

Review Article

Nutritional Issues in Neurointensive care

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ABSTRACT

Acute and chronic neurological diseases are associated with a high incidence of malnutrition due to the stress response created by them. Early recognition of nutritional deficiency by careful assessment of nutritional status by various means is essential. These patients may have decreased intake, disease related changes in resting energy expenditure, and effect of drug therapy on food intake, all of which predisposes for undernutrition. Enteral route of nutrition is the preferred route for supplementation of nutrition in these patients however, parenteral route also can be utilised if the enteral route is contraindicated. A close monitoring of the patient on nutrition supplementation is required to prevent complications of nutrition. A reassessment of nutritional status is required in the chronic phase of these neurological diseases as diseases related reduction in resting energy expenditure and requirements may lead to overnutrition and obesity.

Key words: Neurointensive care unit, nutrition, spinal cord injury, traumatic brain injury

INTRODUCTION

Nutrition is considered to be a major factor in the control of several major clinical diseases affecting the outcome and a determinant of the associated mortality and morbidity. Nutritional deficiency has been described in hospitalized population with varying degrees and is often reversible if recognized early.^[1,2] Patients with neurological diseases are at high risk of development of malnutrition due to low intake and high metabolic rate in these diseases. These include patients with polytrauma and associated head injury, maxillofacial trauma, postoperative neurosurgical patients, neuro-oncology patients, patients operated for spine lesions and so on.^[3-11] The nutritional aspects need special attention in this group of population on an individual basis. An association has been found between nutrition and neurological diseases with some of the neurological diseases found more commonly in particular

diet group and few of the neurological diseases being less common in population with intake of particular nutrient. However, reduced energy expenditure and immobility due to some neurological diseases can also predispose them to obesity and other.

Factors leading to malnutrition

Numerous factors are responsible for causing malnutrition in such subset of population which can be summarized as:

Decreased intake

The various factors implicated in reduction in intake of food in patients with neurological diseases are:

- Depression
- Impaired cognitive functions
- Dementia
- Apraxia
- Self-imposed dietary restrictions.

Dysphagia

It is defined as difficulty or discomfort during swallowing and can be classified as oropharyngeal or esophageal and organic or functional. The various neurological diseases are associated with dysphagia with varying degrees e.g. 30% in stroke patients, 40% in patients with muscular dystrophies and up to 84% in patients with Alzheimer's disease.^[12]

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Nausea and vomiting

The presence of nausea and vomiting may be due to raised intracranial pressure and which may impair the intake. Any third ventricular pathology may cause severe intractable nausea and vomiting. The use of certain drugs in chronic neurological diseases may also cause nausea and vomiting e.g. levodopa, bromocriptine, anticholinergics, phenytoin, phenobarbitone, etc Newer long acting anti-emetic drugs are available which can tide over these crises.^[13]

Delayed gastric emptying

Delayed gastric emptying may cause decreased appetite, early satiety, reflux, abdominal bloating, nausea, and vomiting, all of which can lead to decreased intake and malnutrition. This delayed gastric emptying may be due to raised intracranial pressure, autonomic neuropathy, and myopathy, effect of various drugs or gastro paresis as seen in Parkinson's disease.^[14]

Constipation

It can be manifestation of various neurologic diseases and can lead to decreased dietary intake. It may be due to slow intestinal motility, weakness of pelvic muscles, due to various drugs and decreased intake of fluid and fibre.

Alteration in resting energy expenditure

The resting energy expenditure may be altered in various neurologic diseases leading to hypo or hypermetabolism and which can lead to malnutrition if the alteration in resting energy expenditure (REE) is increased. Obesity can also result if the REE is decreased as in seen in paralysis, malnutrition, and drugs like sedatives, baclofen etc The diseases with an increased REE thus causing malnutrition are Huntington's chorea, myoclonus, fasciculations, spasticity, refeeding, and infections.^[15-18]

Nutritional assessment

The assessment of nutritional status is very important before planning for nutritional supplementation. It is a comprehensive approach done by a registered dietician for defining nutrition status using medical, social, nutritional and medication histories, physical examination, anthropometric examination, and laboratory data. The malnutrition can be protein malnutrition, protein-calorie malnutrition or combination of both. The nutrition assessment can be done by following tools:

Nitrogen balance

It is an index of protein status, that is, the amount of nitrogen required to maintain the nitrogen equilibrium. It is used to measure the severity of protein catabolism and adequacy of the nutrition regimen. A negative nitrogen balance suggests inadequate intake of proteins

and/or calorie and increased catabolism of proteins. The goal of nutritional supplementation should be to maintain a positive nitrogen balance. About 90% of the nitrogen is excreted in urine, so nitrogen balance is estimate from urinary nitrogen excretion over 24 hours. The main usage of nitrogen balance is in nutritional assessment as well as in establishing the adequacy of the nutrition regimen.

Physical evaluation

The physical signs may be non-specific and mild and sometimes only suggest a deficiency of a particular nutrient. They should be evaluated along with the anthropometric examinations, dietary history and laboratory parameters to effectively assess the degree of malnutrition.^[2,19]

Laboratory parameters

The few laboratory findings suggestive of malnutrition are: Albumin and prealbumin: Prealbumin is more sensitive indicator of the deficient nutritional status as it has a lower half-life and is the first variable to register an increased demand for protein synthesis during stress.

Transferrin: It is also considered to be a sensitive indicator of visceral protein status due to its shorter half-life.

Types of nutritional support

There are two main types of nutritional supports available which are enteral nutrition and parenteral nutrition. The type of support depends upon the type of neurologic defect and its effect on the resting and total energy expenditure and on the digestive tract of the body.

Enteral nutrition

The gastrointestinal tract is considered to be the first choice for nutrition supplementation as a functioning gastrointestinal tract decreases the occurrence of bacterial translocation, improves gut mucosal integrity and enzymatic activity.^[20] Most of the feeding in the neurocritical intensive care unit (NICU) is done by tube feeding.^[21,22] As a variety of enteral tube feeding formulas are available, so it is mandatory to take into account the indications, contraindications and the clinical settings of the patient before starting on enteral feeding.^[23] The various contraindications to enteral nutritional support include:

- Complete mechanical bowel obstruction
- High output enterocutaneous fistula (more than 500 ml enteric effluent)
- Intolerance to enteral feeds as evidenced by high aspirates, abdominal distention or ileus
- Severe acute pancreatitis
- Severe gastrointestinal hemorrhage
- Severe enterocolitis.

The disadvantages of enteral nutrition include inadequate calorie intake, intolerance, and *Clostridium difficile* enterocolitis induced diarrhoea. It has also been held that the incidence of ventilator associated pneumonia is increased due to increased gastric pH and colonisation by bacteria.^[24] Two forms of enteral feeds are usually supplied:

Polymeric preparations

It is composed of intact proteins, complex fats, and carbohydrates with added minerals, vitamins, and trace elements. It is lactose free to prevent any lactose intolerance. Fibre is generally added to maintain the structural integrity of enterocytes.

Elemental preparations

It is composed of nutrients in a readily absorbable form e.g. carbohydrates in the form of mono or disaccharides, fats in the form of medium chain fatty acids and proteins in the form of peptides or amino acids. This type of feed may be helpful in malabsorption states.

Parenteral nutrition

This route is employed in patients with failure of enteral nutrition, when the enteral feeding is not anticipated to be started within 5-7 days and in supplementation of enteral feeds as it is shown that enteral feeds alone may lead to underfeeding.^[25,26] It is administered by means of central venous line but can also be administered through peripheral intravenous line depending on the osmolality of the solution. The complications associated are mainly due to catheter insertion and infection so it is mandatory that a strict asepsis should be maintained while inserting central venous line, a dedicated port is used for infusion and minimal interruptions of the infusion are done.

Parenteral nutrition is given as sterile emulsion containing proteins in the form of soluble mixture of essential and non-essential amino acids, fat as intralipid formed of soya with chylomicron sized particles and the carbohydrates in the form glucose. The electrolytes and minerals are added to the emulsion depending on the disease and condition of the patient. As the incidence of metabolic complications associated with parenteral nutrition is high, regular monitoring is required with daily monitoring of electrolytes, intake and output, complete blood count and prealbumin monitored once a week. Stress needs to be given for micronutrient supplementation both during enteral and parenteral nutrition practices.^[27]

The complications associated with parenteral nutrition are mainly catheter related and may be due to insertion e.g. hemorrhage, pneumothorax, arterial puncture, or due to infection. The major metabolic complications

involved are hyper or hypoglycaemia, hypo/hyperkalemia, hypo/hyponatremia, hypo/hyperphosphatemia, hypo/hypermagnesemia, hyperchloremic metabolic acidosis, etc., In addition, the patients may have deficiency of vitamins, minerals and fatty acids and may develop liver function abnormalities.

Special considerations in neurologic diseases

Traumatic brain injury

Traumatic brain injury cause acute metabolic response mediated by release of cytokines and other inflammatory mediators and stress related hormones. The consequence is a hypermetabolic state with a rise in resting energy expenditure and hypercatabolism. Hypermetabolism with insulin resistance causes hyperglycemia, impaired immunity, endothelial integrity and an increased risk of infection. These clinical manifestations are exaggerated in patients with obesity, endocrine diseases and diabetes.^[28-31] The energy expenditure in acute brain trauma depends upon the neurologic status, intracranial pressure, medical or surgical therapy and the presence of infection or renal failure. The resting energy expenditure is usually underestimated by conventional methods. The loss of proteins is also accelerated in traumatic brain injury and is further aggravated by use of steroids leading to a negative nitrogen balance.^[32]

There is reduction in the lean body mass of the patient due to hypercatabolism with 10-15% lost in one week and over 30% lost within 2-3 weeks without nutritional intervention.^[33] Hypoalbuminemia is also common due to inflammation induced redistribution of albumin and by dilution due to crystalloid administration.^[34] Geriatric patients pose nutritional challenges in these clinical circumstances as these patients are invariably on polypharmacy and exhibit wide range of clinical symptomatology during traumatic brain injury.^[35-38]

Spinal cord injury

The physiological changes due to acute spinal cord injury also mimics those of traumatic brain injury with activation of stress response with release of inflammatory mediators in proportion with the severity of injury. The patients with spinal cord injury are usually not hypermetabolic and have 90-95% of the predicted resting energy expenditure which may be attributed to decreased lean body mass, decreased muscular activity due to paralysis and to a lower sympathetic system activity.^[39] However, in a chronic spinal cord injury, the resting metabolic rate may be lower than the normal subjects by 14-27% which may overestimate the caloric requirements.^[40,41] Obesity may result due to overnutrition and can lead to glucose intolerance, insulin resistance, and hyperlipidemia. Surgical intervention

in these patients is highly challenging and may involve designing of numerous therapeutic strategies during perioperative period to attenuate stress response related complications.^[42,43]

Chronic spinal cord and traumatic brain injury

The transition from acute to chronic phase causes changes in the resting energy expenditure which should be reassessed accordingly. Patients with spasticity and decerebrate or decorticate posturing causes an increased caloric needs. Inability to consume required nutrients due to physical and cognitive impairment in chronic phase, often lead to malnutrition in these patients which impairs the rehabilitation. Hypoalbuminemia and anaemia in these chronic patients are a common finding and often lead to increased hospital length of stay and difficulties in rehabilitation. The clinical scenario becomes highly complicated if such patients are having viral and fungal infections.^[44,45] Optimisation of the nutritional status results in decreased length of stay and improves functional output.

In high spinal cord injuries, the patients in chronic phase have lower caloric needs and a calorie intake approximating 23 kcal/kg/day may be sufficient in quadriplegic patient.^[46] These lowered metabolic demands may be due to replacement of muscles with fat in chronic phase. Enteral feeding is usually well tolerated and should be continued till the patients' starts taking orally.

Chronic neurologic diseases

These may include cerebrovascular disease, Alzheimer's disease, Parkinson's disease, Amyotrophic lateral sclerosis, multiple sclerosis, neuro-endocrine pathologies, Huntington's disease, and dementia. The challenging aspects are encountered when such patients are either taken for surgical procedures or require a prolonged intensive care stay.^[47,49] These patients also suffer from poor intake, malnutrition, dysphagia, loss of mobility, decreased cognitive skills, and bowel and bladder disturbances. In Parkinson's disease, the diet has to be modified to have low protein content to enhance the effect of levodopa with a carbohydrate to protein ratio of 5:1.^[50] Osteoporosis is often found in these chronic patients due to prolonged immobilization, lack of weight bearing, and decreased nutrient intake and absorption. The supplementation of calcium and vitamin D should always be included in the diet plan of these patients.^[51] Patients with multiple sclerosis may develop obesity due to overnutrition, immobility and steroid intake. It requires a dedicated team work and cohesive motivational unit to manage such patients in neuro-intensive care.^[52] The modern day nutritional

practices do favor the principles of logical empiricism in such subset of population.^[53]

CONCLUSION

Patients with neurologic diseases are at increased risk of development of malnutrition owing to decreased intake, disease related changes in resting energy expenditure and effect of drug therapy. A structured nutritional supplementation is mandatory in these patients that should be changed according to the changes in energy expenditure and metabolism. Malnutrition in these patients can increase the morbidity and reduce the positive rehabilitative outcome. Both enteral and parenteral route can be employed; however, enteral route is preferred for obvious reasons and a balanced diet containing the trace elements, vitamins and minerals is preferred.

REFERENCES

1. Bajwa SS, Kulshrestha A. Critical nutritional aspects in intensive care patients. *J Med Nutr Nutraceutical* 2012;1:9-16.
2. Frankmann CB. Medical nutrition therapy for neoplastic disease. In: Mahan K, Escott-Stump S, editors. *Krause's Food, Nutrition and Diet Therapy*. 10th ed. Philadelphia: WB Saunders; 2000.
3. Bajwa SJ, Kulshrestha A. Challenges and critical aspects of neuro-oncology in ICU Patients. *J Spine Neurosurg* 2013;S1.
4. Kulshrestha A, Bajwa SJ. Anaesthetic considerations in intracranial neurosurgical patients. *J Spine Neurosurg* 2013;S1.
5. Bajwa SS, Kaur J, Bajwa SK, Kaur G, Singh A, Parmar SS, Kapoor V, *et al.* Designing, managing and improving the operative and intensive care in polytrauma. *J Emerg Trauma Shock* 2011;4:494-500.
6. Bajwa SS, Bajwa SK, Kaur J. Care of terminally ill cancer patients: An intensivist's dilemma. *Indian J Palliat Care* 2010;16:83-9.
7. Bajwa SS, Bajwa SK. Anesthesia and Intensive care implications for pituitary surgery: Recent trends and advancements. *Indian J Endocrinol Metab* 2011;15:S224-32.
8. Bajwa SJ, Bajwa SK, Bindra GS. The anesthetic, critical care and surgical challenges in the management of craniopharyngioma. *Indian J Endocrinol Metab* 2011;15:123-6.
9. Bajwa SJ, Kaur J, Singh A, Kapoor V, Bindra GS, Ghai GS. Clinical and critical care concerns of cranio-facial trauma: A retrospective study in a tertiary care institute. *Natl J Maxillofac Surg* 2012;3:133-8.
10. Bajwa SJ, Jindal R. Epilepsy and nonepilepsy surgery: Recent advancements in anesthesia management. *Anesth Essays Res* 2013;7:10-7.
11. Bajwa SJ, Kulshrestha A. Spine surgeries: Challenging aspects and implications for anaesthesia. *J Spine Neurosurg* 2013;2:3.
12. Clave P, Terre R, de Kraa M, Serra M. Approaching oropharyngeal dysphagia. *Rev Esp Enferm Dig* 2004;96:119-31.
13. Bajwa SS, Bajwa SK, Kaur J, Sharma V, Singh A, Singh A, *et al.* Palonosetron: A novel approach to control postoperative nausea and vomiting in day care surgery. *Saudi J Anaesth* 2011;5:19-24.
14. Heetun ZS, Quigley EM. Gastroparesis and Parkinson's disease: A systematic review. *Parkinsonism Relat Disord* 2012;18:433-40.
15. Bajwa SS, Kwatra IS. Nutritional needs and dietary modifications in patients on dialysis and chronic kidney disease. *J Med Nutr Nutraceutical* 2013;2:46-51.

16. Gaba AM, Zhang K, Marder K, Moskowitz CB, Werner P, Boozer CN. Energy balance in early-stage Huntington disease. *Am J Clin Nutr* 2005;81:1335-41.
17. Cuching ML, Traviss KA, Calne SM. Parkinson's disease: Implications for nutritional care. *Can J Diet Pract Res* 2002;63:81-7.
18. Sherman MS, Pillai A, Jackson A, Heiman-Patterson T. Standard equations are not accurate in assessing resting energy expenditure in patients with amyotrophic lateral sclerosis. *JPEN J Parenter Enteral Nutr* 2004;28:442-6.
19. De Jonghe B, Appere-De-Vechi C, Fournier M, Tran B, Merrer J, Melchior JC, *et al.* A prospective survey of nutritional support practice in intensive care unit patients: What is prescribed? What is delivered? *Crit Care Med* 2001;29:8-12.
20. Hillhouse JH, Neiger R. Pregnancy and lactation. In: Gottschlich MM, Fuhrman MP, Hammond KA, Holcombe BJ, Seidner DL, eds. *The Science and Practice of Nutrition Support: A Case-Based Core Curriculum*. Dubuque, IA, Kendall/Hunt Publishing Company, 2001, p. 302-19.
21. Bajwa SJ, Gupta S. Controversies, principles and essentials of enteral and parenteral nutrition in critically ill-patients. *J Med Nutr Nutraceutical* 2013;2:77-83.
22. Bajwa SS. Nutritional facts in critically ill patients: The past, present and the future. *J Med Nutr Nutraceut* 2014;3:6-10.
23. Merritt RJ, editor. *The A.S.P.E.N. Nutrition Support Practice Manual*. 2nd ed. Silver Spring, MD: A.S.P.E.N.; 2005.
24. Drakulovic MB, Torres A, Bauer TT, Nicolas JM, Nogue S, Ferrer M. Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: A randomised trial. *Lancet* 1999;354:1851-8.
25. Hayland DK, Schroter-Noppe D, Drover JW, Jain M, Keefe L, Dhaliwal R, *et al.* Nutritional support in the critical care setting: Current practices in Canadian ICUs-opportunities for improvement? *JPEN J Parenter Enteral Nutr* 2003;27:74-83.
26. Verhage AH, van Vliet AC. Clinical practice of nutritional support in Dutch intensive care units: A survey. *Eur J Intern Med* 2002;13:496-9.
27. Bajwa SJ. The underestimated nutritional aspects of micronutrients supplementation in intensive care. *J Med Nutr Nutraceutical* 2013;2:114-6.
28. Bajwa SJ. Intensive care management of critically sick diabetic patients. *Indian J Endocrinol Metab* 2011;15:349-50.
29. Bajwa SJ, Sehgal V, Bajwa SK. Clinical and critical care concerns in severely ill obese patient. *Indian J Endocrinol Metab* 2012;16:740-8.
30. Bajwa SJ, Jindal R. Endocrine emergencies in critically ill patients: Challenges in diagnosis and management. *Indian J Endocrinol Metab* 2012;16:722-7.
31. Bajwa SJ, Kalra S. Diabeto-anaesthesia: A subspecialty needing endocrine introspection. *Indian J Anaesth* 2012;56:513-7.
32. Frankenfield D. Energy expenditure and protein requirements after traumatic injury. *Nutr Clin Pract* 2006;21:430-7.
33. Rosbalt MB, Hattton J. Brain and spinal cord injuries. In: Rolandelli, editor. *Clinical Nutrition: Enteral and Tube Feeding*. 4th ed. Philadelphia: Elsevier Saunders; 2005.
34. Thomson MA, Carver AD, Sloan RL. Nutritional status of traumatic and anoxic brain injured patients on admission to rehabilitation. *Proc Nutr Soc* 2001;60:A223.
35. Sehgal V, Bajwa SJ, Sehgal R, Bajaj A, Khaira U, Kresse V. Polypharmacy and potentially inappropriate medication use as the precipitating factor in readmissions to the hospital. *J Family Med Prim Care* 2013;2:194-9.
36. Kalra S, Bajwa SS, Baruah M, Sehgal V. Hypoglycaemia in anesthesiology practice: Diagnostic, preventive, and management strategies. *Saudi J Anaesth* 2013;7:447-52.
37. Sehgal V, Bajwa SS, Khaira U, Sehgal R, Bajaj A. Challenging aspects of and solutions to diagnosis, prevention, and management of hypoglycemia in critically ill geriatric patients. *J Sci Soc* 2013;40:128-34.
38. Bajwa SS, Panda A. Alternative medicine and anesthesia: Implications and considerations in daily practice. *Ayu* 2012;33:475-80.
39. Barco KT, Smith RA, Peerless JR, Plaisier BR, Chima CS. Energy expenditure assessment and validation after acute spinal cord injury. *Nutr Clin Pract* 2002;17:309-13.
40. Buchholz AC, McGillivray CF, Pencharz PB. Differences in resting metabolic rate between paraplegic and able-bodied subjects are explained by differences in body composition. *Am J Clin Nutr* 2003;77:371-8.
41. Buchholz AC, Pencharz PB. Energy expenditure in chronic spinal cord injury. *Curr Opin Clin Nutr Metab Care* 2004;7:635-9.
42. Bajwa SS, Kaur J, Singh A, Parmar S, Singh G, Kulshrestha A, *et al.* Attenuation of pressor response and dose sparing of opioids and anaesthetics with pre-operative dexmedetomidine. *Indian J Anaesth* 2012;56:123-8.
43. Bajwa SJ, Kulshrestha A. Dexmedetomidine: An adjuvant making large inroads into clinical practice. *Ann Med Health Sci Res* 2013;3:475-83.
44. Bajwa SJ, Kulshrestha A. The potential anesthetic threats, challenges and intensive care considerations in patients with HIV infection. *J Pharm Bioallied Sci* 2013;5:10-6.
45. Bajwa S, Kulshrestha A. Fungal infections in intensive care unit: Challenges in diagnosis and management. *Ann Med Health Sci Res* 2013;3:238-44.
46. Nutritional support after spinal cord injury. *Neurosurgery* 2002;50 Suppl 3:S81-4.
47. Kulshrestha A, Bajwa SJ. Nutritional and eating disorders: Clinical impact and considerations during anesthesia procedures. *J Med Nutr Nutraceut* 2012;1:77-82.
48. Bajwa SJ, Sethi E, Kaur R. Nutritional risk factors in endocrine diseases. *J Med Nutr Nutraceut* 2013;2:86-90.
49. Bhaskar SB, Bajwa SS. Pharmacogenomics and anaesthesia: Mysteries, correlations and facts. *Indian J Anaesth* 2013;57:336-7.
50. Bajwa SK, Bajwa SJ, Kaur J, Singh A. Anesthesia implications in emergency oncologic surgery in a case of untreated Parkinsonism. *Saudi J Anaesth* 2011;5:317-9.
51. Weinstock-Guttman B, Gallagher E, Baier M, Green L, Feitcher J, Patrick K, *et al.* Risk of bone loss in men with multiple sclerosis. *Mult Scler* 2004;10:170-5.
52. Bajwa S, Virdi SS, Bajwa SK, Ghai GK, Singh K, Rana CS, *et al.* In depth analysis of motivational factors at work in the health industry. *Ind Psychiatry J* 2010;19:20-9.
53. Bajwa SS, Kalra S. Logical empiricism in anesthesia: A step forward in modern day clinical practice. *J Anaesthesiol Clin Pharmacol* 2013;29:160-1.

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