Section: Physical Medicine & Rehabilitation

Case Series

CHALLENGES IN MANAGING MEDICAL EMERGENCIES IN SPINAL CORD INJURY PATIENTS ADMITTED FOR NEUROREHABILITATION: A RETROSPECTIVE CASE SERIES

Arvind Kumar Sharma[1], Ivanah P Nongrum[2], Satyasheel Singh Asthana[3]

[1]Assistant Professor, Department of Physical Medicine & Rehabilitation, AIIMS Raebareli, India.
2PG Resident, Department of Physical Medicine & Rehabilitation, AIIMS Raebareli, India.
3Assistant Professor, Department of Physical Medicine & Rehabilitation, AIIMS Raebareli, India.

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Corresponding Author:
Dr. Arvind K Sharma,
Assistant Professor, Department of Physical Medicine and Rehabilitation, AIIMS Raebareli, India.
Email: draksusha2015@gmail.com

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ABSTRACT

Background: Spinal cord injury (SCI) refers to damage to the spinal cord due to traumatic or non-traumatic causes resulting in sensory or motor deficits with bladder and bowel dysfunction. Chronic SCI is often associated with a risk of developing complications. The study highlights the fact that these patients are prone to sudden medical problems and emergency situations due to their injury, which can have a substantial impact on their quality of life and functional independence. Hence awareness of the various medical emergencies along with their diagnosis and timely management is necessary to avert further complications and mortality.

Material and Methods: Eight patients with spinal cord injury, during the past two years have been included in this retrospective case series. These patients were admitted for neurorehabilitation and were found to develop spinal cord injury-related complications during their hospitalization.

Results: Out of eight patients, two were having sepsis with quick sequential organ failure assessment (qSOFA) score of two for which they were stabilized and given intensive care The complications included Heterotopic Ossification, urosepsis, Autonomic Dysreflexia, Appendicitis, grade four Pressure Injuries with sepsis, and Pneumonia. All patients received timely identification, accurate diagnosis, and prompt treatment, in conjunction with the standard neurorehabilitation protocol. As a result, they all achieved favorable functional recovery.

Conclusion: When SCI patients show clinical evidence of fever, increased spasticity, bladder symptoms, decreased range of motion of any large joint, deranged baseline vitals or laboratory reports, differential diagnoses should be kept in mind while trying to search etiology or precipitating factors. Early diagnosis and treatment must be carried out in order to reduce morbidity and avoid mortality as they may have altered or absent sensations below their level of injury, making it challenging to recognize medical emergencies in a timely manner. The importance of preventing, early diagnosing, and effectively treating these complications cannot be overemphasized, as it can improve the patient's survival, community participation, and overall health-related quality of life.

Key Words: spinal cord injury, physical medicine and rehabilitation, emergency medicine, intensive care, pressure injury, autonomic dysreflexia, sepsis.

INTRODUCTION

Spinal cord injury (SCI) is defined as an acute traumatic lesion of the spinal cord resulting in any degree of sensory/motor deficit or bladder/bowel dysfunction temporarily or permanently.[1] It refers to damage to the spinal cord resulting from trauma (from falls and road traffic injuries) or non-traumatic causes like tumours, degenerative and vascular conditions, infections, toxins or birth defects. The extent of SCI related impairment depends on injury severity and location in the spinal cord. SCI results in sensory and/or motor impairment below the neurological level of injury (NLI).[1] It is associated with a number of disabilities, economic impact and life changes, adding to the burden of the patient and caregivers. In India, about 1.5 million of the population live with SCI. On an average, around twenty thousand new spinal cord injury cases are added annually.[2] Sixty to seventy percent of them are part of the rural population of the country and are faced with issues such as poverty and illiteracy, majority being male and in the age group of sixteen to thirty years.[2] Thus, there is a higher incidence in the young, active working age group of the population, thereby adding to the economic and social burden of the country.[2] In other countries, road traffic accidents and fall from height have been seen to be the most common causes of SCI. Fall from more than ten feet and road traffic accidents are seen in younger population. Older population, already having osteoporosis, commonly develop SCI due to fall from a lower height.[3]

Management of acute SCI begins immediately following injury. Mortality at the time of initial injury ranges from 48% to 79% with an additional 4.4% to 16% deaths occurring prior to hospital discharge.[4] Patients in the acute stage of SCI need to be aggressively monitored to attenuate morbidity and mortality arising from complications.[4] SCI is often associated with a risk of developing complications which requires emergency care including spasticity, pain, urinary tract infections, pressure injuries, pneumonia, Autonomic dysreflexia, Osteoporosis, Deep vein thrombosis. Additionally, SCI patients may develop clinical signs of depression, impairing their progress in neurorehabilitation and adversely affecting their general health.

Mortality risk is highest in the first year after injury, remaining high compared to the general population. Injury level and severity, availability of timely, quality medical care, transfer method to hospital after injury and time to hospital admission are important factors.

MATERIALS AND METHODS

Patient inclusion criteria

In this case series, eight patients were included with whom emergencies occurred due to complications associated with SCI, during the course of their hospitalization. The patients included were in the age group of 18-75 years. All patients had been admitted for neurorehabilitation in the Department of Physical Medicine and Rehabilitation at All India Institute of Medical Sciences, Raebareli where patient information, complete physical examination and routine laboratory investigations including blood and urine tests were done to record baseline values. Skiagrams, Ultrasonography (USG) whole abdomen or kidney ureter bladder (KUB) and other radiological imaging were done as and when required. Written and informed consent was obtained from all the participants for usage of clinical, pathological and radiological data for research purposes and publication.

RESULTS

Case 1

Diagnosis: Seventeen-month-old elsewhere operated case of traumatic spinal cord injury with spastic tetraplegia with neurogenic bowel and bladder with subacute appendicitis. NLI: C7-ASIA **Impairment scale (AIS) Grade D**: 43-year-old male gave history of a fall from a height and was admitted for neurorehabilitation. No previous history of any acute abdominal emergency or surgery. On day 23 post admission, the patient complained of diffuse abdominal pain. On examination, pulse was 102/min, BP was 104/62 mmHg. Patient was afebrile and had right iliac fossa tenderness. On examination, tenderness was present over McBurney's point. Bowel sounds were present in all quadrants. Abdominal girth was 69cm. There was no abdominal distension, no complaints of vomiting. Ultrasound (USG) whole abdomen was done showing 8mm thickened and edematous wall, suggestive of subacute appendicitis. Alvarado scale was calculated to be 3/10 (migrating pain and tenderness in right lower quadrant were present). General Surgery reference was taken and advice followed. Complete blood count (CBC) report showed normal leukocyte count (7.12 x1000/l) with neutrophilic predominance (85%). CRP was 1.73 mg/dl. Renal function test (RFT) and Liver function test (LFT) were normal. Patient was kept nil per oral (NPO) and IV fluids along with appropriate IV antibiotic medication after sensitivity testing were administered. Patient was passing stools and voiding urine. Abdominal pain reduced after three days. He was kept NPO for three days after which oral sips of water followed by liquid diet was started. Vitals monitoring, input-output and abdominal girth charting was done. Patient was observed and monitored in the ward for the next ten days during

which his condition improved and he continued with his neurorehabilitation. Patient was discharged with stable vitals and improved Functional independence measure (FIM) score from 60 to 108.

Case 2

Diagnosis: Six-month-old elsewhere managed non traumatic spinal cord injury (cause: Potts spine) with paraplegia with neurogenic bladder and bowel with Grade II pressure injury on sacrum and unstageable pressure injury over right greater trochanter with Pneumonia with Diabetes mellitus.

35-year-old female was admitted for neurorehabilitation. MRI was done elsewhere and she had been diagnosed with Pott's spine: vertebral level T6 and was taking anti-tubercular treatment (ATT) for the last three months. Patient was on per urethral catheterization for bladder emptying for the last three months.

On admission, the patient was afebrile, tachycardic (pulse 116/min) and her blood pressure (BP) was 130/86mmHg, respiratory rate (RR) was 14/min and oxygen saturation (SpO2) at room air was 98%. Routine blood and urine investigations were done along with chest x-ray, ECG. Debridement followed by thorough cleaning of the wound over the right greater trochanter was done under aseptic precautions. Empirical antibiotics were started after taking blood and pus samples from all wounds. At 10pm on the day of admission, the patient had a sudden decrease in SpO2 for which oxygen inhalation was started at 2 litres/min which was increased up to 3 litres/min with SpO2 at 100% using nasal prongs. On auscultation of the chest, bilateral air entry along with bilateral fine crepts were present. Chest x-ray revealed presence of right upper lobe fibrosis. Injection Hydrocortisone 100mg IV was given stat and nebulisation was done with Budesonide 2cc. Pulmonary medicine and General medicine references were sought. D dimer and coagulation profile was done to rule out Pulmonary embolism secondary to deep vein thrombosis, which was normal. ESR was 100mm/hr, CRP >6.5mg/dl and PCT 0.137 ng/ml. To exclude any cardiac manifestation ECG and Troponin I (0.012ng/ml) was also done. Oxygen support with nasal prongs along with IV antibiotic medication after sensitivity testing were advised. Oral hypoglycaemic agents were stopped and the patient was given insulin subcutaneously using sliding scale. Quick sequential organ failure assessment (qSOFA) was calculated to be 2. HRCT thorax films of the patient showed fibrobronchiectatic changes in the medial segment of the right middle lobe and basal segments of the right lower lobe with partial collapse of right middle lobe, suggestive right middle lobe syndrome. Expert opinion from Department of General Medicine and Pulmonary Medicine was taken and patient was diagnosed with sepsis and required intensive care unit (ICU) care. Patient was then referred for admission in ICU for haemodynamic stabilisation.

Case 3

Diagnosis: Seven-month-old elsewhere operated case of traumatic spinal cord injury with paraplegia with neurogenic bowel and bladder with unstageable pressure injury over sacral area, Grade II pressure injury over left gluteal region. NLI: L1, AIS Grade B

21-year-old female presented with complaints of on and off fever and developed a wound over the lower back seven days back which gradually increased in size over the one week prior to admission. She was admitted in PMR ward for neurorehabilitation.

On admission, patient was on Foley's and dressing over sacral wound was soaked and foul smelling. On day two after admission, the patient was febrile with a temperature of 102°F, pulse was 106/min, BP was 110/64mmHg, RR was 19/min. Chest, on auscultation, was clear with equal bilateral air entry. Unstageable pressure injury (PI) 10 x 5 x 2 cm over sacral area covered by blackish eschar and foul-smelling necrotic debris, Grade II Pressure injury over left gluteal region, 4x4cm, no discharge, clear margins, healthy granulation tissue were present. FIM at admission was found to be 96/126. Hb was 6.6g/dl with a raised TC of 13.8x1000 per microlitre with a neutrophilic predominance (68%). Serum iron was <10microgram/dl, raised Ferritin of 154ng/ml. ESR was 100mm/hr, raised CRP >6mg/dl, PCT was 1.69 ng/ml. ALP was 142 U/l, GGT 51.9 IU, Total protein was 5.68g/dl. The patient underwent transfusion with one packed red blood cell (PRBC) following which her Hb improved to 8.7g/dl. TC continued to be raised ($25.69x10^3/\mu L$). General Surgery reference was sought for wound management and debridement under local anaesthesia was done, following which, pus and tissue culture were sent and appropriate antibiotics were administered as per sensitivity reports. Urine and blood culture was found to be sterile. Fever subsided, Hb improved (9g/dl), cleaning and dressing of the wounds were done under aseptic precautions. Patient was started on high protein diet with Iron supplementation. Caregivers of the patient were taught aseptic precautions while doing the cleaning and dressing of the pressure injuries. Vitals were stable at the time of discharge with healthy granulation tissue in both wounds. FIM was 100/126.

Figure 1 indicates the progression of the pressure injuries as seen on follow up visits after discharge

(PI over lower back: Healed Grade IV; PI over Left gluteal region: Healed Grade II)

Case 4

Diagnosis: Six-month-old elsewhere conservatively managed case of traumatic SCI (cause - fall from height; Fracture C6 vertebra) with quadriplegia with neurogenic bowel and bladder with healing grade IV pressure injury over sacral region; NLI C4, Vertebral level C6, AIS Grade A

25-year-old male patient was admitted for neurorehabilitation and on admission had stable vitals. The pressure injury over the sacral region was Grade IV with dimensions of 17 x 10 cm, foul smelling with greenish discharge. Chest, on auscultation, was clear with no added sounds. His foleys catheter was last changed 10 days before admission, turbid urine was present in the urobag, there was poor hygiene at the catheter insertion site. Hb was 10g/dl, ESR 100mm/hr, CRP of 5.69mg/dl, PCT was 0.053ng/ml. The sacral pressure injury was debrided surgically under aseptic precautions following which pus culture was taken. On day seven post admission, on assessment patient was febrile with a temperature of 101.4degF, pulse of 110/min, BP 100/62mmHg, SpO2 98%. Pus culture was positive, for which appropriate antibiotics were started as per sensitivity reports. Cleaning and dressing of the wound was done under aseptic conditions and his father was taught the same to continue at home. Urine culture was sterile. Patient was discharged with stable vitals and with a healthy pressure injury having granulation tissue.

Case 5

Diagnosis: Seven-month-old elsewhere operated case of traumatic spinal cord injury with neurogenic bowel and bladder with Grade III pressure injuries over sacral region and over bilateral greater trochanters. Sensory level T10, motor level T10, NLI: T10. AIS grade A.

48-year-old male was admitted for neurorehabilitation. On initial assessment, pulse was 78/min, BP 116/78mm Hg. Patient and his caregivers were explained about the condition and prognosis. Dimensions of Grade III PIs over sacral region: 7cmx3cm, over left greater trochanter: 5cmx3cm, over right greater trochanter: 4cmx2cm. Daily cleaning and dressing of the wounds were done under aseptic conditions.

During the course of his hospitalisation, three weeks post admission, the patient developed a fall in blood pressure along with increased respiratory rate. The condition of the patient was assessed and fluid resuscitation was started in the ward itself. Blood and urine investigations along with pus and blood cultures were sent, opinion from Medicine department was taken and their advice was followed.

There was no improvement in his BP despite fluid resuscitation, due to which inotropic drugs were started to maintain BP and to prevent multiple organ dysfunction syndrome (MODS). Renal function tests showed deranged electrolytes and raised creatinine. The patient was assessed and qSOFA was calculated as two. In order to avoid further deterioration and to prevent impending MODS, the patient was transferred to the ICU for further care. The patient was diagnosed with septicaemia and was in septic shock. He underwent PRBC transfusion in view of low Hb 7g/dl. Patient was treated aggressively with fluid resuscitation, with strict input output monitoring and inotropic Noradrenaline. On day 3, inotrope support was weaned off gradually, following which his condition had improved and he was stable. ESR was 60mm/hr and CRP >6mg/dl. Pus culture from the three wounds and blood culture reports were positive for which appropriate antibiotic medication was administered as per sensitivity reports after calculating creatinine clearance. PCT was initially 15.3ng/ml. During the course of the ICU stay, a decreasing trend of PCT was observed, where it came down to 3.19ng/ml before shifting out the patient to the PMR ward. Patient was then managed in the PMR ward during which PCT came down to

0.84ng/ml, renal and liver function tests normalised, total count decreased, blood and urine cultures were sterile. Standing with support in walking frame, strengthening exercises were taught to the patient. Patient was discharged with stable vitals, healthy pressure injuries having granulation tissue and a decreased surface area. Patient was given bilateral hip knee ankle foot orthosis (HKAFO) for therapeutic standing and home-based neurorehabilitation program.

Case 6

Six-month-old case of non-traumatic spinal cord injury (cause: psammomatous meningioma WHO grade I) operated (D10 D11 Laminectomy with excision of intradural extramedullary tumour) with paraplegia with neurogenic bowel and bladder. Sensory level T10, Motor level T10, NLI T10.

36-year-old female was having an indwelling catheter. Catheter insertion site was clean and personal hygiene was good. On initial assessment, pulse was 85/min, BP 110/78mmHg, afebrile and was admitted for neuro-rehabilitation. On day 12 after admission, the patient developed fever with chills. On examination, pulse was 106/min, BP 112/88mmHg, Temperature 101.4 °F. Blood and urine investigations were done. ESR was 100mm/hr, PCT was 1.74 ng/ml, TC of 15.06x1000/µL, Hb of 8.7g/dl. Urine routine analysis showed numerous pus cells. Urine culture was positive for which IV antibiotic medication was started. Patient was started on Ferrous salt with folic acid orally as well. Patient stopped having fever episodes from day 2 after starting the antibiotics. Ultrasound (USG) showed suspicion of vesical calculus for which Urology reference was sought. Once the patient recovered, clamping of the catheter was done and Tablet Oxybutynin 2.5mg was started to increase the bladder filling capacity. Eventually, Foley's was removed and the patient was taught reflex voiding. Patient was discharged with stable vitals and was advised to continue bladder management as taught.

Case 7

Diagnosis: Three-month-old elsewhere managed traumatic spinal cord injury (cause: pedestrian hit by four-wheeler) with traumatic brain injury (right frontal bone fracture with extradural haemorrhage) with blunt trauma to the chest with multiple ribs fracture with neurogenic bowel and bladder with paraplegia. AIS grade C. Sensory level: T3, Motor level: T3, NLI: T3.

44-year-old female patient was admitted for neurorehabilitation. On initial assessment, vitals were stable, patient was afebrile, chest was clear with bilateral equal air entry, Foley's catheter was in situ. Clamping was initiated following which Foley's was removed and she was able to void by self. On day eight after admission the patient developed fever with chills. Patient was tachycardic with pulse of 108/min, BP of 128/88mmHg, RR 13/min and Temperature of 101.8deg F. Blood and urine investigations were sent along with urine culture. TC 6000/microlitre with neutrophilic predominance (72%), raised CRP of 4.070mg/dl, urine culture was positive for which appropriate antibiotics were administered as per sensitivity reports. USG showed mild concentric urinary bladder wall thickening with significant post void residual volume (40ml) for which Tablet Tamsulosin 0.4mg was started. Fever subsided and vitals stabilised following which she continued with her progress in neurorehabilitation where therapeutic standing was initiated.

Case 8

Diagnosis: 9-month-old elsewhere operated case of spinal cord injury (fracture Body of T6 T7 T8 T10 decompression with pedicle screw fixation) with paraplegia with neurogenic bowel and bladder with Heterotopic Ossification (HO) with Autonomic Dysreflexia (AD). Sensory level – T6 Motor Level – T6, NLI – T6. AIS Grade B. FIM at admission 90/126.

17-year-old male was admitted for neuro-rehabilitation. He also complained of restriction in movement of his left hip in the past 4 months. On initial examination, pulse was 88/min, BP 116/78mmHg, RR 18/min, SpO2 99% and he was afebrile. Chest was clear and abdomen was soft with normal bowel sounds. It was the third day since he had last changed his catheter, urine draining in the bag was clear and the catheter insertion site was clean. He gave history of occasional rise in BP for which he had previously consulted in the department of General medicine. Routine blood and urine investigations were sent along with radiological investigations. Pelvis skiagram with both hip joints showed development of lamellar bone in surrounding peripheral joint region. Hb was 6g/dl, Serum iron was 30.1 microgram/dl, ESR 100mm/hr, CRP 6.01mg/dl. The patient was transfused with 1 PRBC without any complications. Four days later, Hb had improved to 8.6g/dl. On day seven of admission, the patient developed a rise in BP: 144/92mmHg, pulse was 100/min. Along with restricted ROM at the left hip, there was increasing spasticity in the Hamstrings and Gastrocnemius and Soleus. The patient was given Tablet Cilnidipine 10mg with Telmisartan 40mg as advised by the department of General Medicine. Continuous vitals monitoring was done, in order to ensure that there was no recurrence of AD. The patient continued with his neurorehabilitation, where coordination and balance exercises, wheelchair training, bladder and bowel management, transfer training, therapeutic standing with bilateral Hip knee ankle foot orthosis (HKAFO) and bilateral lower limb traction was applied. His vitals were monitored cautiously.

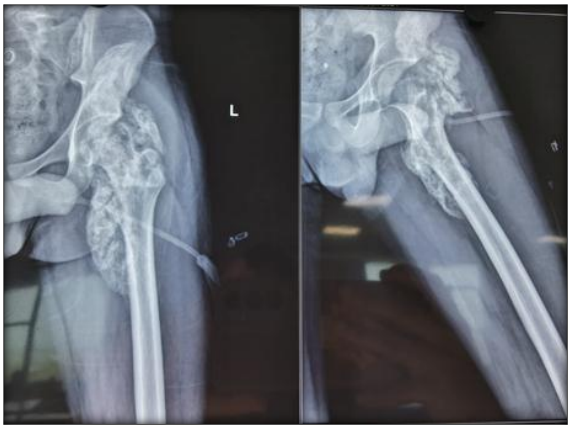

Figure 2: Indicates Heterotopic ossification in case 8

DISCUSSION

Complications developing subsequent to sustaining injury, contribute to the mortality rate of SCI patients. Disease of the respiratory system, mainly pneumonia, is the leading cause of mortality among SCI patients, accounting for 21.9% deaths as reported to the National Spinal cord injury Model systems database (NSCID) in 2015 [5]. This is followed by infectious diseases (12.9%) including occurrence of pressure injuries, urinary tract infections along with many others, leading to sepsis. This is followed by Hypertensive and ischaemic heart disease (10%), followed by other heart diseases causing unexplained heart attacks (8.4%).[6] From a critical care standpoint, stabilisation of the patient is the main concern following a traumatic event. From a rehabilitation perspective, this period is also vital for implementation of time-sensitive interventions that can limit secondary injury and prevent or minimize complications.[7] Complications occurring during the course of SCI can result in life-threatening emergencies, which need to be addressed aggressively and at times, ICU care may also be required.

Our study contains a brief account of some of the emergencies along with their management and prevention in a rehabilitation setting of a tertiary care centre. These included cases of pneumonia, sepsis occurring due to pressure injuries, urosepsis, and abdominal emergencies, heterotopic ossification and autonomic dysreflexia. To identify these, thorough physical examination, record of baseline values of blood, urine investigations and radiological imaging is essential, so as to identify the focus of infection and immediately manage it.

Pneumonia- Respiratory infections

Respiratory complications, especially pneumonia and respiratory failure, are the major causes of death in SCI patients. They mostly occur in the first year following injury.[5] Pneumonia in acutely injured SCI patients is more common and catastrophic in patients with cervical or high thoracic injuries and in geriatric age group. In spinal cord injury patients, due to respiratory muscle fatigue associated with defective sensations and altered perceptions, pneumonia may very easily progress to respiratory failure associated with sepsis.[8] Risk of developing pneumonia is greater in the post-injury period, due to the fact that SCI patients commonly do not have an effective cough. If phrenic and intercostal nerves are affected, it impairs the respiration cycle, thus increasing their vulnerability to develop pneumonia. In case of intubation or mechanical ventilation, there is a high risk of developing ventilator associated pneumonia (VAP). The most common organisms responsible for pneumonia in SCI patients are Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumonia, Pseudomonas aeruginosa, Klebsiella pneumonia, Acinetobacter baumannii, Serratia marcescens and Methicillin-resistant Staphylococcus aureus (MRSA).[9] For patients with NLI C1-C4 AIS scale A to C tetraplegia, pneumonia accounts for 30% of rehospitalizations during follow-up. If NLI is at C5 or rostral, patient could have weakness or complete paralysis of diaphragm muscles. Expiratory muscle weakness, causing a reduced peak cough flow ineffective for clearing bronchial secretions, is more common among SCI patients, contributing to case fatality by pneumonia. Expiratory strength increases with each additional neurological level and is normal at T12 or below.[9]

Prevention

- Pulmonary toileting to remove retained secretions
- Proper positioning and cough assistance to clear secretions so as to prevent aspiration pneumonia
- Use incentive spirometry postoperatively, remobilise the patient out of bed whenever feasible

Treatment: Symptomatic management of patients is done. Antibiotics are administered based on sensitivity reports for endotracheal secretions culture. For empirical treatment, combination therapy of antibiotics was used.[9] Thus, pneumonia and other respiratory infections occurring as complications in SCI patients should be ruled out in patients with fever and other clinical signs of sepsis. Timely management can help to decrease morbidity and mortality.

APPENDICITIS

Most patients do not present with the classically described history or physical findings and some may not have any abdominal discomfort early in the disease process. Other differentials of acute abdominal pain need to be ruled out. Clinically, patients may present with changes in bowel habits, intermittent, crampy, abdominal pain in the epigastric or periumbilical region. Appendicitis should be kept in mind as a differential diagnosis for pain in abdomen for all patients, unless patient has history of removal of the appendix organ.[10]

In patients with SCI, majority acute abdominal emergencies are delayed in their diagnosis. This

occurs because of two reasons. One being absence of somatic and visceral sensation due to disruption in the normal visceral and somatic pathways responsible for recognition of symptoms. Additionally, SCI patients have other comorbid conditions such as urinary tract infection, gastroparesis, constipation, decubitus ulcers that may mask or present with similar signs of appendicitis. Appendicitis and other acute intra-abdominal conditions can become serious events calling for emergency care in patients with SCI and are reported to account for 10 percent of all deaths in this patient population.[11] Acute abdomen in SCI patients is a not a very common complication and is often misdiagnosed or delayed in being identified, which can predispose the patient to complications like intestinal perforation, small bowel obstruction and autonomic dysreflexia.[12]

In adult population, Alvarado scale (MANTRELS) is used to diagnose appendicitis based on clinical evidence of migrating pain in right lower quadrant (M), anorexia (A), nausea/vomiting(N), right lower quadrant tenderness(T), rebound pain(R), elevated temperature of more than 37.3°C (E), leukocytosis (L), leukocyte shift to the left (Ls) on a scale from one to ten. Sonographic diagnosis of acute appendicitis can be made if a noncompressible appendix is visualised with anteroposterior (AP) diameter of or greater than 6mm. Other USG findings include single wall thickness of or more than 3mm, target sign, free fluid surrounding the appendix, local abscess formation, peritoneal wall thickening, signs of secondary small bowel obstruction.[13]

SCI patients have chronic abdominal distension. Low grade fever can be due to respiratory infections, urinary tract infections, pressure injuries and the initial symptoms are very subtle including general unwell feeling, fatigue, decreased appetite. It may be associated with changes in pulse rate, fever, dull aching abdominal pain, pyrexia, cardiac arrhythmias. Nausea and vomiting can indicate development of bowel obstruction.[11] Patients with a high NLI (above T6) may also present with symptoms of AD including pounding headache, sweating, increased BP. Thus, it is essential to pay heed to nominal complaints of SCI patients and regularly monitor their vitals so as to avoid further complications as silent abdomen can lead to sepsis causing mortality.

Pressure Injuries: Pressure injury (PI) is defined as 'localized damage to skin and underlying soft tissue, usually overlying a bony prominence or caused by contact with a medical device or another object, which develops because of intense and/or prolonged pressure or pressure combined with shearing.[14]' Despite recent advancements in the management of complications of SCI and other bedridden patients, pressure injuries continue to remain one of the most common and debilitating complications seen. Patients of SCI are at risk of skin breakdown, thus making them vulnerable to developing PIs, thereby contributing to difficulty in mobility, community involvement, hampering their progress in neurorehabilitation. Approximately 8% of patients of acute SCI developing pressure injuries succumb to the severity of their complications.[14] 50-80% SCI patients develop pressure injuries at some point of time in their life.[15]

Management of Pressure Injuries

Patient and caregivers have to be educated and counselled about the condition and prognosis of the patient. They need to be educated about etiology, risk factors, proper positioning, skin care and when to seek medical attention. Skin over bony prominences need to be examined multiple times and skin should be kept clean and dry, especially for patients with neurogenic bowel/bladder. Frequent change of posture and weight shifting causes redistribution of the weight of the body which allows reperfusion of the tissues, thus preventing necrotic changes. Additionally, maintaining a nutritionally complete diet and appropriate body weight, avoiding smoking, and avoiding alcohol intake also helps in cutting down on the risk factors contributory to formation of PIs. National Pressure Injury Advisory Panel (2016) guidelines are used to stage pressure injuries.[16] Debridement is necessary to remove necrotic tissue and slough. It can be done mechanically, surgically, autolytic, enzymatic and biological methods are also present. In general, stage I and II PIs are usually treated with local care non surgically where conventional dressing with 0.9% Normal Saline is done daily under aseptic conditions. Stage III and IV PIs have a high recurrence and are often chronic non-healing ulcers, hence may require surgical intervention. A positive wound culture report does not necessarily act as an indication for administering systemic antibiotics, unless the patient shows clinical evidence of systemic sepsis, spreading cellulitis or underlying osteomyelitis.

Vacuum Assisted Closure (VAC)/ Negative Pressure Wound Therapy (NPWT) is used to enhance healing process of Grade III and IV pressure injuries and applies negative pressure at the site of the wound, thus decreasing the edema at that site.[17]

URINARY TRACT INFECTIONS (UTIs)

UTIs are the most common secondary complication seen in SCI patients. It is a major contributory factor to the development of vesicourethral reflux, renal calculi, obstructive uropathy and nephropathy, autonomic dysreflexia, hydronephrosis, bladder cancer and chronic renal failure.[18] Symptomatic UTI is clinically seen as presence of increased bacterial urine colony counts, pyuria in terms of raised white blood cell count and presence of new onset symptoms such as fever, cloudy urine, suprapubic discomfort, burning micturition, lethargy, urgency, incontinence and specifically for SCI patients- increasing spasticity and vulnerability to develop autonomic dysreflexia.[19] Complications arising from lower UTIs include epididymitis,

prostatic or scrotal abscess, sepsis, ascending UTIs. Upper UTIs can cause pyelonephritis, renal deterioration, renal calculi, papillary necrosis, bacteraemia and sepsis.[18]

Vesical and renal calculi are the second most common urological complication seen in the SCI population. According to a previous study in SCI population, the prevalence of vesical calculi was found to be 11% in those with suprapubic catheters, 5% in those with indwelling catheter. 36% of SCI population develop urolithiasis within the first eight years post-injury.[20] It has been noticed that those with indwelling catheters commonly have calcified pubic hair or residual stone from an indwelling catheter, which becomes a nidus around which the calculi are formed. Failure to treat bladder stones increases susceptibility to develop autonomic dysreflexia which can be life-threatening. Bladder stones can be visualized using abdominal X-ray, bladder USG, CT scan and cystoscopy. Caution during USG should be taken so as to not over-distend the bladder, as it can lead to urinary tract infections, urinary sepsis and AD.[21] Bladder stone management includes gentle irrigation with normal saline till clear, cystoscopy with aspiration, lithotripsy, open surgery, dissolution with citric acid, glucono deltalactone and magnesium carbonate solution.[21]

HETEROTOPIC OSSIFICATION (HO)

It is defined as the formation of extraosseous lamellar bone in soft tissue surrounding peripheral joints located below the NLI in SCI patients, causing restricted range of motion. It causes a reduction in function affecting around 10 to 20 % of SCI patients and resulting in ankylosis for 5-8% of them.[22]

Clinical presentation: It develops at three to twelve weeks after injury, peaking in incidence at two months. It affects the hip, followed by knee, elbow, shoulder and very rarely in small joints of the hands and feet. It may present as:

- Fever
- Joint swelling
- Limited range of motion
- Effusion if involving the knee

Complications of HO include

- Loss of function
- Difficulty sitting in a wheelchair secondary to reduced Range of motion (ROM)
- Chronic pain
- Development of pressure injuries
- Deep venous thrombosis
- Increased spasticity
- Compromise of neurovascular structures causing distal extremity swelling and nerve entrapment.[23]

Diagnosis of HO,[23]

- Elevated serum alkaline phosphatase (ALP)- non-specific but earliest laboratory indicator, preceding radiographic changes
- Elevated creatine phosphokinase (CRP)- more reliable marker
- Elevated non-specific markers of inflammation: Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP)
- Urinary excretion of hydroxyproline and collagen metabolites
- Triple phase bone scan (radiolabelled pyrophosphate bone scintigraphy)- most sensitive imaging
- Standard x-rays
- Ultrasonography showing echogenic peripheral zone and echo lucent center
- MRI- due to increased T2 signal (edema) in muscles, fascia and subcutaneous tissue
- CT scan

Treatment of HO

- ROM exercises with gentle stretching
- Avoid aggressive ROM as additional tissue microtrauma can induce increased degrees of HO
- Gentle mobilisation is encouraged to prevent additional loss of ROM
- Medications: Bisphosphonates and Non-steroidal anti-inflammatory drugs (NSAIDs), if not contraindicated
- Bisphosphonates block late mineralisation phase in bone formation, thereby decreasing the rate of new bone formation. Etidronate 20mg/kg/day is given for six months if CPK is normal. If not, then it is given 20mg/kg/day for three months following which 10mg/kg/day for the next three months.[24]
- Radiation therapy
- Surgical excision (later in the course)- reserved for patients with severely limited ROM/ functional limitations/ predisposition to skin breakdown.[23]

AUTONOMIC DYSREFLEXIA

Spinal cord injury at the level T6 or above predisposes the patient to Autonomic Dysreflexia (AD). It is defined as a sudden and significant rise in the blood pressure, both systolic and diastolic, classically associated with bradycardia. Blood pressure rise of more than 20 to 40mmHg above baseline is an indicator of AD.[25]

Mechanism: AD is triggered by noxious stimuli which cause sympathetic hyperactivity. Any stimuli below the level of injury can trigger this response, including overdistended bladder, manipulation of indwelling catheter, faecal impaction, pressure injuries, ingrown toenails, tight clothing, stress fractures, detrusor sphincter dyssynergia (DSD), ejaculation, labor and delivery, appendicitis, erosive gastritis, gastric and duodenal ulcers, cholecystitis, Heterotopic ossification, deep venous thrombosis, joint dislocation, pulmonary emboli. In spinal cord injury patients, inhibitory impulses arising above the neurological level of injury (NLI) are blocked, thus leading to unopposed sympathetic outflow. Another contributory factor to AD in SCI patients includes

denervation hypersensitivity of peripheral adrenergic receptors below the NLI.[25]

Clinical features:

Signs and symptoms due to hyperactive sympathetic outflow include

- Sudden and significant rise in blood pressure
- Pallor
- Piloerection

Compensatory parasympathetic response causes vasodilatation above the NLI resulting in

- Pounding headache
- Nasal congestion
- Flushing of skin above NLI
- Miosis of pupils
- Bradycardia (Tachycardia can occur)

Complications that can occur include

- Cardiac arrhythmias
- Seizures
- Intracranial bleeding,[26]

Management of Autonomic Dysreflexia

a) Prevention of AD

- Patient and caregiver education about recognizing signs and symptoms of AD and to seek emergency care

b) Non pharmacological management: Removal of noxious stimuli,[27]

i. Bladder management program- regular bladder emptying, prevention of overdistension of bladder, regular urological assessment, treatment of urinary tract infections and calculi

ii. Prevention of faecal impaction- effective elimination, minimizing noxious stimuli during bowel evacuation, prevention of constipation, haemorrhoids and anal fissures

iii. Loosening of tight clothing or constrictive devices

iv. In case of seizures arising from AD, patients should be placed in left lateral position, tongue bite should be monitored and prevented.

v. In pregnant females with NLI at or above T6, Epidural anaesthesia during labour and delivery prevents AD.

c) Pharmacological treatment: Antihypertensives with rapid onset and short duration are preferred

- Nitroglycerin ointment (2%) applied on the chest or back, easy to remove in case of hypotension, contraindicated in patients on Phosphodiesterase-5 inhibitors
- Nifedipine 10mg- patients are instructed to bite and swallow its contents, can be repeated after 10-15minutes, contraindicated in geriatric population and those with known cardiovascular disease.
- Hydralazine 10mg
- Captopril 25mg sublingual
- Prostaglandin E2 agents: Diazoxide, Phenoxybenzamine, Clonidine, Mecamylamine
- Prazosin relaxes blood vessels and has been used to treat AD in doses of 0.5 to 1mg twice or thrice daily

The patient's vitals including BP must be monitored every five minutes during an episode of AD and also for 2 hours after the episode subsides, so as to ensure that it does not recur.[25]

The purpose of this small case series of eight patients is to highlight acute emergencies that can occur and to emphasize the importance of screening SCI patients for additional comorbidities and conditions which could be a contributory cause to various life-threatening complications. The classical signs of these conditions may be absent in SCI population, due to which clinicians may ignore such emergencies while assessing the patient. Failure to appreciate these contributory factors can delay the diagnosis of the emergency in the SCI population and can prove to be fatal. Therefore, it is essential to minimize and preferably eliminate these risks during the course of their hospitalization.

Another point to highlight is the importance of patient and their caregivers' education regarding the condition and possible risks and complications that can occur. This will help in quick action and they will seek emergency care at the earliest so that the source can be identified and the complication can be averted.

CONCLUSION

Spinal cord injury (SCI) patients are vulnerable to both hospital- and community-acquired infections. When SCI patients show clinical evidence of fever or a general feeling of being unwell, differential diagnoses should be considered while trying to locate the source of the infection. An appropriate and timely approach, diagnosis, and treatment must be carried out in order to reduce morbidity and avoid mortality.

Complications such as pneumonia, urosepsis, pressure injuries, appendicitis, acute abdominal emergencies, and heterotopic ossification can manifest with vague symptoms or trigger autonomic dysreflexia in SCI patients, leading to serious outcomes if not diagnosed or addressed on time. Therefore, clinical acumen and knowledge about the timely management of such patients are essential, as they can lead to improved outcomes and better quality of life.s.

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