

Challenges in the Management of Pressure Ulcers

Dr. Ritu Majumdar, MBBS, DNB (PMR), Resident Doctor

Dr. SY Kothari, MBBS, MS (Ortho), DNB (PMR), Consultant

Dr. Ajay Gupta, MBBS, DPMR, DNB (PMR), Senior Resident

Department of Physical Medicine and Rehabilitation, VMMC & Safdarjang Hospital, New Delhi – 110029

Abstract

Pressure ulcers primarily affect persons with impairment of sensation, spinal cord injured, diabetic and mobility impaired persons.

Twenty seven patients of spinal cord injury (SCI) with extensive pressure ulcers were studied. All of them had very low serum hemoglobin and serum protein level along with other complications of SCI hampering healing of pressure ulcers. This vicious cycle of paraplegia / quadriplegia → pressure ulcers → low serum hemoglobin and serum protein level → reduced healing capacity and more pressure ulcers, was broken with increase in serum hemoglobin and serum protein levels.

All these patients improved in general condition, serum hemoglobin, serum protein levels and were discharged ambulatory.

Key words: Pressure ulcers, spinal cord injury.

INTRODUCTION

Despite advances in medicine, surgery, nursing care and self-care education, pressure ulcers remain a major cause of morbidity and mortality, particularly for persons with impaired sensation and prolonged immobility.¹ Persons with spinal cord injury and associated co-morbidity are at an increased risk for the formation of pressure ulcers.² The incidence of pressure ulcers in this population ranges from 25% to as high as 66%.³

The National Pressure Ulcer Advisory Panel⁴ (NPUAP) of USA defines a pressure ulcer as “An area of unrelieved pressure over a defined area, usually over a bony prominence, resulting in ischemia, cell death, and tissue necrosis”.

Pressure ulcers are caused by the interaction of multiple and diverse, etio-pathological factors that can be classified as patho-mechanical or patho physiological.^{5,6}

Contributing factors to pressure ulcers

<i>Patho-mechanical (Extrinsic or primary)</i>	<i>Patho-physiological (Intrinsic or secondary)</i>
Compression	Fever
Maceration	Anemia
Immobility	Injection
Pressure	Ischemia
Friction	Hypoxemia
Shear	Malnutrition

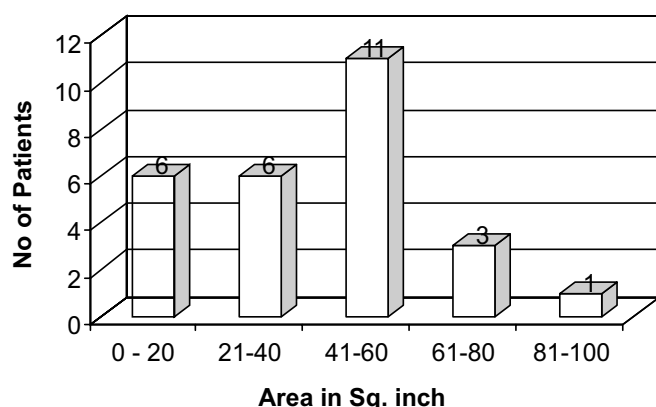
Address for Correspondence : Dr. Ritu Majumdar, A-50, Yojana Vihar Delhi-110092.

Spinal cord injury
Neurological disease
Decreased lean body mass
Increased metabolic demands

We admitted patients with extensive pressure ulcers suffering from SCI (Fig.1 and Graph 1), who were neglected by all other medical specialties like Plastic surgery, General Surgery, Orthopedics and Medicine, and treated them with successful outcome.



Graph 1 : Extent of Pressure Sores.

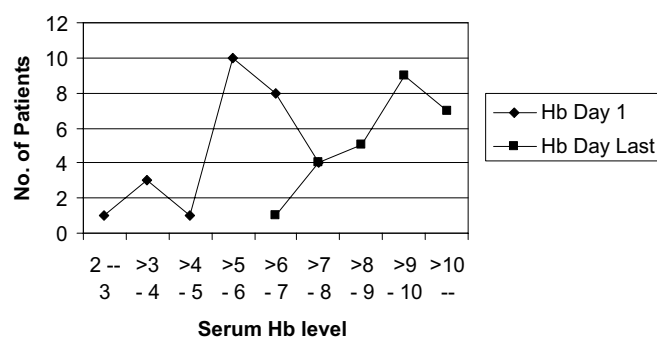


Material and Methods

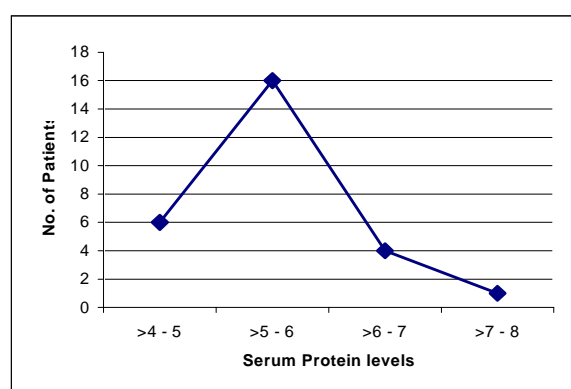
The retrospective study was done on twenty seven patients of spinal cord injury with extensive pressure ulcers who were admitted in spinal injury ward of the department of Rehabilitation Medicine, Safdarjang Hospital and VMMC, New Delhi, from the years 1998-2005. An explicit informed consent was taken from all the patients explaining possible untoward reaction and prognosis of the existing disease.

The patients' age ranged from 10 years to 40 years. They were of both sexes and belonged to both rural and urban backgrounds. The cause of SCI was both trauma and disease.

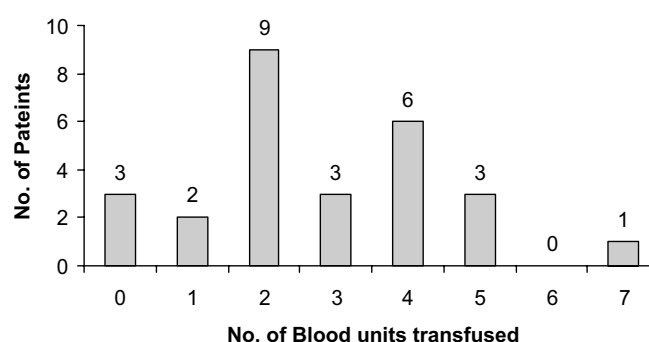
Graph 2 : Improvement in Hemoglobin (Hb) Levels.



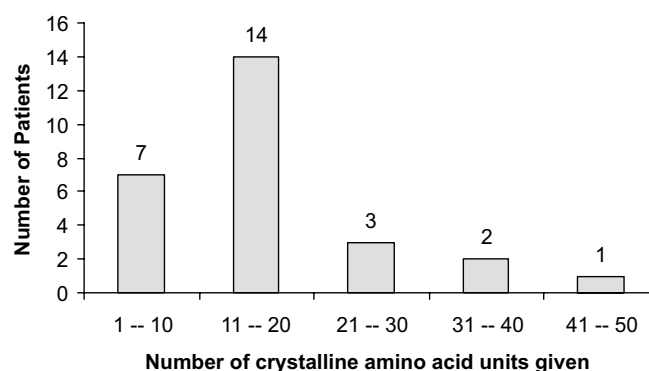
Graph 3 : Serum Protein Levels at Beginning



Graph 4 : Serum Protein Levels at Beginning



Graph 5 : Number of Crystalline Amino Acid Infusions Given.



Inclusion criteria

1. SCI patients with more than 2 weeks of injury
2. SCI patients with extensive pressure ulcers and with low serum hemoglobin levels.

Exclusion criteria

1. Patients with acute spinal cord injury (less than 2 weeks).
2. Patients having any psychiatric disorder.

The study included patients having both quadriplegia and paraplegia. ASIA impairment scale⁷ was used for classification of spinal cord injury.

The level of serum hemoglobin in all these patients varied from 2.3-8.0gm/dl (Graph 2) and the serum protein levels ranged from 4-7gm/dl. (Graph 3).

The staging of pressure ulcers was done as per NPUAP classification.⁴ Our study included patients with pressure ulcers having stage II to stage IV.

The pressure ulcers were measured initially and during healing, clinically. (Graph 1).

All other relevant investigations were also done. All the patients had other complications also, which too contributed to poor general condition and slow healing of ulcers. (Table 6)

Patients were given both general care by providing proper nutrition in the form of high protein diet, tablets of Iron and Folic acid, Vitamin C and 'B' complex.

Patients were given blood transfusion depending on the level of Serum hemoglobin and availability of donors. Intravenous nutritive infusion of pure crystalline amino acids containing essential and non essential amino acids in a balanced ratio (Hermin™, Alamin™) was also given to raise the serum protein levels, assuming that improvement in serum protein and serum hemoglobin level would break the vicious cycle as mentioned earlier and lead to faster healing of pressure ulcers.

The local care of pressure ulcer was given by providing daily Eusol dressing along with antibiotic ointment wherever required. Periodic shifting of patients on bed and wheel-chair was done 2 hourly. Water mattress was discouraged as it obstructs in proper turning of patients, in effect leading to slow healing of pressure ulcers.

Observation and Results

Age, sex, background of patients, cause of SCI, level of injury and co-morbid conditions are shown in Tables 1-6.

Table 1 : Age Distribution

0-10	11-20	21-30	31-40
1	4	11	11*

*1 died (36 years)

Table 2 : Sex Distribution

Male	Female
22*	5

*1 died

Table 3 : Background

Urban	Rural
10	17*

*1 died

Table 4 : Cause

Trauma	Disease (TB)
25*	2

*1 died

Table 5: Level of injury

	C ₁₋₇	D ₁₋₆	D ₇₋₁₂	L _{1->}
Vertebral	9*	6	10	2
Neurological	9*	6	7	5
Classification	Quadri	High para	Low para	

*1 died (C₇)

Table 6: Co-morbid conditions

Head injury	1
Major fracture (Extremities)	1 (Tibia)
Abdominal injury	1 (Colostomy done)
Depression	1
Hematuria	1
Urinary tract infection	22
Septicemia, ARF, DIC	1 (died)

Maximum number of patients were in the age groups 21-30 yrs (Table 1).. The male to female ratio being 4.2:1 (Table 2), ratio of rural to urban background was found to be 1.7:1 (Table 3).

Trauma was the most common cause of SCI (Table 4). Ratio between trauma and disease was 12.5:1, whereas the paraplegia to quadriplegia ratio was 2:1 (Table 5).

The hospital stay of patients varied from 5 wks to 20 wks with most patients staying for 5 wks.

The area of pressure ulcers in the given study in sq.cms. is shown in Graph 1, with most patients having pressure ulcers with an area of 261-390 sq. cms. Maximum no. of patients had serum hemoglobin level in the range of 5-6gm/dl (from 2.7 to 7.4 gms/dl.) (Graph 2) and Serum Protein level in the range of 5-6 gm/dl (Graph 3) at the time of admission. No. of units of whole blood transfusion to patients varied from 0-7 units with most of the patients receiving 2 units of whole blood (Graph 4).

The level of serum hemoglobin was shown to improve in all these patients with average hemoglobin rising up to 9 gm/dl. at the time of discharge. (Graph 2). Intravenous infusion of crystalline amino acids received by the patients is shown in Graph 5, with most of the patients receiving 11-20 units (Unit of 200 ml).

Maximum number of patients had urinary tract infection as the most common associated complication at the time of admission which contributed to poor general condition of these patients.

At the end of the study we found that most patients had improved serum hemoglobin (Graph 2) and serum protein level along with improvement in their general condition.

All the pressure ulcers were healing in respect to their size, number and depth. The pressure ulcers were clean, granulating and contracting in size to be fit for plastic surgery, except in case of one patient who died as a result of septicemia.

All the patients were ambulatory with maximum number of patients on wheel-chair and one patient being able to walk with bilateral Ankle Foot Orthoses (AFO) and elbow crutches. Two patients left against medical advice, when their hemoglobin levels had improved but final ambulatory status was not attained.

Discussion

Maximum number of patients in the given study were in age group 21-40 years with a mean age of 30.5 years. This correlates with studies by Marc D Basson, Richard E Burney et.al. who also had patients with SCI having decubitus ulcers with mean age of 31 years.²

Male to female ratio of 4.2:1 is in correlation with studies by Stover S L et. al. who found a male to female ratio of 4:1 in their study.⁸

Trauma being the common cause of SCI in our study is similar to a study by Gary M. Yarkony⁹ who mention trauma to be the most common cause of SCI.

All the patients in our study were anemic as it was our selection criteria and was a natural result of extensive pressures ulcer (cause and effect). According to a study by Hirsch GH et al¹⁰, on patients with SCI, anemia is fairly common in chronic spinal cord injury persons with an incidence ranging from 30% to 56%.

The type of anemia in our patients was normocytic normochromic and microcytic hypochromic. Anemia is an intrinsic risk factor for the development of pressure ulcers and persons who have a serum hemoglobin level below 10gm/dl have difficulty in healing of pressure ulcers¹¹. Therefore we took special care in improving serum hemoglobin levels by multiple blood transfusions as and when available together with multiple transfusions of amino acids and oral iron and vitamin supplements.

Most of the patients in our study had urinary tract infection (UTI) as a co -morbid condition. According to a study by Hirsch G H et al, it is reported, that the increased risk and frequent recurrence of UTI is the most common cause of anemia in persons with SCI.¹¹

One patient died in our study due to septicemia. Allman R M⁵ reported that infection is the major complication associated with pressure ulcers. Systemic sepsis and wound related bacteremia are life threatening complications and can increase the risk of mortality to 55%.

At Discharge

1. Most of the patients had serum hemoglobin level of 9gm%.
2. The ulcers were clean, granulating, healing and contracting in size, and fit for plastic surgery.
3. The general condition of all the patients was stable.

4. All the patients were ambulatory. (Except one that died for other reasons)

Conclusion

It can be concluded that pressure ulcers in spinal cord injured persons can be successfully treated by improving the serum hemoglobin and serum protein levels. Proper attention if required for associated complication and care of ulcers locally, breaking the vicious cycle of low serum hemoglobin/ serum protein and pressure ulceration.

References

1. Abrussezze RS. Early assessment and prevention of pressure ulcers. In: Lee BY, ed. Chronic ulcers of the skin. New York: McGraw-Hill; 1985: 1-9.
2. Basson MD, Burney RE: Defective wound healing in patients with paraplegia and quadriplegia. Surg Gynecol Obstet 1982; 155 : 9-12.
3. Fuhrer MJ, Garber SL, Rintala DH, Clearman R, Hart KA. et al. Pressure ulcers in community resident persons with spinal cord injury: prevalence and risk factors. Arch Phys Med Rehabil 1993; 74(11):1172-7.
4. National Pressure Ulcer Advisory Panel. Pressure ulcers, Incidence, economics, risk assessment, Consensus Development Conference Statements West Dundee, IL S-N Publications; 1989.
5. Allman RM. Pressure ulcers among the elderly. N Engl J Med 1989; 320: 850-3.
6. Anthony JP, Huntsman WT, Mathes SJ. Changing trends in the management of pelvic pressure ulcers: a 12-year review. Decubitus. 1992;5(3):44-7, 50-1.
7. American Spinal Injury Association. International standards for neurological classification of spinal cord injury. Chicago: American Spinal Injury Association: 1992.
8. Stover SL, Fine PR, eds. Spinal cord injury. The facts and figures. Birmingham: University of Alabama at Birmingham; 1986.
9. Gary M. Yarkony, David Chen. Rehabilitation of patients with spinal cord injuries. In: Randall L. Braddom, ed. Text book of physical medicine and rehabilitation. Philadelphia: WB Saunders; 1996 : 1149-79.
10. Hirsch GH, Menard MR, Anton HA: Anemia after traumatic spinal cord injury. Arch Phys Med Rehabil 1991; 72: 195-201.
11. Perkash A, Brown M. Anemia in patients with traumatic spinal cord injury. J Am Paraplegia Soc 1986; 9 : 10-15.