all complicates the prognosis. The extent to which reduction of CSF pressure during shunt surgery might minimize or reverse brain damage is unknown.^[10]

Hydrocephalus can harm a person's cognitive and physical development, thus affected people and their family should be aware of this. Many children with the disease, on the other hand, benefit from rehabilitative therapy and educational interventions and go on to enjoy normal lives with little restrictions. A positive outcome requires treatment from a multidisciplinary team of medical professionals, rehabilitation specialists, and educational experts. Persistent hydrocephalus can be lethal if left untreated. Due to the frequent and variable significant complications, a ventriculoperitoneal shunt procedure should only be performed if the patient cannot be treated with medicinal or surgical techniques.^[8]

Neurosurgeons expect that in the future, there will be alternatives to the ventriculoperitoneal shunt for the treatment of hydrocephalus. The surgical management line of hydrocephalus still relies on the implantation and revision of ventriculoperitoneal shunts.^[11,12] Obstruction and infection are the mainly frequent VP shunt consequences.^[13-15] After shunt installation, the incidence of infection was described to range from 2.2 percent to 39 percent.^[12,16-18] The age and general condition of the patient, the etiology of hydrocephalus, and the type of shunt implanted, and the surgeon's skill and technique of performing the treatment and post-operative care have all been linked to an increased possibility of infection.^[19]

Pathogenesis and Pathology

The organisms almost always come from the patient's skin, where they acquire access to the device after insertion.^[20, 21] These may be alternative sources if surgical asepsis is seriously compromised or the operating room environment or air is excessively contaminated. Although treatments such alcoholic chlorhexidine can reduce the bacterial population on the surface of the patient's skin to almost nothing, indigenous bacteria quickly recolonize. As a result, coagulase-negative staphylococci are frequently found in the incision during the surgery. These are inconsequential in nonimplant surgery, but they are highly likely to cling to and colonize a biomaterial or device if one is placed. In shunt infections, coagulase-negative staphylococci, notably *Staphylococcus epidermidis*, predominate. They grow and create enormous amounts of exopolysaccharide ('slime') after sticking to the shunt material, allowing a biofilm to form. Growth is exceedingly sluggish as most nutrients were depleted, which explains why there are sometimes extensive periods of time between surgery and the clinical appearance of infection. Unlike *Staph. epidermidis*, *Staphylococcus aureus* has a distinct clinical picture and is more commonly associated with external shunt infections. It also produces a-toxin, which shields it from phagocytosis.^[22]

Erythema and suppuration are also elicited by a very strong inflammatory response. Infection in VA shunts has a different clinical manifestation than infection in VP shunts. In the former, bacteria reach bloodstream directly, causing an intermittent fever that can last for

months or years with little other evidence of infection in infections caused by Staph. epidermidis, propionibacteria, or coryneforms. Antibodies against bacterial components, on the other hand, are produced in great quantities, and immunological complex illness might result, with C3, C4, IgG, and IgM deposits on synovial and glomerular basement membranes. Hypertension, renal failure (shunt nephritis), and arthropathy are all possible complications.^[23] The germs in VP and LP shunt infections are released into the peritoneal cavity, causing the larger omentum to close off the distal catheter. Shunt blockage and elevated CSF pressure result from this and related adhesions. Peritoneal abscesses are occasionally found. Ventriculitis occurs in the majority of cases with all types of shunts;

however the inflammatory response is usually weak. Only a small percentage of shunt infections are caused by factors other than surgery. Skin erosion over the shunt can occur in neonates or adults with poor nutritional status, resulting to subsequent infection with Staph. aureus or Gram-negative bacteria. Visceral perforation by the distal catheter is an unusual but well-documented cause of VP shunt infection, resulting in polymicrobial infection of the cerebral ventricles.^[24] Peritonitis caused by this etiology, however, is uncommon. There have been no reports of hematogenous dissemination, including to VA shunts. Cerebrospinal fluid shunts appear to be very safe from this danger. During abdominal surgery or continuous peritoneal dialysis, however, VP shunts might get contaminated.

Table 1: Clinical features of ventriculoperitoneal shunt infections of surgical origin.

Intermittent fever	<50%
Anorexia, lassitude, poor sleep pattern	>50%
Shunt obstruction	>75%
Other features	Chills, rigors: 20% Abdominal pain, bloating: >75% Arthralgia: 50% Swelling, erythema over shunt tubing: >60% Rash: 70% Headache, vomiting, etc. (i.e. recurrence of hydrocephalus): 75% Nephritis: 30%

The percentages represent the estimated proportion of cases with features. It's crucial to remember that each instance is unique, and many of these characteristics may be missing or altered. Bacteria and bacterial compounds that enter the peritoneal cavity through the shunt catheter trigger an inflammatory response in the larger omentum, which closes off the catheter outlet. The cyst that forms fills with CSF, causing the hydrocephalus to return. Noninfectious causes of cystic blockage can develop at any time, unlike infection-related cases, which usually manifest within 6–9 months of surgery.^[8] Many patients have a fever of low-grade that comes and goes, while some do not. Chills and rigors have been reported by some. Intermittent fevers are common. Muscle and joint problems, as well as a sore throat, are common complaints. Anemia is nearly universally found, while irritability, anorexia, lassitude, and poor sleep are all on the rise. Dyspepsia could potentially be an issue. Arthralgia becomes increasingly common as the disease advanced. Unfortunately, these characteristics are non-specific and are frequently confused with those of other diseases. Nephritis and vasculitis may develop as the disease advances.^[24]

The purpose of this study was to determine the rate and type of VP shunt infections, as well as the causative pathogens, in congenital hydrocephalus patients admitted to the Mosul teaching hospital's neurosurgical department, as well as to evaluate clinical features, management, and outcomes of patients with CSF shunt infections in congenital hydrocephalus.

Patients and Methods

This is a Prospective case series descriptive study was conducted upon 36 patients (17 boys and 19 girls), below 2years of age, having postoperative shunt infection after ventriculoperitoneal shunts in 262 patient with congenital hydrocephalus from the period starting at January 2011 till January 2013. This study was conducted in the department of neurosurgery in Mosul teaching hospital.

The patients with VP shunt inf. will be subjected to the following

1-Clinical assessment:

- A - Thorough medical history.
- B - Complete clinical examination.
- C - Neurological examinations.

2-Investigations:

- A - Routine laboratory investigations. (CBC ...).
- B - Radiological investigations including X-ray, CT brain.
- C - CSF analysis (Aspiration from disc or ventricular tap).

Protocol of VP shunt surgery in neurosurgical department of Mosul teaching hospital

In pediatric patients, a loading dose of 3rd generation cephalosporin ceftriaxone (50mg/kg/day) was given one hour before surgery as prophylaxis and continued for 48 hours subsequent to surgery, followed by oral antibiotic for 5 days. VP Patients who needed a shunt were given priority in the operating room. Shunt hardware was not removed from its sterile packaging until the tunneling

procedure was completed and the head and belly wounds were linked. Before opening the hardware, replace your gloves. While waiting to be inserted into the peritoneal cavity, the peritoneal catheter was wrapped in a sterile cloth.

To avoid air contamination, the head wound was promptly closed after clamping the burr hole valve. After surgery, sterile gauze padding and a crepe bandage are put to the head to prevent wound exposure and maintain uniform pressure on the flap to prevent serous fluid collection, which can lead to infection. A dressing was also applied to the abdominal wound. Unless there was drainage from the wound, the dressing was changed on the fifth day after surgery and subsequently on alternate days. On the eighth or tenth day post-operative, the removal of stitches was started. Medtronic California provided medium and medium-low pressure shunts, a Burr-hole valve shunt, codman shunts, and flat bottom shunts, as well as codman shunts and flat bottom shunts. At the discharging time, parents were given instructions on how to recognize shunt issues and their symptoms, particularly shunt infection.

The patients who presented with possible shunt infections were investigated according to timings of presentation at our hospital, age, symptoms, clinical features and clinical outcome. Microbiological biochemical, in addition to hematological variables were also evaluated. Shunt infection was suspected with following clinical findings: fever, recurrent vomiting, decreasing of consciousness level, irritability, seizures, tense fontanelle, and neck stiffness, redness and local infection ant the site of tube or upper end or abdomen. A CSF sample was collected aseptically from the reservoir

before been sent for routine investigation, gram staining analysis, and culture sensitivity testing. In the absence of culture and sensitivity data, empirical intravenous antibiotics (vancomycin and third generation cephalosporin) were started. CT brain with and without contrast was also performed and compared with previous CT scan, if available.

Once CSF infection had been confirmed.

The management protocol consisted of

- The removal of the infected shunt and EVD system putted and antibiotic and put new shunt after CSF culture clear in 21 patients.
 - Or without removal of V-P system just antibiotic and tapping if needed in 9 patients.
 - Use of IV antibiotic and wound debridement ,re-stitching and dressing without shunt removal in 4 patients presented with wound infection, and 2 patients with shunt exposed need new shunt system
 - Use of intraventricular amikacin or gentamicine in 5 patients with ventriculitis.
- Lastly, Ethical approval was gained before collection of patient data.

RESULTS

A total of 262 patients with congenital hydrocephalus were operated for ventriculo-peritoneal shunt from the period January 2011-january 2013. Thirty six patients (13.74%) readmitted with symptom and sign of shunt infection, and the result as follows for the infected shunt.

Age distribution; 15 (41.66%) are below 6 months, 10 (27.77%) are between 6-12 months, and 11 (30.56%) are between the age of 1-2 years.

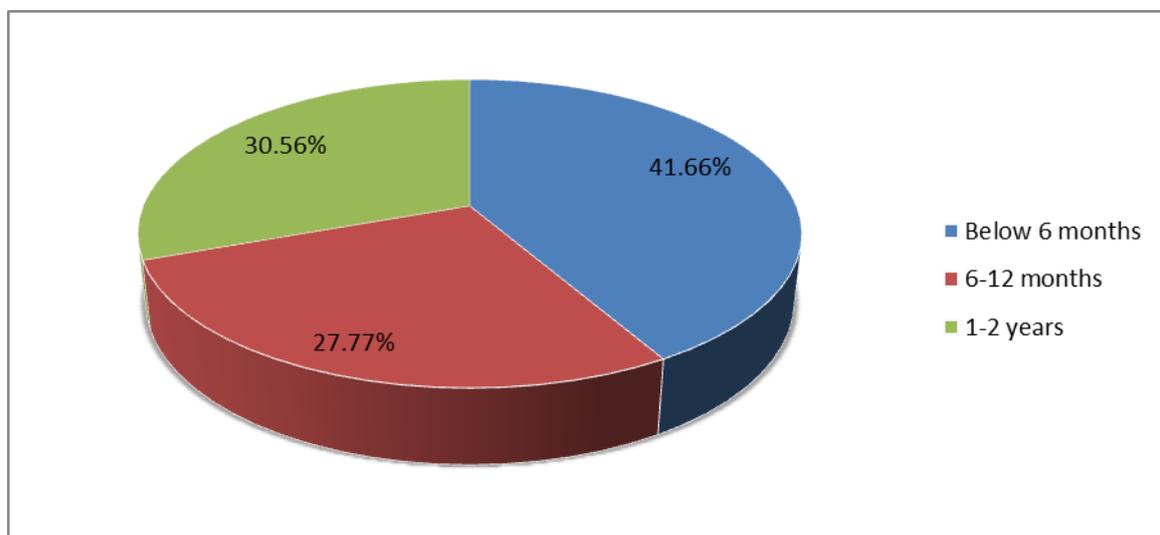


Figure 1: Age distribution of the study sample.

Time between the surgery and the shunt infection

Figure (2) shows that 22 (61.11%) patients are present within 6 months of surgery while 14 (38.88%) patients are present between 6 months to 2 years.

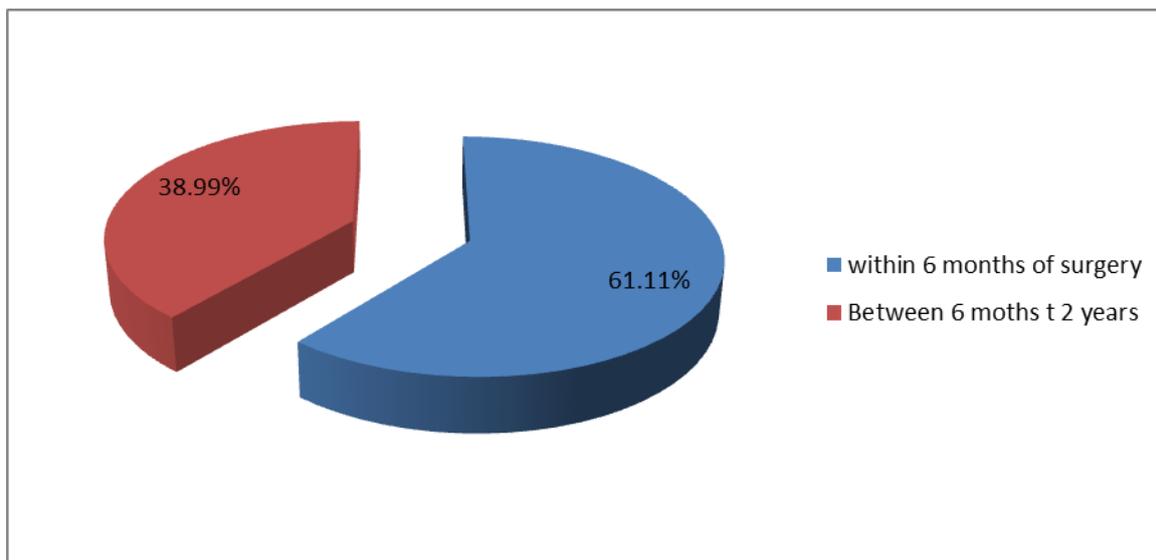


Figure 2: The time between surgery and shunt infection in the study sample.

Hospital admission

Single admission with shunt infection is found in 30 patients representing (83.3%). Patients who improved and discharged well are 27 (90%) and only 3 (10%) are

dead. Multiple admissions is noticed in 6 (16.66%) patients, 4 (66.66%) patients are improved and 2 (33.33%) patients are dead as shown in fig (3).

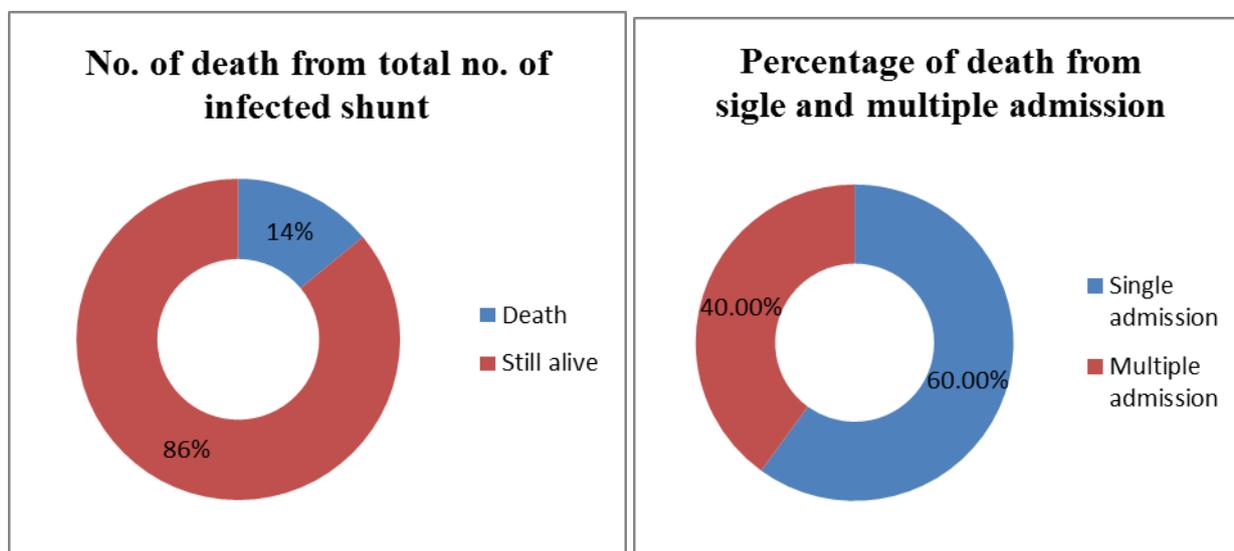


Figure 3: The fate of infected shunts in the study sample.

Table 2: The relationship between number of admissions and fate of shunt infection in the study sample.

No. of admissions	Outcome		Total No. (%)	P-value*
	Survived and improved No. (%)	Died No. (%)		
Single	27 (90.0)	3 (10.0)	30 (83.3)	0.186
Multiple	4 (66.7)	2 (33.3)	6 (16.7)	
Total	31 (86.2)	5 (13.8)	36 (100.0)	---

* Fisher Exact test was used.

Patients divided in 3 main groups

Group1: Six patients (16.66%) of the infected patients presented with wound infection, partial wound dehiscence, stitch abscess and were treated with intravenous antibiotics, wound debridement, re-stitching and dressing without removal of VP shunt, except 2

patients with shunt exposed needed for shunt replacement, those patients had negative CSF examination.

Group2: Twenty one patients (58.33%) (With positive CSF examination) of the infected patients had removal of shunt and insertion of external ventricular drain with

periodic CSF sampling and culture sensitivity and delayed shunt replacement after 3 negative culture results.

Group3: Nine patients (25%) (Positive CSF examination) of the infected patient treated without shunt removal just repeated tapping (as needed) and antibiotic.

Table 3: The relationship between method of management of infected shunt and fate of shunt infection in the study sample.

Mode of management	Outcome		Total No. (%)	P-value*
	Survived and improved No. (%)	Died No. (%)		
Shunt removed or replaced (group II & 2 of group I)	25 (92.6)	2 (7.4)	27 (75.0)	0.051
Shunt reside (group III & 4 of group I)	6 (66.7)	3 (33.3)	9 (25.0)	
Total	31 (86.2)	5 (13.8)	36 (100.0)	---

*Chi Square test

Intraventricular injection (amikacin or gentamicine) used in 5patients with ventriculitis, 3 patients from group2: 2patients improved, one died. Two patients from group3: the two patients improved.

Causative microorganism identified was *Staphylococcus aureus* in 14 (two died), *Staphylococcus epidermidis* in 9 cases (no death), Gram negative bacilli in 7 (two died) cases, *Pseudomonas spp* in 2 (one died), no growth of organism was found in 4 cases (11.11%).

Five patients died (13.8%); no patient from group1; two patients from group2; three patients from group3.

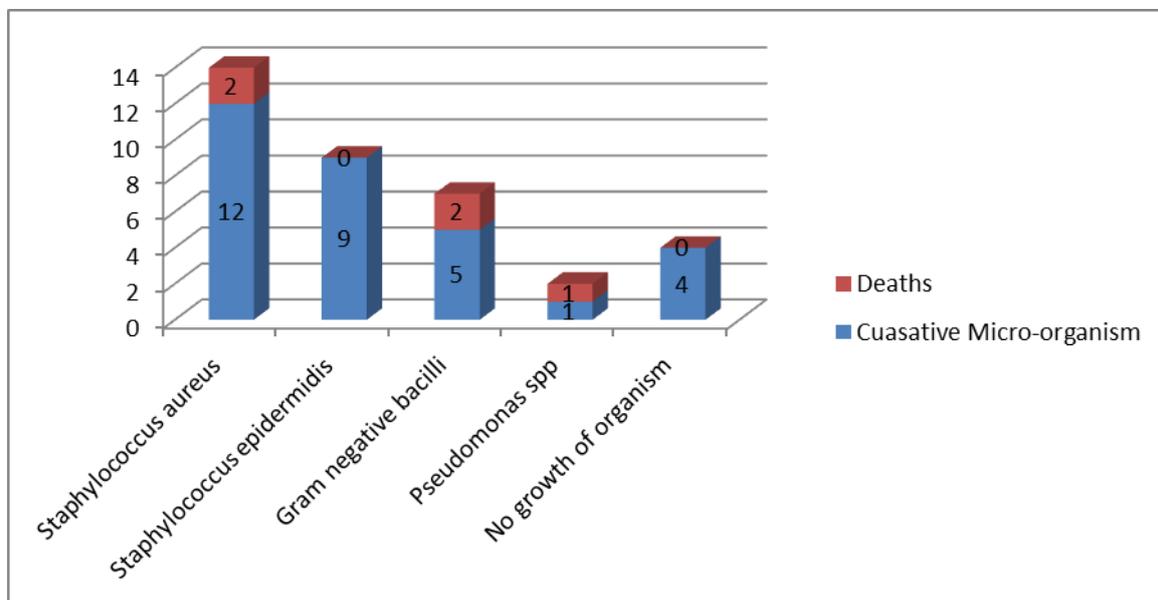


Figure 4: The causative microorganism in the study sample.

Radiological findings were shown in fig. (5) which, illustrates that Shunt dysfunction and hydrocephalus 26 (44.8%), Ventriculitis 5 (8.6%), Multiloculate ventricles 2(3.5%), Subdural effusion 1 (1.7%), Periventricular edema 15 (25.9%), and Normal CT 9(15.5%).

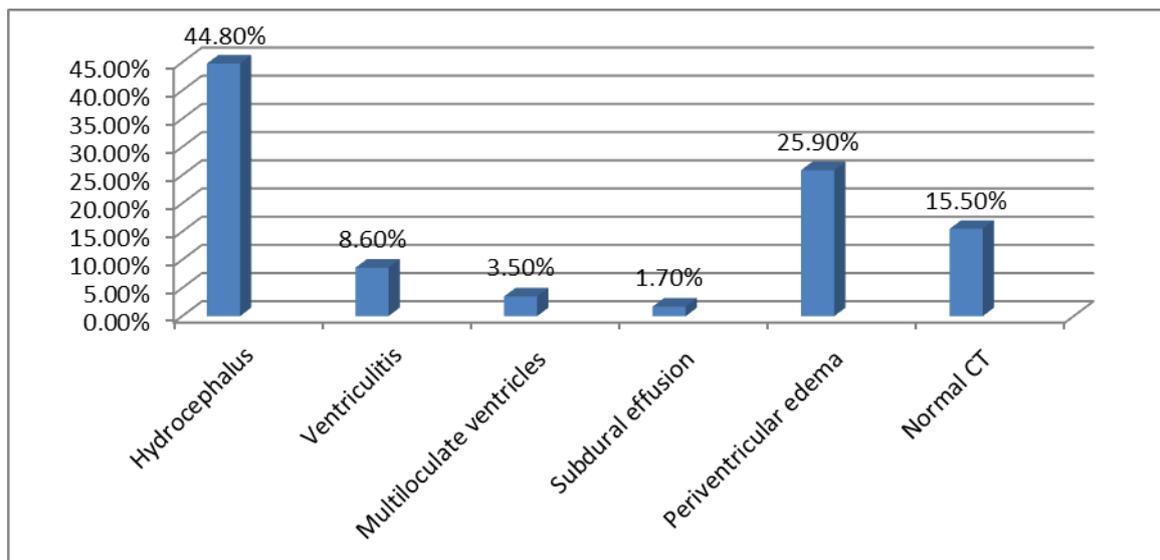


Figure 5: The radiological findings of infected VP shunt children in the study sample.

DISCUSSION

Infections form the most common complication of shunting procedures in children and remain the most frequent source of morbidity and mortality.^[25, 26] The way of shunt infections is not known for certain, but many neurosurgeons and pediatricians believe that in most shunt infections the causative organism spreads and reach the surgical wound either directly from the adjacent skin or by gloves or instruments contaminated with the patient's flora during the operation.^[27, 28]

A shunt infection occurs when bacteria or fungi grow on the plastic parts of the shunt system.^[29, 30]

The occurrence of the majority of infections within the first 30 weeks after surgery supports the hypothesis that many shunt infections result from perioperative contamination.^[31]

The time of onset of infection after shunt insertion ranges from 1 month to 2 year, but a great majority of the cases occur within the first 3 months (80%) and 90% within 6 months.

In our study, most of the shunt infections occurred within the first 6 months (61.11%) and the most common pathogens cultured in the blood and CSF were *S. aureus* and *S. epidermidis*.

Cultures of CSF aspirated from the shunt were more frequently positive than blood cultures; we attributed this to the possibility that the patient had received antibiotics before admission. These results agree with the theory that there is a link between the shunt's insertion and the later development of infection; that is, organisms introduced during the perioperative period (probably via a cutaneous source) are a key determinant of staphylococcal shunt infections.^[32, 24]

The physician must be highly suspicious of shunt infections because of the paucity of reliable clinical signs. Fever as well as vomiting were the most common nonspecific symptoms in our series.

Fever is a consistent finding, but frequently it is unheeded by parents and physicians for several weeks – a common cause of delay of diagnosis.

Patients with ventriculoperitoneal shunts should be examined thoroughly when erythema overlying the shunt tubing is detected, because routine cultures may be negative despite an ongoing shunt infection. Due to the significant cost, morbidity, and the occasional mortality associated with these infections and their treatment, effective prevention is the key to reducing the incidence of these infections. Alterations in the hardware, changes in operating techniques as well as modifications in sterile techniques in the operating theater have all been suggested. Certainly, the most discussed method involves the administration of antibiotics as a preventative measure during the procedure.^[31,33-35] Anti-staphylococcal antibiotics are often given intravenously just after the induction of anesthesia and before incision and they are discontinued postoperatively. Vancomycin is often used for neurosurgical prophylaxis^[31,33], but it is not used routinely for fear of developing resistant strains and losing the most effective antibiotic against methicilline-resistant staphylococcus strains.

However, in our hospital, we used 3rd generation cephalosporin for prophylaxis because the operations involved a second or third manipulation of the central nervous system and reinfection carried a high morbidity and mortality risk for these patients.

The high social and financial cost of infections of VPS leads us to seek effective measures of prevention. But though there are many studies on the VPS infections,

with various forms of prevention, remaining work statistically consistent.^[36]

Choux *et al.*^[37] describe the decline in the rates of infection from catheter per patient, in his unit, 15.56% to 0.33%, after implementation of a protocol for prevention of these infections. This protocol involved actions in all perioperative period. The patients were prepared meticulously, the infections were treated, and they underwent hair shampoo the evening before and the morning of the procedure using povidine preparation. The VPS operations were performed early in the day prior to other neurosurgical procedures, they were programmed to last from 20 to 40 minutes, and no more than four VPS surgeries were accomplished a day. The number of people in the surgical room was limited to four: an experienced surgeon in shunt implantation, his/her assistant, the anesthetist and the circulating nurse.

The appropriate equipment was chosen by the surgeon, in agreement with the etiology of the hydrocephalus and with the patient's age. This sterile device was opened minutes before the implant to avoid larger time of exposition, and immediately immersed in a gentamicine bath. Only two small skin incisions were made without an intermediate incision, beginning for the abdominal.

The preassembled shunt was placed in position and the exposed distal catheter was covered by a sterile drape during cannulation of the lateral ventricle and insertion of the proximal catheter.

The valve was placed carefully in the subgaleal area to avoid skin lesions and equipment damage, and the distal catheter was placed into the peritoneal cavity under direct visualization.

Finally, the incision of the skull was sutured in a single layer using interrupted sutures and the abdominal suture was closed in layers in classic manner. In the surgical induction all patients received 100mg/kg of oxacillin. Antibiotics were not administered in the postoperative, and the curatives were changed every 2 days under sterile technique. On average, admission of patients was for 4 days in a primary shunt, and hospitalized for 2 days for shunt revision. With this protocol the authors lowered their infection rate for patient from 15.56% to 0.33% and for procedure from 7.75 % to 0.17%.

Other papers demonstrate reduction in the infection rate by the utilization of shunt catheters impregnated with antibiotics, as rifampicin or clindamicin. The prophylaxis with endovenous third generation cephalosporins (ceftriaxone) has also shown effectiveness in this subject.^[38, 39]

According to Schreffler *et al.*^[40] study of available data from published research, the optimal treatment for infection of CSF shunt appears to be the removal of this shunt along with systemic antibiotics, followed by the

installation of a new shunt as CSF sterility is established. This therapy option has the highest cure rate (86%) as well as the lowest rates of failure and mortality. EVD has a lower complication risk than other treatment alternatives, with a complication rate of less than 35.8%. Shunt removal and quick replacement should be attempted if an EVD complication rate of more than 35.8 is expected or if the EVD method is not possible.^[33, 41-44]

Vancomycin can be administered intraventricularly or intrathecally for the management of meningitis. Some clinicians recommend that vancomycin be administered intraventricularly only if the CSF is not sterile after 48 hrs of intravenous therapy.^[45-47]

The results of the treatment of infection with antibiotics alone were significant, as these patients had been admitted very early on and their symptoms were not serious. Urgent antibiotic administration may prevent the progression of infections, so early application is very important for success and the avoidance of invasive procedures. However, the number of reported cases who have received antibiotic treatment alone are low, and more data are needed for a correct assessment of the cure rate. Because of their high incidence and associated morbidity, infections of CSF shunts remain a significant problem in neurosurgery. Detecting these infections early and treating them aggressively reduces morbidity and mortality. Prevention, however, will ultimately yield the greatest decline in morbidity. Shunt insertion should include prophylaxis with an intravenous anti-staphylococcal agent capable of penetrating the CSF, as staphylococci cause the majority of the episodes.

CONCLUSIONS

Gram-positive organisms were the commonest cause of shunt infections, (staphylococcus aureus and epidermidis). The timely usage of empirical antibiotic at the time of admission and appropriate antibiotics according to antimicrobial susceptibility testing afterwards, are essential for successful treatment.

Recommendations

- ❖ VP shunt should be inserted under strict meticulous and aseptic techniques.
- ❖ In case of well-known infection of VP shunt, it is critical to remove VP shunt, put external ventricular drain and initiate systemic antibiotics and substitute it with new hardware after having 3 negative cultures and when patient is hemodynamically stable.

REFERENCES

1. Aschoff A, Kremer P, Hashemi B, Kunze S. The scientific history of hydrocephalus and its treatment. *Neurosurgical Review*, 1999; 22 (2-3): 67–93.
2. Rizvi R and Anjum Q. Hydrocephalus in children. *J Pak Med Assoc*, 2005; 55: 502-506.
3. Dandy WE and Blackfan KD. Internal hydrocephalus: An experimental, clinical and

- pathological study. *Am J Dis Child*, 1914; 8: 112-116.
4. ReKate HL and cherney WB. *Pathophysiology, Diagnosis and clinical features of hydrocephalus in infants and children*. In: Tindall GT, Cooper PR, Barrow DL, Eds. *The practice of Neurosurgery*. Baltimore, Williams and Wilkins, 1996; 3: 1219-1223.
 5. David MF and Nalin G. Hydrocephalus, pediatric neurosurgery. *Landes bioscience publishing*, 2006; 6: 118-120.
 6. Abdelkader AS. Postoperative complications of ventriculoperitoneal shunt. A thesis presented in master degree in neurosurgery to Ain shams university, 2011; 3-4.
 7. Becker DP and Nulsen FE. Control of hydrocephalus by valve-regulated venous shunt: Avoidance of complications in prolonged shunt maintenance. *J Neurosurg*, 1968; 28: 215-226.
 8. Borkar SA and Satyarthee GD. Spontaneous Extrusion of Migrated Ventriculoperitoneal Shunt Catheter Through Chest Wall: A Case Report. *Turkish Neurosurgery journal*, 2008; 18(1): 95-98.
 9. Siddharth K, Shuck J, Kapoor S, Williams MA, Rigamonti D. Hydrocephalus Association. Selection of patients with idiopathic normal-pressure hydrocephalus for shunt placement: a single-institution experience. *Journal of Neurosurgery*, 2009; 1-10.
 10. Tamburrini G, Caldarelli M and Di Rocco C. Diagnosis and management of shunt complications in the treatment of childhood hydrocephalus, *Reviews in neurosurgery*, World federation of neurosurgical societies, 2008; 1(3): 31.
 11. Sacar S, Turgut H, Toprak S, Cirak B, Coskun E, Yilmaz O, *et al.* A retrospective study of central nervous system shunt infections diagnosed in a university hospital during a 4-year period. *BMC Infect Dis*, 2006; 6: 43.
 12. Gathura E, Poenaru D, Bransford R, Albright AL. Outcomes of ventriculoperitoneal shunt insertion in Sub-Saharan Africa. *J Neurosurg Pediatr*, 2010; 6: 329-335.
 13. Ali M, Khan A, Khan H, Khwanzada K. Short term complications of ventriculoperitoneal shunt in children suffering from hydrocephalus. *J Pediatr Neurol*, 2009; 7: 165-169.
 14. Rehman A, Rehman T, Bashir HH, Gupta V. A simple method to reduce infection of ventriculoperitoneal shunts. *J Neurosurg Pediatr*, 2010; 5: 569-572.
 15. Conen A, Walti LN, Merlo A, Fluckiger U, Battagay M, Trampuz A. Characteristics and treatment outcome of cerebrospinal fluid shunt-associated infections in adults: A retrospective analysis over an 11-year period. *Clin Infect Dis*, 2008; 47: 73-82.
 16. Wong GKC, Poon WS, Wai S, Yu LM, Lyon D, Lam JMK. Failure of regular external ventricular drain exchange to reduce cerebrospinal fluid infection: result of a randomized controlled trial. *J Neurol Neurosurg Psychiatr*, 2002; 73: 759-761.
 17. Crnich CJ, Safdar N, Maki DG. Infections associated with implanted medical devices. In: Finch RG, Greenwood D, Norrby SR, Whitley RJ (Editor). *Antibiotic and chemotherapy: Anti-Infective agents and their use in therapy*. 8. Churchill Livingstone, 2003; 575-618.
 18. Sarguna P and Lakshmi V. Ventriculoperitoneal shunt infections. *Indian J Med Microbiol*, 2006; 24: 52-54.
 19. Bisno AL, Sternau L. Infections of central nervous system shunts. In: Bisno AL, Waldvogel FA (Editor). *Infections associated with indwelling medical devices*. American Society for Microbiology, Washington, 1994; 91-109.
 20. Bayston R, Lari J. A study of the sources of infection in colonised shunts. *Dev Med Child Neurol*, 1974; 16(Suppl.32): 16-22.
 21. Shapiro S, Boaz J, Kleiman M, Kalsbeck J, Mealey J. Origins of organisms infecting ventricular shunts. *Neurosurgery*, 1988; 22: 868-872.
 22. Bayston R. *Hydrocephalus shunt infections*. London: Chapman and Hall Medical, 1989.
 23. Bayston R and Swinden J. The aetiology and prevention of shunt nephritis. *Zeit Kinderchir*, 1979; 28: 377-384.
 24. Brook I, Johnson N, Overturf G, Wilkins J. Mixed bacterial meningitis: a complication of ventriculo- and lumbo-peritoneal shunts. *J Neurosurg*, 1977; 47: 961-964.
 25. Lee JY, Wang KC, Cho BK: Functioning periods and complications of 246 cerebrospinal fluid shunting procedures in 208 children. *J Korean Med Sci*, 1995; 10: 275-280.
 26. Yogev R: Cerebrospinal fluid shunts infections: A personal view. *Pediatr Infect Dis*, 1985; 4: 113-118.
 27. Winston KR: Hair and neurosurgery. *Neurosurgery*, 1992; 31: 320-329.
 28. Pople IK, Bayston R, Hayward RD: Infection of cerebrospinal fluid shunts in infants: A study of etiological factors. *J Neurosurg*, 1992; 77: 29-36.
 29. Bayston R and Penny SR. Excessive production of mucoid substance in Staphylococcus SIIA: A possible factor in colonisation of Holter shunts. *Dev Med Child Neurol*, 1972; 27: 25-28.
 30. Chiou C, Wong T, Lin H, *et al.* Fungal infection of ventriculoperitoneal shunts in children. *Clin Infect Dis*. 1994; 19: 1049-1053.
 31. Ronan A, Hoog GG, Klug GL. Cerebrospinal fluid shunt infections in children. *Pediatr Infect Dis*, 1995; 14: 782-786.
 32. Schmidt K, Flemming G, Osgaard O, Huldberg E, Kristiansen J, Dahlenup B, *et al.* Antibiotic prophylaxis in cerebrospinal fluid shunting: A prospective randomized trial in 152 hydrocephalic patients. *Neurosurgery*, 1985; 39: 1-5.
 33. Schoenbaum SC, Gaardner P, Shillito J. Infections of cerebrospinal fluid shunts: Epidemiology, clinical

- manifestations, and therapy. *J Infect Dis*, 1975; 131: 543–55.
34. Quigley MR, Reigel DH, Kortyna R. Cerebrospinal fluid shunt infections: Report of 41 cases and a critical review of the literature. *Pediatr Neurosci*, 1989; 15:111-120.
 35. Schmidt K, Flemming G, Osgaard O, Huldberg E, Kristiansen J, Dahlenup B, *et al.* Antibiotic prophylaxis in cerebrospinal fluid shunting: A prospective randomized trial in 152 hydrocephalic patients. *Neurosurgery*, 1985; 39: 1-5
 36. Pattavilakom A, Xenos C, Bradfield O, Danks RA. Reduction in shunt infection using antibiotic impregnated CSF shunt catheters: An Australian prospective study. *Journal of Clinical Neuroscience*, 2007; 14: 526-531.
 37. Choux M, Genitori L, Lang D, Lena G. Shunt implantation: reducing the incidence of shunt infection. *J Neurosurg*. 1992; 77: 875-880.
 38. Pattavilakom A, Xenos C, Bradfield O, Danks RA. Reduction in shunt infection using antibiotic impregnated CSF shunt catheters: An Australian prospective study. *Journal of Clinical Neuroscience*, 2007; 14: 526-531.
 39. Gupta N, Park J, Solomon C, Kranz DA, Wrench M, Wu YW. Long-term outcomes in patients with treated childhood Hydrocephalus. *J Neurosurg*, 2007; 106: 334-339.
 40. Schreffler RT, Schreffler AJ, Wittler RR. Treatment of cerebrospinal fluid shunt infections: A decision analysis. *Pediatr Infect Dis*, 2002; 21: 632–636.
 41. James HE, Walsh JW, Wilson HD, Connor JD, Bean JR, Tibbs PA. Prospective randomized study of therapy in cerebrospinal fluid shunt infection. *Neurosurgery*, 1980; 7: 459–463.
 42. Walters BC, Hoffman HJ, Hendrick EB, *et al.* Cerebrospinal fluid shunt infection: Influences on initial management and subsequent outcome. *J Neurosurg*, 1984; 60: 1014–102.
 43. Shurtleff DB, Foltz EL, Weeks RD, *et al.* Therapy of Staphylococcus epidermidis infections associated with cerebrospinal fluid shunts. *Pediatrics*, 1974; 53: 55–62.
 44. Sells CJ, Shurtleff DB, Loeser JD, *et al.* Gram negative cerebrospinal fluid shunt-associated infections. *Pediatrics*, 1977; 59: 614–618.
 45. Haines SJ. Efficacy of antibiotic prophylaxis in clean neurosurgical operations. *Neurosurgery*, 1989; 24: 401–405.
 46. Swayne R, Rampling A, Newsom SWB: Intraventricular vancomycin for treatment of shunt associated ventriculitis. *J Antimicrob Chemother*, 1987; 19: 249-253.
 47. Kaufman BA. Infections of cerebrospinal fluid shunts; in Scheld WM, Whitley RS, Durack DT (eds): *Infections of the Central Nervous System*. Philadelphia, Lippincott-Raven Publishers, 1997; 129: 555–577.