TO ASSESS CARDIOVASCULAR RISK IN CASES OF SPINAL CORD INJURY BY SCREENING IMPAIRED GLUCOSE TOLERANCE AND DYSLIPIDEMIA – CROSS SECTIONAL STUDY

Dissertation Submitted in Partial Fulfilment of the Requirements

for the award of the degree of

MD (Physical Medicine and Rehabilitation)

University Examinations, May – 2019

Registration No. 201629002

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMIL NADU

2016 – 2019

DECLARATION

I, **DR. KAMAKSHI RM**, declare that this dissertation entitled "**To Assess Cardiovascular Risk in Cases Of Spinal Cord Injury by Screening Impaired Glucose Tolerance and Dyslipidemia – Cross Sectional Study**", is the original work done by me, **DR. KAMAKSHI RM**, registration number **201629002** in the Government Institute of Rehabilitation Medicine, **Madras Medical College**, Chennai, under the direct guidance and supervision of **Prof. Dr. C. RAMESH**, Government Institute of Rehabilitation Medicine, **Madras Medical College**, Chennai as Guide and is submitted to the **Tamil Nadu DR. M.G.R Medical University**, Chennai, in partial fulfilment of the regulations for the degree of **MD (Physical Medicine and Rehabilitation)**

DR. KAMAKSHI RM

(Reg.No.201629002)

CERTIFICATE

This is to certify that this dissertation entitled "**To Assess Cardiovascular Risk in Cases Of Spinal Cord Injury by Screening Impaired Glucose Tolerance and Dyslipidemia – Cross Sectional Study**", is the bona fide work carried out by **DR. KAMAKSHI RM**, registration number 201629002, in the Government Institute of Rehabilitation Medicine, **Madras Medical College**, Chennai, submitted in partial fulfilment of the board regulations for the award of the degree of **MD (Physical Medicine and Rehabilitation)**

DEAN

Madras Medical College

Chennai.

DIRECTOR & HOD

Government Institute of Rehabilitation Medicine

KK Nagar

Chennai.

CERTIFICATE

This is to certify that this dissertation entitled "**To Assess Cardiovascular Risk in Cases Of Spinal Cord Injury by Screening Impaired Glucose Tolerance and Dyslipidemia – Cross Sectional Study**", is the original work done by **DR. KAMAKSHI RM**, registration number 201629002, in the Government Institute of Rehabilitation Medicine, **Madras Medical College**, Chennai, from March 2017 to September 2018 under my guidance and submitted in partial fulfilment of the regulations for the degree of **MD (Physical Medicine and Rehabilitation).**

Prof. DR. C. Ramesh, DA, D.Phys. Med.,
MD(PMR)., DNB(PMR).,
Guide
Director & HOD
Government Institute of Rehabilitation Medicine
Madras Medical College, Chennai

CERTIFICATE

This is to certify that this dissertation entitled "**To Assess Cardiovascular Risk in Cases Of Spinal Cord Injury by Screening Impaired Glucose Tolerance and Dyslipidemia – Cross Sectional Study**", is the original work done by **DR. KAMAKSHI RM**, registration number 201629002, in the Government Institute of Rehabilitation Medicine, **Madras Medical College**, Chennai, from March 2017 to September 2018 under my guidance and submitted in partial fulfilment of the regulations for the degree of **MD (Physical Medicine and Rehabilitation).**

Prof. DR. T. Jayakumar, D.Ortho., DPMR.,
MD(PMR)., DNB(PMR).,
Co-Guide
Professor
Government Institute of Rehabilitation Medicine
Madras Medical College, Chennai

ACKNOWLEDGEMENT

I would like to express my special thanks to my guide **Prof. DR. C. Ramesh** and co-guide, **Prof. DR. T. Jayakumar**, who gave the opportunity and provided timely guidance and support during the entire tenure of my research. Their vast knowledge helped me in getting different perspectives on the dissertation refine my analysis approach. For this insight no amount of gratitude and thanks would from my end not suffice.

I also like to thank **Prof. DR. R. Jayanthi**, Dean, Madras Medical College and **Prof. DR. Sudha Sesaiyan**, Vice Principal, **Madras Medical College** for their support.

I owe my thanks to **Prof. DR. K. Ramadevi**, Director of Biochemistry Department who allowed me to utilize the lab services for my dissertation work.

I also extend my thanks to **DR. A. Rajakumar**, **DR. K. Premalatha**, **DR. K. Uma** and **DR. B. Jayanthi** for their help and constant support.

I am grateful to my seniors and colleagues for their steady support.

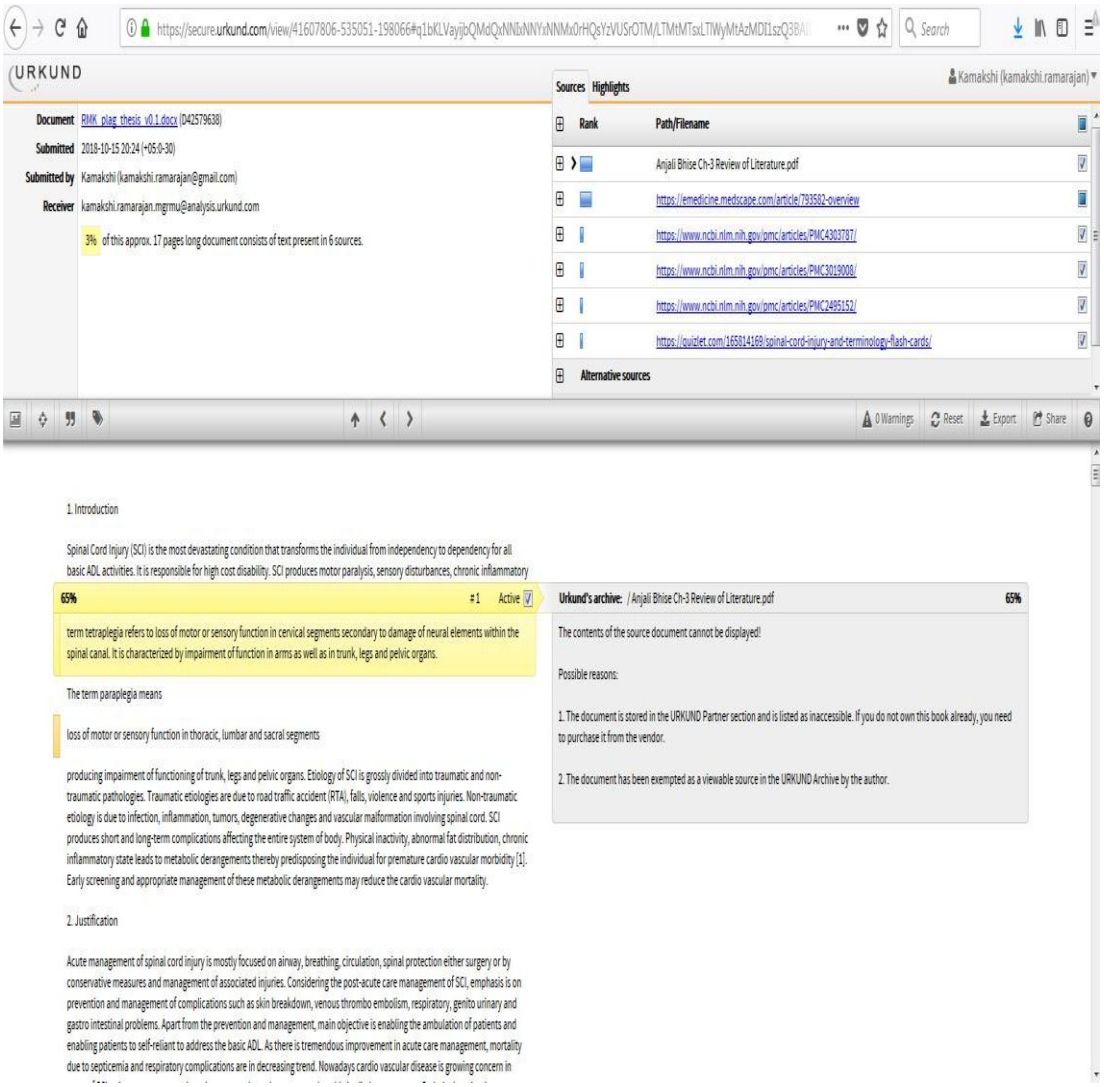
I am grateful to department of physiotherapy, Government Institute of Rehabilitation Medicine, Chennai for their support.

I am indebted to all the patients who willingly agreed to be part my research and readily participated in the rehabilitation program.

Last but not the least, I would like to thank my family for their support during this academic period

DR. KAMAKSHI. RM

PLAGARISM CERTIFICATE



URKUND

Document RMK_plag_thesis_v0.1.docx (D42579638)
Submitted 2018-10-15 20:24 (+05:0-30)
Submitted by Kamakshi (kamakshi.ramarajan@gmail.com)
Receiver kamakshi.ramarajan.mgrmu@analysis.urkund.com

3% of this approx. 17 pages long document consists of text present in 6 sources.

Sources Highlights — Kamakshi (kamakshi.ramarajan)

Rank	Path/Filename
	Anjali Bhise Ch-3 Review of Literature.pdf
	https://emedicine.medscape.com/article/793582-overview
	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4303787/
	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3019008/
	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2495152/
	https://quizlet.com/165814169/spinal-cord-injury-and-terminology-flash-cards/
	Alternative sources

0 Warnings Reset Export Share

1. Introduction

Spinal Cord Injury (SCI) is the most devastating condition that transforms the individual from independency to dependency for all basic ADL activities. It is responsible for high cost disability. SCI produces motor paralysis, sensory disturbances, chronic inflammatory

65% #1 Active

term tetraplegia refers to loss of motor or sensory function in cervical segments secondary to damage of neural elements within the spinal canal. It is characterized by impairment of function in arms as well as in trunk, legs and pelvic organs.

The term paraplegia means

loss of motor or sensory function in thoracic, lumbar and sacral segments

producing impairment of functioning of trunk, legs and pelvic organs. Etiology of SCI is grossly divided into traumatic and non-traumatic pathologies. Traumatic etiologies are due to road traffic accident (RTA), falls, violence and sports injuries. Non-traumatic etiology is due to infection, inflammation, tumors, degenerative changes and vascular malformation involving spinal cord. SCI produces short and long-term complications affecting the entire system of body. Physical inactivity, abnormal fat distribution, chronic inflammatory state leads to metabolic derangements thereby predisposing the individual for premature cardio vascular morbidity [1]. Early screening and appropriate management of these metabolic derangements may reduce the cardio vascular mortality.

2. Justification

Acute management of spinal cord injury is mostly focused on airway, breathing, circulation, spinal protection either surgery or by conservative measures and management of associated injuries. Considering the post-acute care management of SCI, emphasis is on prevention and management of complications such as skin breakdown, venous thrombo embolism, respiratory, genito urinary and gastro intestinal problems. Apart from the prevention and management, main objective is enabling the ambulation of patients and enabling patients to self-reliant to address the basic ADL. As there is tremendous improvement in acute care management, mortality due to septicemia and respiratory complications are in decreasing trend. Nowadays cardio vascular disease is growing concern in

Urkund's archive: / Anjali Bhise Ch-3 Review of Literature.pdf 65%

The contents of the source document cannot be displayed!

Possible reasons:

1. The document is stored in the URKUND Partner section and is listed as inaccessible. If you do not own this book already, you need to purchase it from the vendor.

2. The document has been exempted as a viewable source in the URKUND Archive by the author.

CERTIFICATE

This is to certify that this dissertation work titled "**To Assess Cardiovascular Risk in cases of Spinal Cord Injury by screening Impaired Glucose Tolerance and Dyslipidemia – Cross sectional Study** " of the candidate **DR. Kamakshi RM** with registration Number **201629002** for the award of **MD** in the branch of **Physical Medicine & Rehabilitation**. I personally verified the urkund.com website for plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 3 percentage of plagiarism in the dissertation.

Prof. DR. C. Ramesh, MD (PMR), DNB (PMR), D Phys Med, DA.

Guide & Supervisor sign with Seal

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.RM Kamakshi
I Year PG in MD PMR
Institute of Rehabilitation Centre/
Madras Medical College
Chennai 600 003

Dear Dr.RM Kamakshi,

The Institutional Ethics Committee has considered your request and approved your study titled **"TO ASSESS CARDIOVASCULAR RISK IN CASE OF SPINAL CORD INJURY BY SCREENING IMPAIRED GLUCOSE TOLERANCE AND DYSLIPIDEMIA – PROSPECTIVE STUDY " - NO.08032017(II)**

The following members of Ethics Committee were present in the meeting hold on **07.03.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr. K.Narayanasamy,MD,DM.,Dean(FAC), MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.S.Mayilvahanan,MD.,Director,Inst.of Int.Med.MMC,Ch-3	: Member
5.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
6.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
7.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

LIST OF ABBREVIATIONS

ADA	-	American Diabetes Association
ADL	-	Activities of Daily Living
AGE	-	Advanced Glycation End products
ATP	-	Adult Treatment Panel
ASIA	-	American Spinal Injury Association
BMI	-	Body Mass Index
BP	-	Blood Pressure
CAD	-	Coronary Artery Disease
CHART	-	Craig Handicapped Assessment and Reporting Technique
CVS	-	Cardio Vascular System
DVT	-	Deep Vein Thrombosis
FBS	-	Fasting Blood Sugar
FIMS	-	Functional Independence Measure
GRASP	-	Graded Redefined Assessment of Strength, Sensibility and Prehension
GERD	-	Gastro Esophageal Reflux Disorder
HbA1C	-	Glycosylated Hemoglobin
HDL	-	High Density Lipoprotein

HIV	-	Human Immune Deficiency Virus
HO	-	Heterotopic Ossification
HS - CRP	-	Highly Sensitive C Reactive Protein
ICF	-	International Classification of Functioning
IFG	-	Impaired Fasting Glucose
IGT	-	Impaired Glucose Tolerance
IHD	-	Ischemic Heart Disease
LDL	-	Low Density Lipoprotein
LMN	-	Lower Motor Neuron
MBG	-	Mean Blood Glucose
MSK	-	Musculo Skeletal
MVO2	-	Myocardial Volume Oxygen (Consumption)
NO	-	Nitric Oxide
NSCISC	-	National Spinal Cord Injury Statistical Center
OGTT	-	Oral Glucose Tolerance Test
PPBS	-	Post Prandial Blood Sugar
ROS	-	Reactive Oxygen Species
RTA	-	Road Traffic Accident
SCI	-	Spinal Cord Injury

SMA	-	Superior Mesenteric Artery
TB	-	Tuberculosis
TGL	-	Triglycerides
UTI	-	Urinary Tract Infection
VLDL	-	Very Low - Density Lipoprotein
WHO	-	World Health Organization

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TEMPLATES

LIST OF TABLES

LIST OF CHARTS

Charts

1. INTRODUCTION

Spinal Cord Injury (SCI) is the most devastating condition that transforms the individual from independency to dependency for all basic ADL activities. It is responsible for high cost disability. SCI produces motor paralysis, sensory disturbances, chronic inflammatory state and dysautonomia.

SCI are divided into two broad categories like paraplegia and tetraplegia. The term tetraplegia refers to loss of motor or sensory function in cervical segments secondary to damage of neural elements within the spinal canal. It is characterized by impairment of function in arms as well as in trunk, legs and pelvic organs. The term paraplegia means loss of motor or sensory function in thoracic, lumbar and sacral segments producing impairment of functioning of trunk, legs and pelvic organs.

Etiology of SCI is grossly divided into traumatic and non-traumatic pathologies. Traumatic etiologies are due to road traffic accident (RTA), falls, violence and sports injuries. Non-traumatic etiology is due to infection, inflammation, tumors, degenerative changes and vascular malformation involving spinal cord.

SCI produces short and long-term complications affecting the entire system of body. Physical inactivity, abnormal fat

distribution, chronic inflammatory state leads to metabolic derangements thereby predisposing the individual for premature cardio vascular morbidity [1].

Early screening and appropriate management of these metabolic derangements may reduce the cardio vascular mortality.

2. JUSTIFICATION

Acute management of spinal cord injury is mostly focused on airway, breathing, circulation, spinal protection either surgery or by conservative measures and management of associated injuries. Considering the post-acute care management of SCI, emphasis is on prevention and management of complications such as skin breakdown, venous thrombo embolism, respiratory, genito urinary and gastro intestinal problems.

Apart from the prevention and management, main objective is enabling the ambulation of patients and enabling patients to self-reliant to address the basic ADL.

As there is tremendous improvement in acute care management, mortality due to septicemia and respiratory complications are in decreasing trend. Nowadays cardio vascular disease is growing concern in cases of SCI as it occurs prematurely and more prevalent when compared to able bodied counterparts.

Carbohydrate intolerance, insulin resistance, lipid abnormalities, heart disease and cerebro vascular disease occur prematurely and at higher prevalence in patients with SCI [1]. This is because of metabolic changes, changes in body composition that results from paralysis, loss of lean tissue from denervation, obesity, greater adiposity above and below the neurological level of injury.

Symptoms of diabetes are more often masked, and patient may not be aware of symptom of diabetes because of the overlapping of symptoms associated with SCI.

There is disconnection between autonomic circuit and supra spinal control in SCI and Coronary Artery Disease (CAD) is asymptomatic due to reduced sensory feedback of angina. Physical inactivity, increased abdominal fat promotes insulin resistance and reduces HDL thereby promoting atherosclerosis. In addition to this chronic inflammation, blood pressure irregularities and reduced cardio vascular fitness secondary to SCI further increase the cardio vascular risk.

As of now there is no routine screening undertaken to assess the lipid and carbohydrate abnormality in case of SCI. Hence including these risk parameters during routine follow up would help in minimizing the cardiovascular risk

3. AIMS AND OBJECTIVES

AIM

To screen the individuals with spinal cord injury for impaired glucose tolerance and dyslipidemia for early assessment of cardiovascular risk and mortality.

OBJECTIVE

- To study the incidence of Carbohydrate and lipid abnormality in cases of SCI
- To stress the necessity of early rehabilitative measures

4. REVIEW OF LITERATURE

4.1 SPINAL CORD INJURY MANAGEMENT - EVOLUTION

Spinal Cord Injury (SCI) is life changing event irrespective of etiology. Going through the history of spinal cord injury comes the first Egyptian physician, approximately 5000 years ago (2500 – 2400 BC), Dr. Edwin Papyrus suggested that it is an ailment not to be treated [2,3]. He clearly mentions incontinence and erectile dysfunction associated with vertebral dislocation.

Hippocrates (460 – 330 BC) who is father of medicine and orthopedic described traction to reduce the injuries.

Then Paul of Algina (625 to 680 AD) used windlass to reduce the dislocation and recommended the laminectomy.

Roland of Parma suggested the need for early treatment of SCI. Ambrose Pare (1564–1598) recommended laminectomy for SCI.

Henry Clive (1750–1827 AD) performed the first laminectomy. Charles Bell the Neurologist identified the Renal failure being the cause of death in SCI. William Wagner (1848 – 1900 AD) a German general surgeon described practical treatment of SCI.

Astley Cooper in UK in 19th Century gave detailed description about clinical manifestation of SCI.

Donald Munro (1889 – 1973 AD) started centre for SCI at Boston and he was known as father of paraplegia and was willing to carry out rhizotomies.

Ludwig Guttman followed Munro and suggested modern treatment for SCI.

General George Patton sustained cervical spine injury and refused all the treatment and he is reported t0 have died of CVS complications.

Hence in the past SCI is considered as an ailment not to be treated. In the present an ailment to be treated. In the future an ailment to be cured.

4.2 ANATOMY OF SPINAL CORD

Spinal Cord is located within the vertebral canal and it extends from foramen magnum to the lower part of first lumbar vertebra [4]. The distal end of spinal cord is cone shaped and it is known as conus medullaris. A fine filament of connective tissue known as filum terminale which is the pial extension continues inferiorly from the apex of conus medullaris.

Spinal cord has two enlargements in the cervical and lumbo sacral region. Cervical enlargement occurs in the region associated with origins of spinal nerves (C5 to T1) innervating the upper limbs. Lumbo sacral enlargement is seen in the region associated

with origin of spinal nerves (L1 to S3) innervating the lower limbs.

Spinal cord is covered by meningeal coverings such as pia, arachnoid and dura that are continuous with those of brain.

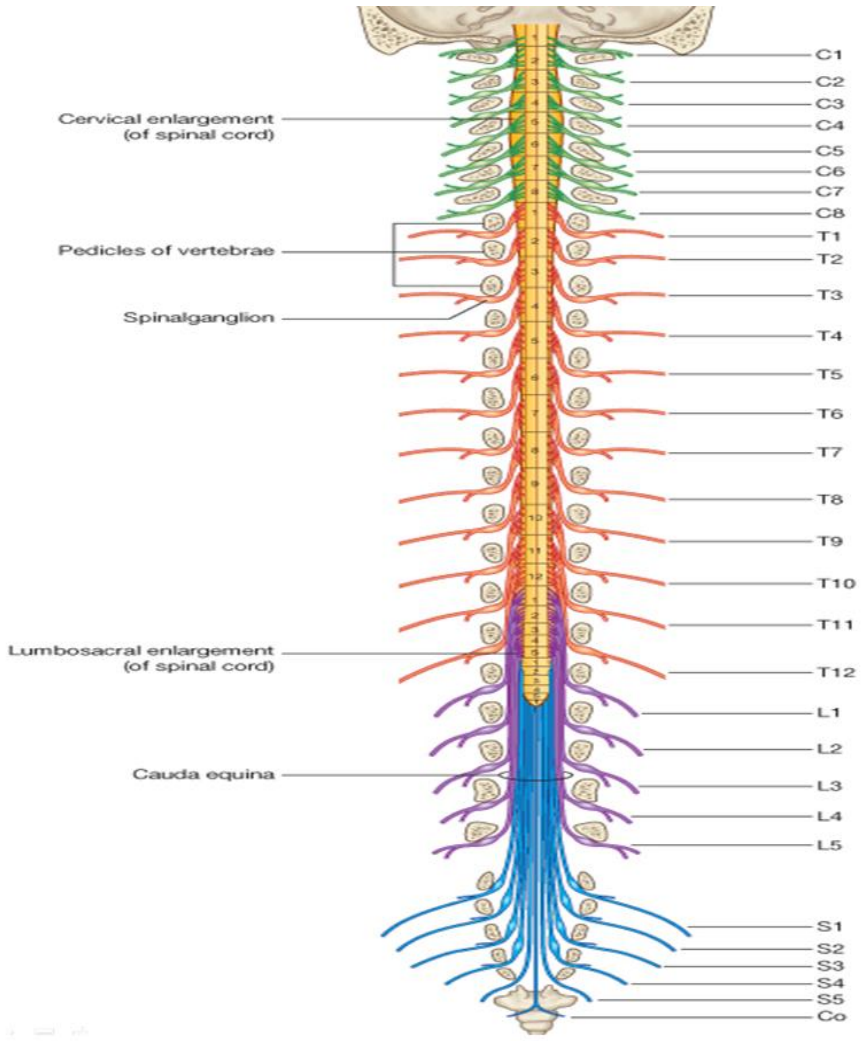


Figure 1. Spinal Cord Anatomy

TABLE - 1

Relationship between Vertebral level and SC level

Vertebral Body	Spinal Cord Level
Upper Cervical (C1 – C4)	Same as vertebral level
Lower Cervical (C5 – C7)	Add one level
Upper thoracic (T1 – T6)	Add two levels
Lower thoracic (T7 – T10)	Add three levels
T11 – T12	Lumbar
T12 – L1	Sacral
L2 and Below	Cauda Equina

4.3 BLOOD SUPPLY OF SPINAL CORD

Arterial supply of spinal cord is through 3 longitudinal arteries (anterior spinal artery and paired posterior spinal arteries) and feeder vessels through segmental arteries.

Anterior spinal artery arises from branch of vertebral artery supply anterior two third of the cord. Posterior spinal artery arises from terminal branch of vertebral artery and supply and posterior one third of the cord.

Segmental spinal artery arises predominantly from vertebral and deep cervical arteries in the neck, posterior intercostal arteries in thorax and the lumbar arteries in the abdomen.

Segmental arteries give of anterior, posterior radicular arteries and segmental medullary arteries

Myelopathy due to vascular cause is secondary to

- Spinal cord infarction
- Hemorrhage within the spinal cord
- Hemorrhage within the epidural or subdural space causing spinal cord compression
- Paraplegia is more common than tetraplegia in myelopathy
- Mid thoracic spinal cord region is the most commonly affected region
- Onset of the disease is acute with rapid progression
- Poor prognosis for neurological recovery in complete lesion

TABLE - 2

Applied Anatomy of Blood Supply

Blood Supply of the Spinal Cord	Clinical Significance
Single Anterior spinal artery supplies anterior two third of the cord and two posterior spinal arteries supplying posterior one third	Spinal cord infarction results in anterior cord syndrome with paralysis and impaired pin prick and temperature sensation with relative sparing of posterior column
Relatively avascular watershed area in the mid thoracic region between anterior spinal artery and artery of Adamkiewicz	Mid thoracic (T4 to T8) level is the common site spinal cord infarction
Anterior horn cells are venerable ischemia due to high metabolic demands	Preferential injury to anterior horn cell produces flaccid paralysis
Border zone in the spinal cord between the penetrating arteries from anterior and posterior circulation can be relatively avascular	It is the contributing factor in the pathogenesis of central cord syndrome

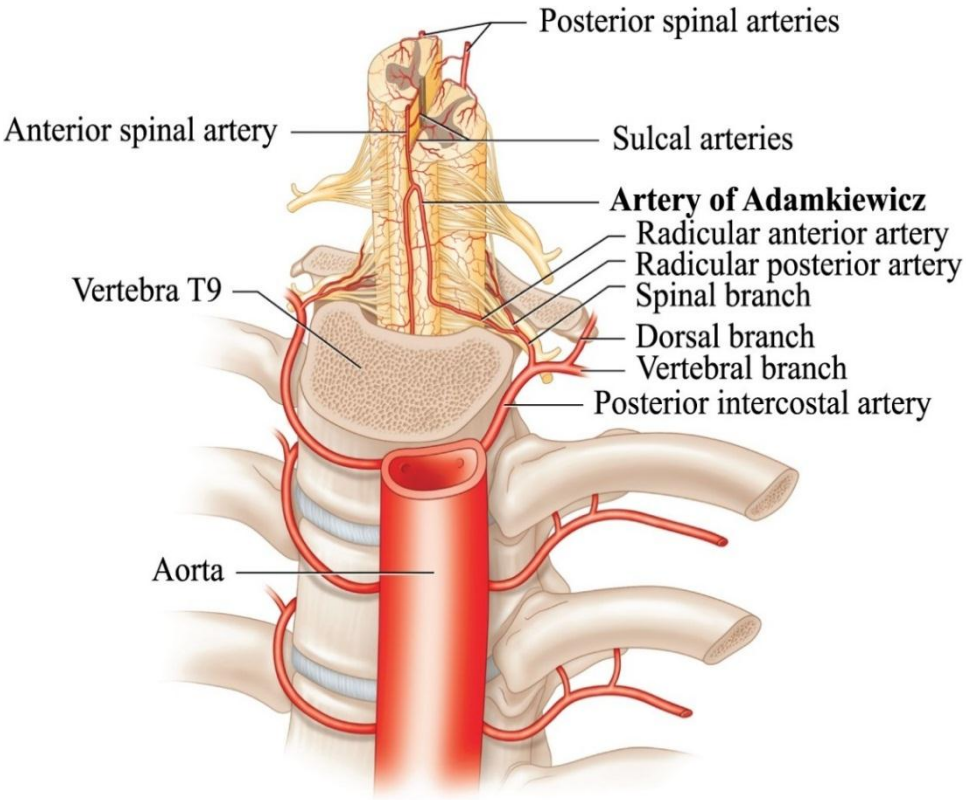


Figure 2. Blood Supply of Spinal Cord

4.4 EPIDEMIOLOGY AND DEMOGRAPHIC FACTORS OF TRAUMATIC SPINAL CORD INJURY

4.4.1 Global Incidence

Based on national spinal cord injury statistical center database (NSCISC), incidence in United States is 54 cases per million population [5,6] or about 17,700 new cases every year whereas in Canada it is 53 cases per million population. In Spain and France 24 to 19 cases respectively. Between 12 and 14 cases

in Netherland, Qatar, Ireland, Finland and Australia. Less than1% of person experiences complete neurological recovery.

4.4.2 Age, Gender and Marital status

Bimodal distribution highest among young adults and older individual (more than 65 years). Majority of SCI occurs in males (70 to 80%). Age of the injury increased from 29 to 43 years. The divorce rate is more after SCI as compared to general population affecting the quality of life and leads to depression. Depression is also considered as one of the risk factors for cardiovascular disease

4.4.3 Indian Scenario

Approximately 1.5 million people live with SCI. 20, 000 new cases are added every year [7].

Males are predominantly affected [age group – 16 to 30 years old]. Higher incidence in young active, reproductive population of society. 60 to 70% are from rural area consisting of poor illiterate population.

Secondary complications after spinal cord injury are more than the western population.

TABLE - 3

Global Variation in Etiology of SCI

Falls from trees and roof top are the most common reported cause of traumatic SCI in South Asia
RTI in South East Asia is likely to involve two wheeled and nonstandard transportation than the four-wheeled motor vehicle. RTI is the most common cause in the developed countries
Violence related SCI is most prevalent in Sub Saharan Africa.
Australia and Western Europe has low proportion of violence related SCI than North America
Non-Traumatic SCI is related to degenerative conditions of the spine followed by spinal tumors in developed countries.
Infection like TB and HIV are predominant cause of non-traumatic SCI in developing countries

4.4.4 Neurological Level and Extent of Neurological Deficit

According to NCISCI and nationwide emergency department sample database, incomplete tetraplegia is more common than paraplegia that accounts for 52 to 57 % of cases. Among these

29% comes under complete SCI category. Older individual with cervical cord lesion has incomplete neurological lesions.

4.4.5 Life Expectancy and Morbidity

When compared to general population life expectancy of people with SCI is 2 to 5 times less.

The mortality is highest during first post injury year and declines thereafter. Significant factors of mortality include level and completeness of injury, age at the time of injury and ventilatory support.

Additional factors affecting the longevity after first post injury period are low life satisfaction, poor health, emotional disturbance, functional dependence and poor adjustment to disability.

Diseases of respiratory system especially pneumonia is the leading cause of death both during first post injury year and during the subsequent years [8]. Heart disease ranks second as per NSCISC 2012 records.

TABLE - 4

Primary cause of death in SCI

Common Cause of Death	%
Diseases of the respiratory System	22
Infective and Parasitic Diseases	12
Neoplasms	10
Hypertensive and Ischemic Heart Disease	10
Other Heart Disease	9
Unintentional Injuries	7
Diseases of Digestive System	5
Cerebro Vascular Diseases	4
Diseases of Pulmonary Circulation	4
Suicides	4

4.5 PATHOPHYSIOLOGY OF SCI

SCI causes primary mechanical damage sustained at the time of impact and secondary damage due to the pathological events following primary injury [9,10]. The extent of damage depends upon the energy delivered to the spinal cord at the time of impact.

Pathophysiological process of secondary damage after the SCI are due to

a) Ischemia and micro vascular perfusion alteration

b) Free radical generation and lipid peroxidation

c) Excitotoxicity and calcium overload

d) Inflammatory and immunological responses

Physical injury to spinal cord results in laceration, contusion, compression, shear and traction of neural tissue.

Key process involved in pathophysiology

- Both necrotic and apoptotic cell death occur
- Necrotic cell death involves swelling and membrane lysis in response to severe insult affecting the cells homeostatic mechanisms.
- After SCI, the cells around the region of injury that are initially scarred may also experience subsequent biochemical insult due to activation of caspases

4.6 COMPLICATIONS OF SCI

SCI affects entire system of body [11]. Depending on the level of vertebra affected lesion are classified as either paraplegia or tetraplegia.

Spinal cord is shorter than the vertebral column and it ends between L1 and L2.

Injury affecting the central part of the spinal cord often have pronounced upper limb weakness than lower limb and they will have sacral sparing. This is known as central cord syndrome.

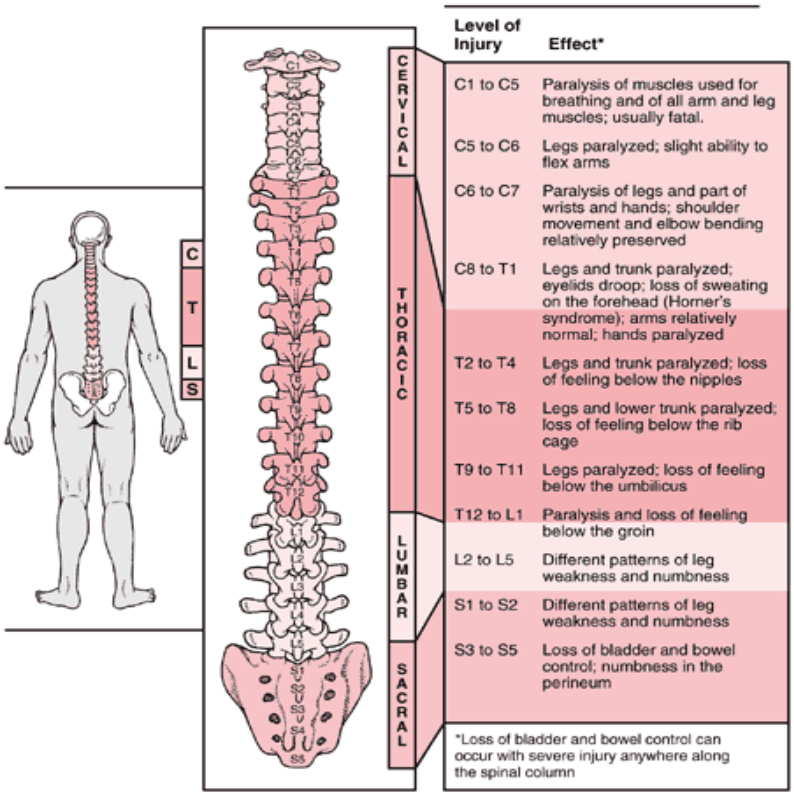


Figure 3: Effect of SCI

Below sub section describes the complications includes

4.6.1 Metabolic and Endocrine Issue

SCI may lead to several endocrine and metabolic abnormalities [12,13,14]

Loss of somatic and autonomic control leads to physical inactivity. There will be abnormal distribution of fat and insulin resistance producing metabolic abnormalities.

TABLE - 5

Metabolic and Endocrine issues

Condition	Considerations
Body composition changes	Loss of lean body mass, increased relative adiposity
Reduced Energy expenditure	Reduced Basal metabolic rate
Carbohydrate Metabolism	Impaired glucose tolerance, Diabetes Mellitus, Metabolic Syndrome
Lipid Metabolism	Low HDL
Bone loss and calcium metabolism	Hypercalcemia in acute stage High rate of bone resorption and bone loss below the level of injury
Anabolic Hormone deficiency	Growth Hormone and testosterone

Condition	Considerations
Hypo albuminemia, anemia	Relatively common in SCI
Hyponatremia	Seen in chronic tetraplegia due to altered regulation of anti-diuretic hormone
Effect and drug metabolism	Rapid absorption of acidic drugs, delayed absorption of basic drugs Reduced renal elimination of drugs Reduced absorption of intramuscular drugs below injury level

4.6.2 Metabolic Syndrome

Risk of metabolic syndrome is increased after SCI. It is associated with proinflammatory and prothrombotic state for accelerating atherosclerosis [14,24,25,44]. Abdominal or visceral adipose tissue secretes inflammatory cytokines and prothrombotic agents that cause indirect vascular endothelial injury and inhibits fibrinolysis respectively. These factors are responsible for increased cardio vascular risk in SCI.

Components of metabolic syndrome as per NCEP ATP – III

- Central obesity (waist circumference more than 102 cm in male, more than 88 cm in female)
- Atherogenic dyslipidemia (TGL more than 150 mg/dl and HDL less than 40 mg/dl in male and less than 50 mg % in female)
- Hyper tension (BP more than 130/85 mm Hg)
- Insulin resistance and hyper glycemia (fasting glucose more than 110 mg / dl) Metabolic syndrome in SCI
- Abdominal muscle paralysis may increase waist circumference measurement so that may not be accurate assessment of central obesity.
- Neurogenic hypotension or autonomic dysreflexia may confound blood pressure readings
- Ideal body weight need to be adjusted downward by 5 to 10% for paraplegia and 10 to 15% for tetraplegia.
- Lowered cut off values for overweight are suggested in SCI

Below figure shows the influence of metabolic syndrome on atherosclerosis

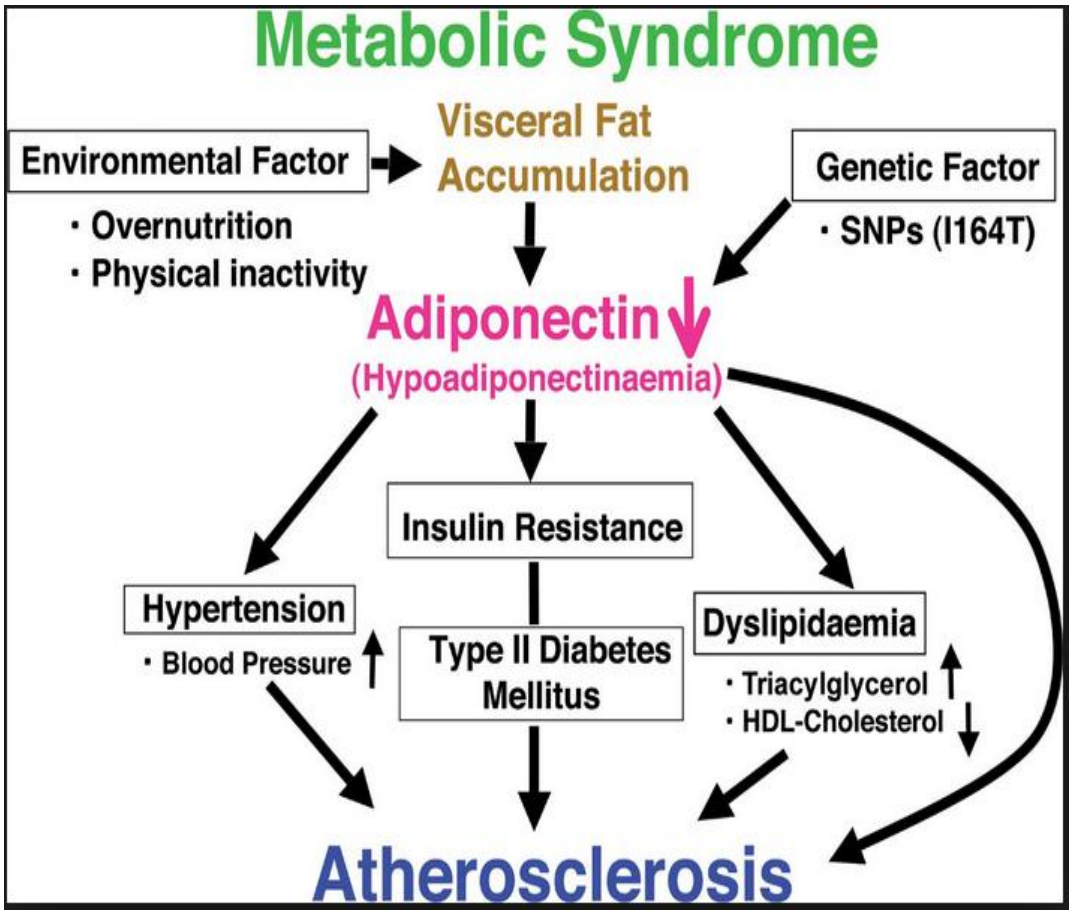


Figure 4: Metabolic Syndrome

4.6.3 Cardio vascular Complication

Autonomic dysfunction is responsible for the altered regulation of heart and vasculature. Autonomic dysregulation of the heart produce alteration in cardiac electrophysiology thereby increases the susceptibility to arrhythmia [15]. Patients with SCI have increased incidence of atherosclerotic disease due to overweight, lipid and carbohydrate abnormalities in addition to inherent risk factors.

Autonomic Innervation

Descending autonomic pathways from brain travel in spinal cord and terminates on pre-ganglionic sympathetic neurons in spinal cord which are located at T1 to L2. Pre-ganglionic parasympathetic neurons to the bladder, reproductive organs and the lower part of the gut are located at S2 to S4. The rest of the parasympathetic innervation to thoracic and abdominal viscera is through the vagus nerve.

Depending on the level of the SCI various parts of sympathetic nervous system will be disconnected from supra spinal control that will result in altered sympathetic activity below the level of injury.

Parasympathetic activity reduces the heart frequency and contractility. Interruption of cardiovascular control following SCI are directly related to the level and degree of injury. In case of complete cervical injury connection between upper autonomic centers in the brain and intermediolateral cell column at levels T1 to L2 of spinal cord will be destroyed. Patients with cervical injury have risk of bradycardia (29%), sudden cardiac arrest (16%) and conduction system disturbance in first few weeks after injury.

Tetraplegia is frequently accompanied by autonomic dysreflexia decreased transmission of cardiac pain, loss of muscle mass in left ventricle and pseudo infarction.

Autonomic innervation of the organs is shown below

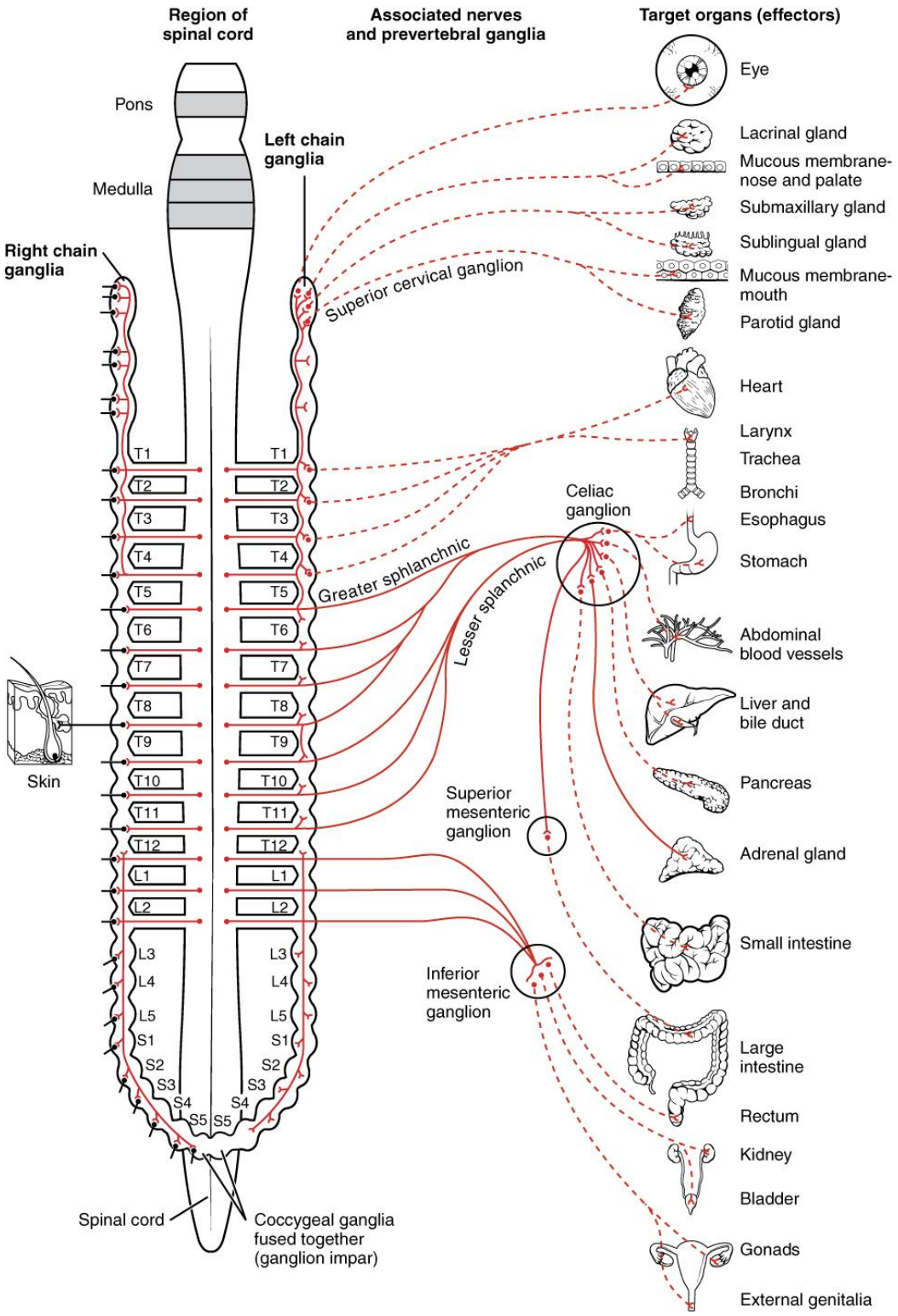


Figure 5. Autonomic Nervous System Anatomy

Tetraplegia is frequently accompanied by autonomic dysreflexia decreased transmission of cardiac pain, loss of muscle mass in left ventricle and pseudo infarction. Sudden loss of autonomic effect of smooth muscle in blood vessel wall produces vasodilatation. Vagus is hyper sensitive immediately after an injury and this last for 2 to 3 weeks. Sometimes it is prolonged requiring pace maker. Hypoxia promotes vagal action and hence hypo ventilation is to be avoided.

Loss of supra spinal regulatory control of sympathetic nervous system results in reduced sympathetic activity below the level of injury and is responsible for hypo tension, bradycardia and blended CVS response to exercise [16].

Common cardio vascular complications in both acute and chronic as per Phillip's et al [17]

Acute phase

a) Sinus bradycardia

b) Loss of vascular tone

c) Supra ventricular / ventricular ectopic beats

d) Arterial hypo tension

e) Orthostatic hypo tension

f) Enhanced vasovagal reflexes

g) Vasodilation and venous stasis

Chronic phase

a) Autonomic dysreflexia

b) Ortho static hypo tension – it is defined as reduction in systolic blood pressure of 20 mm Hg or reduction in diastolic pressure of 10 mm in Hg during first 3 minutes of upright position. It is reported to be more common in traumatic than non-traumatic SCI. Physical exertion, heavy meals, dehydration, rapid position change will exacerbate the symptoms of ortho static hypotension.

c) Impaired cardio vascular reflexes

d) Loss of reflex changes in the heart (T1 to T4)

e) Coronary Artery Disease (CAD)

f) Atrophy of the heart with tetraplegia

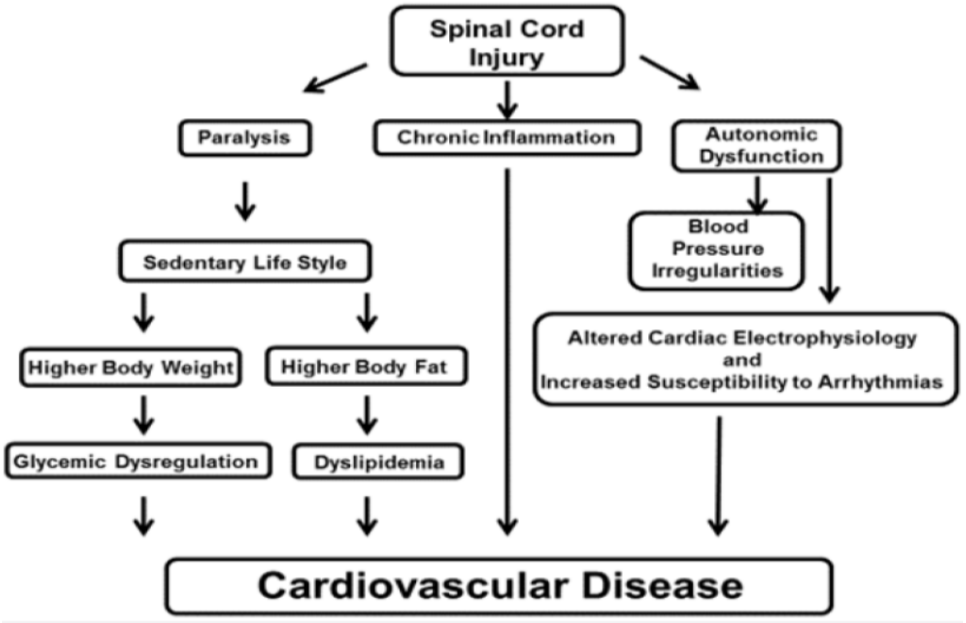


Figure 6: Overview of pathophysiology in SCI

Autonomic dysreflexia

It occurs in patients with injury at T6 or above. It is induced by sensory stimulation below the level of lesion, resulting in uncontrolled sympathetic response [18]. It is characterized by pounding headache, sudden significant increase in BP, flushing of skin above the level of lesion, blurred vision, nasal congestion, piloerection, bradycardia, cardiac arrhythmia. It occurs most frequently during the first two to four months after injury. The life time frequency is 19 – 70 %. In 85% of cases, it is due to full urinary bladder. A rise in systolic blood pressure of 20 to 40 mm of Hg above the normal levels in adults and in children more than 15 mm of Hg. It may be sign of autonomic dysreflexia. If left untreated it may lead to cerebral hemorrhage.

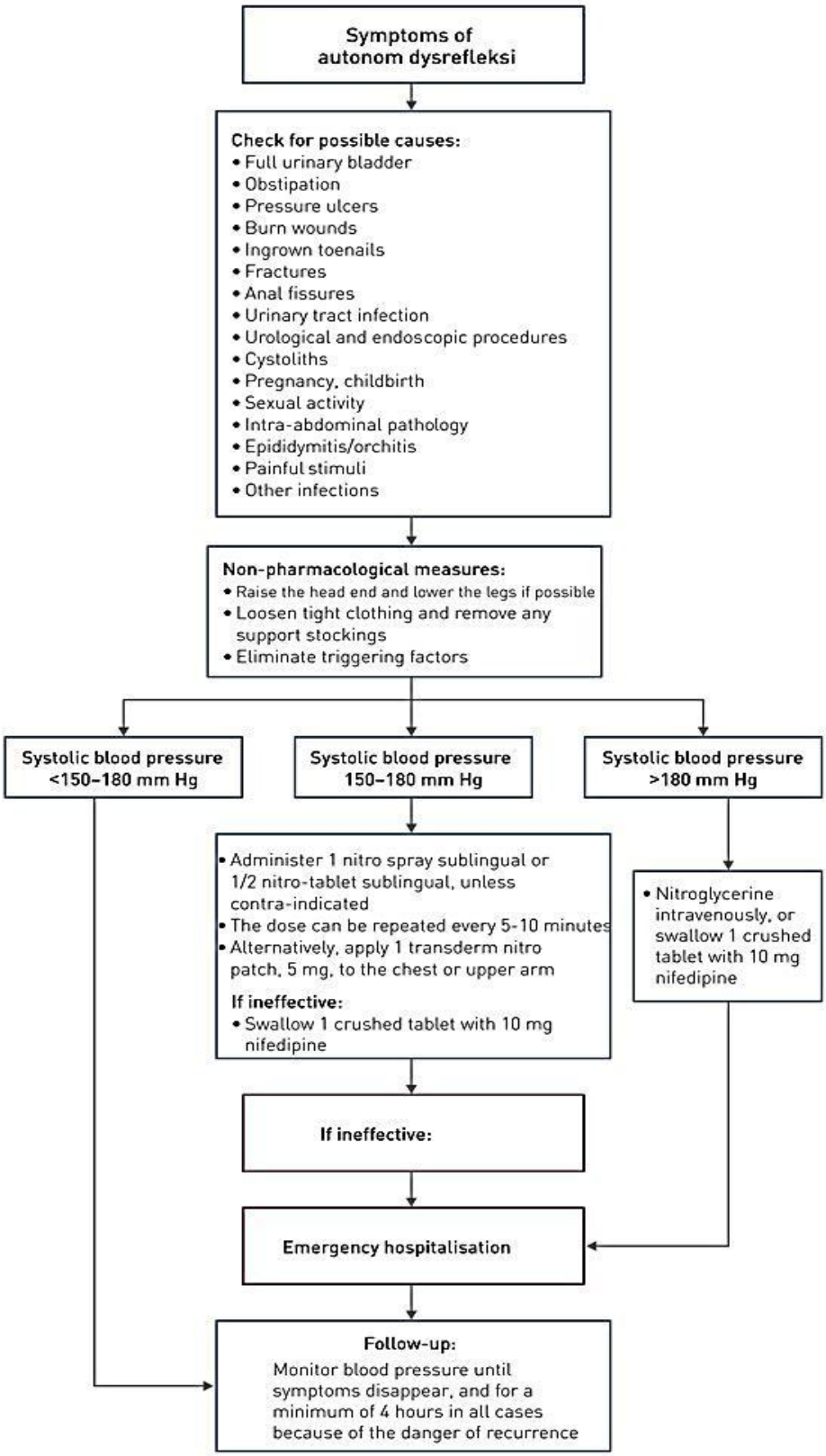


Figure 7: Autonomic dysreflexia

Ischemic Heart Disease (IHD)

It is the major cause of morbidity and mortality in SCI. Low HDL, physical inactivity, increased adiposity, glucose intolerance are specific risk factors for IHD in SCI and these patients will have atypical presentation of CAD [19,20,21].

TABLE - 6

Unique Issues in diagnosis of IHD

Atypical Presentations, lack of chest pain
Under diagnosis of IHD
Delayed treatment, inadequate secondary prevention
Confusing physical signs
Non-Specific ST segment and T wave changes
Cardiac stress testing • Inability to perform, traditional tread mill test • Sub optimal sensitivity of arm vs leg exercise • Indication for pharmacologic stress testing

Below figure depicts the atherosclerotic burden in SCI

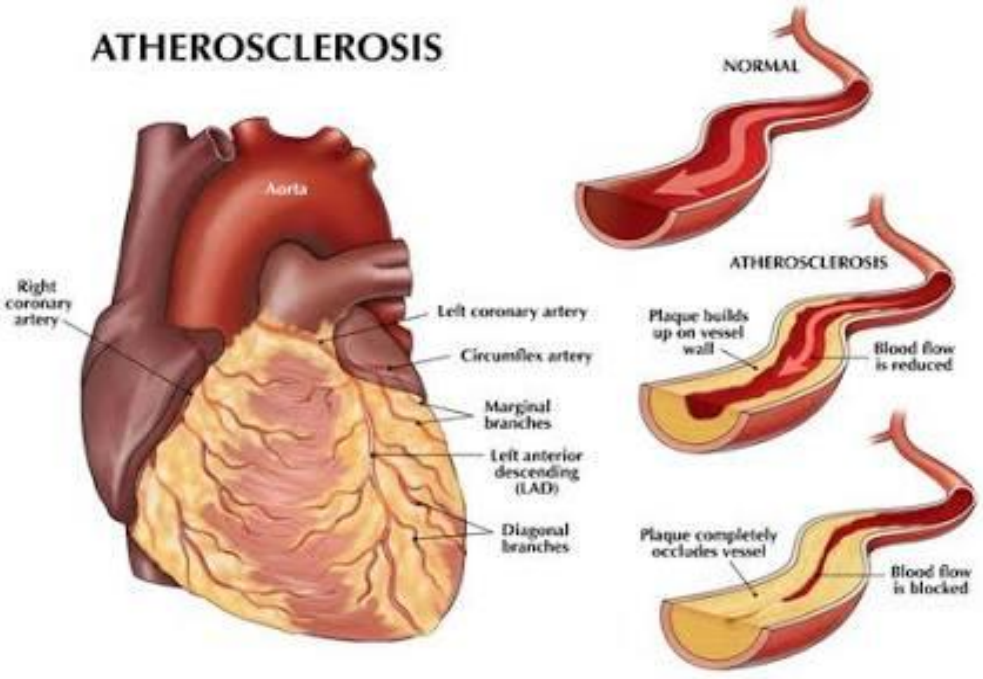


Figure 8: Atherosclerosis

4.6.4 Effect of Hyper Glycemia on Cardio Vascular System

Hyperglycemia has the detrimental effecton

a) Coronary microcirculation

b) Collateral circulation

c) Oxidative stress [21, 22]

d) Coronary vasodilation

e) Signal transduction

f) Endothelial function

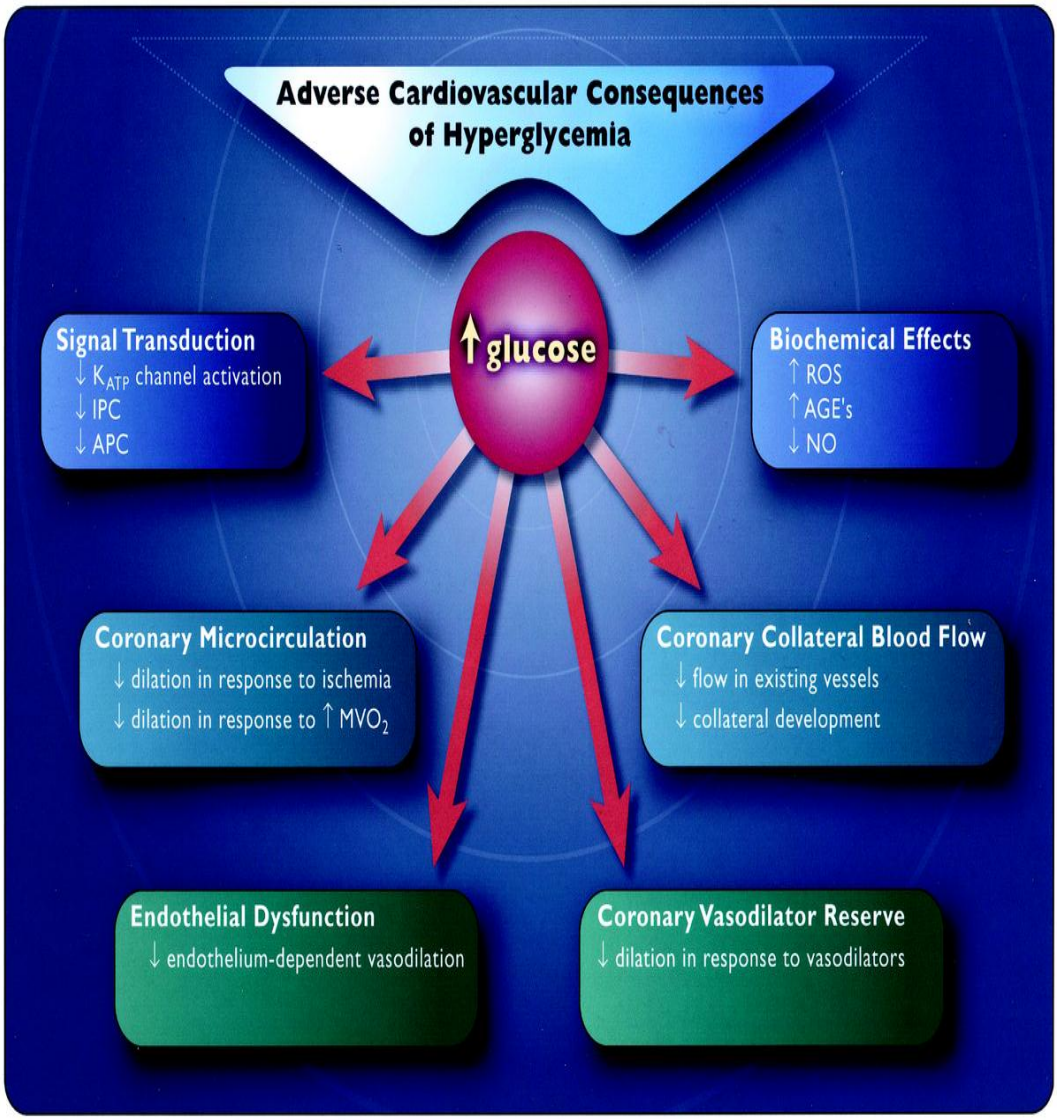


Figure 9: Effect of hyper glycemia

4.6.5 Peripheral Vascular Disease

Delay in the diagnosis of peripheral vascular disease is possible because of lack of cardinal symptom of intermittent claudication, rest pain or numbness. Patients may first present with gangrenous changes. Hence the incidence is high in the cases 0f SCI.

4.6.6 Respiratory dysfunction

SCI results in impaired ventilation secondary to paralysis of diaphragm, impaired cough because of expiratory muscle weakness and paradoxical breathing due to intercostal muscle paralysis [23]. Due to this effect complication encountered are

- Pneumonia
- Atelectasis
- Ventilatory failure
- Sleep disordered breathing
- Pulmonary embolism
- Neurogenic pulmonary edema

Higher incidence of this complication depends on the age, level of injury and type of injury.

It is leading cause of death in SCI in all years, 37% of death in first year and 27% beyond first year.

4.6.7 Vascular System

SCI Promotes

- Venous stasis because of loss of muscle pumping action of lower limbs and peripheral vasodilation [26].
- Hyper coagulability due to release of pro coagulant factors after injury

- Intimal injury due to trauma Problems encountered are Deep Vein Thrombosis (DVT, 50 – 75 % greater incidence during 7 – 10 days after injury without prophylaxis) and pulmonary embolism.

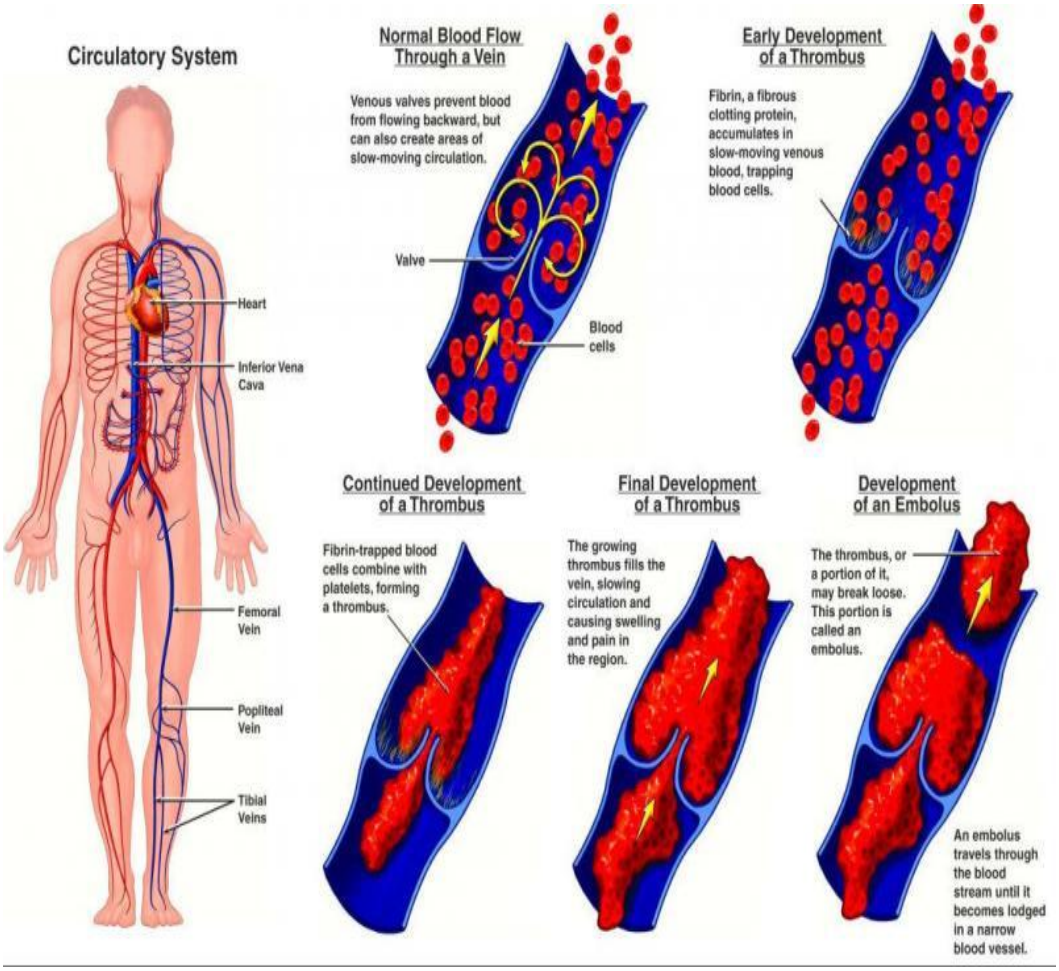


Figure 10: DVT

4.6.8 Impaired Thermo Regulation

Upper GI tract problems

- Poor Dental hygiene
- Dysphagia
- GERD
- Erosive Gastritis, ulcers
- Impaired Gastric motility [28]
- SMA syndrome
- Pancreatitis
- Gall Bladder disease

Neurogenic bowel

- SCI affects bowel activity by the following mechanism
- Temporary loss of reflex activity
- Effect on colorectal complaints and mobility
- Increased colonic transit time
- Alteration in anal sphincter control

Complication of neurogenic bowel include constipation, fecal impaction, diarrhea, rectal bleeding and hemorrhoids. Autonomic dysreflexia may result from constipation.

4.6.10 Genito Urinary Complications

It results in neurogenic bladder and voiding dysfunction. Void dysfunction leads to urinary tract infection (UTI) and renal

and bladder stones, hydronephrosis, vesico ureteric reflex, autonomic dysreflexia and catheter related complications [29,30]

UTI is the most common complication of neurogenic bladder dysfunction. The estimated rate of occurrence is between 1.5 to 2.5 episodes per patient per year.

Patients with SCI develop nonspecific symptoms like

- New onset of urinary incontinence
- Cloudy Urine
- Hematuria
- Increased spasticity
- Generalized malaise

4.6.11 Pressure Ulcer

Pressure, friction and shear are primary factors associated with pressure ulcer. Life time risk of pressure ulcer is estimated to be over 50% in people with SCI [31].

In the early post injury stage, the most common sites of pressure ulcers are sacrum, followed by heels and ischium. After two years of post-injury, most common sites are ischium, sacrum and trochanter.

Risk assessment tool are Braden scale, Salzberg scale and Norton scale.

Long standing ulcers of more than 20 years duration develop into squamous cell carcinoma.

4.6.12 Sexual Dysfunction

Complete SCI involving sacral segments (S2 – S4) leads to loss of reflex erection in men. Psycho genic erection is absent in SCI at or above T10. But often preserved with injury below T10 to L2 [32]

TABLE - 7

Sexual Dysfunction based on SCI level

Complete supra sacral injury, reflex erections are preserved in 90% of men
Complete SCI above T10, psycho genic erections are absent
With complete sacral SCI reflex erection are absent. Psycho genic erections are preserved in 25%
With incomplete SCI, greater the likelihood of preserved reflex and psycho genic erections
With complete SCI above T11 spontaneous ejaculation is rare

Temporary cessation of menses is typical after SCI and normal menstruation is restored within 6 to 12 months.

In addition to direct effect of SCI, pain, spasticity, difficult positioning, impaired hand functioning, neurogenic bowel and bladder issues and psychological / emotional issues can significantly affect sexual function.

Impaired sperm motility is common after SCI even though sperm concentration is less affected.

4.6.13 Musculo skeletal and Neurological Complication

Below are complications

- Post traumatic syringomyelia
- Scoliosis
- Tethered cord syndrome
- Vertebral pain and degeneration around injury site
- Charcot spine
- Fracture in chronic spinal cord injury
- Heterotopic ossification [33]
- MSK overuse syndrome
- Entrapment neuropathy
- Spasticity and fracture

Type	Description
Type 1 a= SCI, b=TBI	HO at the anterior hip or the proximal end of the femur, with or without ankylosis
Type 2 a= SCI, b=TBI	HO at the posterior hip or the proximal end of the femur, with or without ankylosis
Type 3 a= SCI, b=TBI	HO at the anterior & medial hip or the proximal end of the femur, with or without ankylosis
Type 4 a= SCI, b=TBI	HO around hip with or without ankylosis

Figure 11: HO Classification

TABLE - 8

Preventive Measures for Reducing Contracture

Contracture	Specific Preventive Measure
Tendo Achillis	Use of foot board
Hip Flexion	Occasional prone lying
Hip External rotation	Use of trochanter roll
Hand contracture	Use of palmar roll

Spasticity

It is defined as velocity dependent increase in tonic stretch reflexes or muscle tone. It is due to loss of descending inhibitory modulating signals.

Spasticity is common after SCI affecting 50 to 75 % of individuals.

Spasticity may lead to sleep disturbances, joint contracture or subluxation, pressure ulcer and pain.

4.6.14 Pain

It may be either nociceptive or neuropathic pain. Persistent pain is a significant problem affecting the quality of life [34].

Nociceptive MSK pain after SCI is due to activation of nociceptive receptors. It is most often result of over use injury such as wheel chair propulsion, overhead reaching and transfers.

Neuropathic pain is due to central as well as peripheral mechanism like cortical reorganization and abnormal sprouting and connections and loss of inhibitory inter neurons.

Chronic pain leads to depression, sleep disturbances and functional limitations.

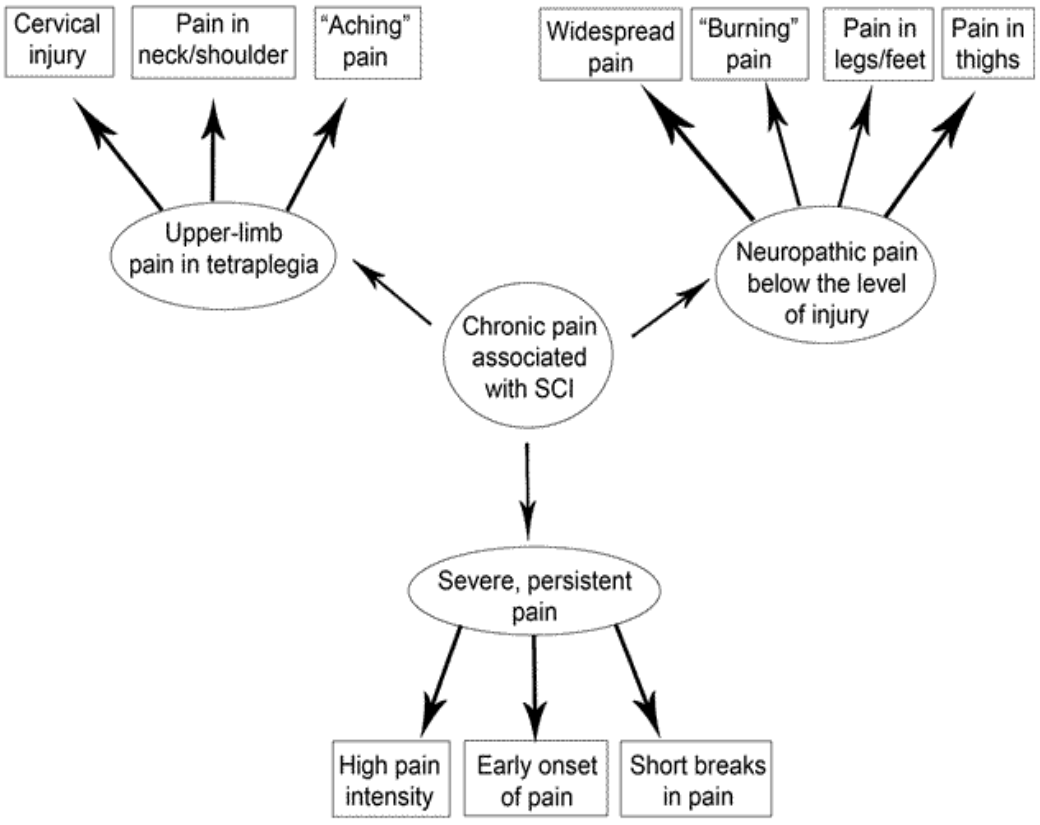


Figure 12: Pain patterns in SCI

Significant pain is estimated to occur in two third of SCI. Shoulder pain is present in 32 to 70% of people with SCI. Psycho social mechanism also play role in pain related sufferings.

SCI pain classification is shown below

TIER 1	TIER 2	TIER 3
Nociceptive pain	• Musculoskeletal pain • Visceral pain • Other nociceptive pain	-Shoulder osteoarthritis -Constipation -Autonomic dysreflexia headache
Neuropathic pain	• At level pain • Below level pain • Other neuropathic pain	-Spinal cord compression -Spinal cord ischaemia -Carpal tunnel syndrome
Other pain		-Fibromyalgia -Irritable bowel syndrome

Figure 13: SCI pain classification

- Pain is separated into 2 components
- Perception of pain (afferent) – due to physiological process
- Reaction to pain (efferent) – due to physiological as well as psychological process
- Spinal cord is responsible for pain transmission as well as modulation of pain signals.
- Below figure shows the pain pathway

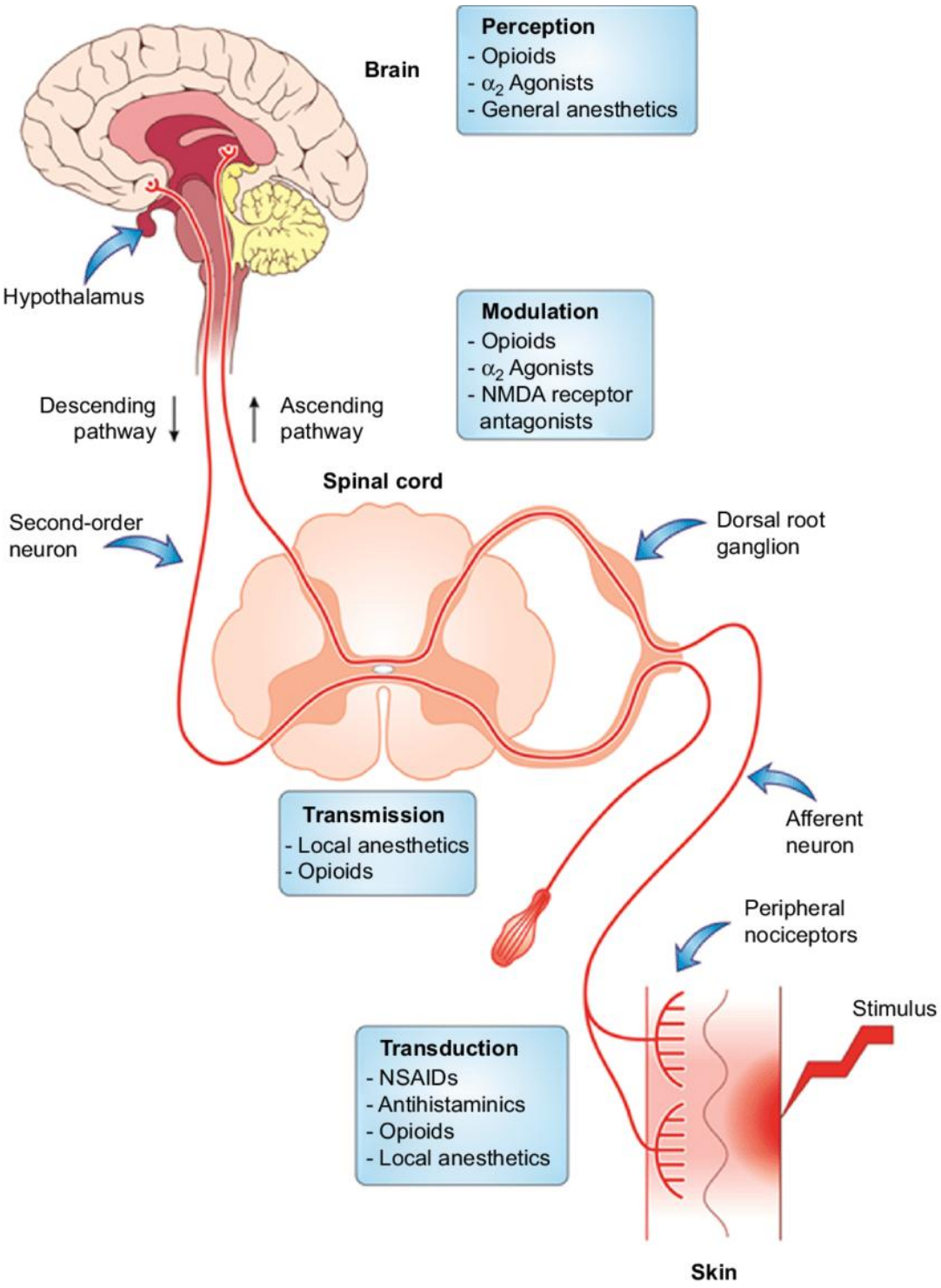


Figure 14: Pain Pathway

4.6.15 Osteoporosis

Dysfunction in calcium metabolism promotes immobilization hyper calcemia and secondary osteoporosis with fracture risk [35].

Excess bone resorption occurs below the level of injury. Bone loss starts within days to weeks after injury and continues for the first 6 to 12 months at the rate of 4% loss per month. Hyper calciuria develops within first week and continues for 6 to 18 months. Hyper calcemia can occur and it peaks between 1 and 6 months.

Pathophysiology of osteoporosis

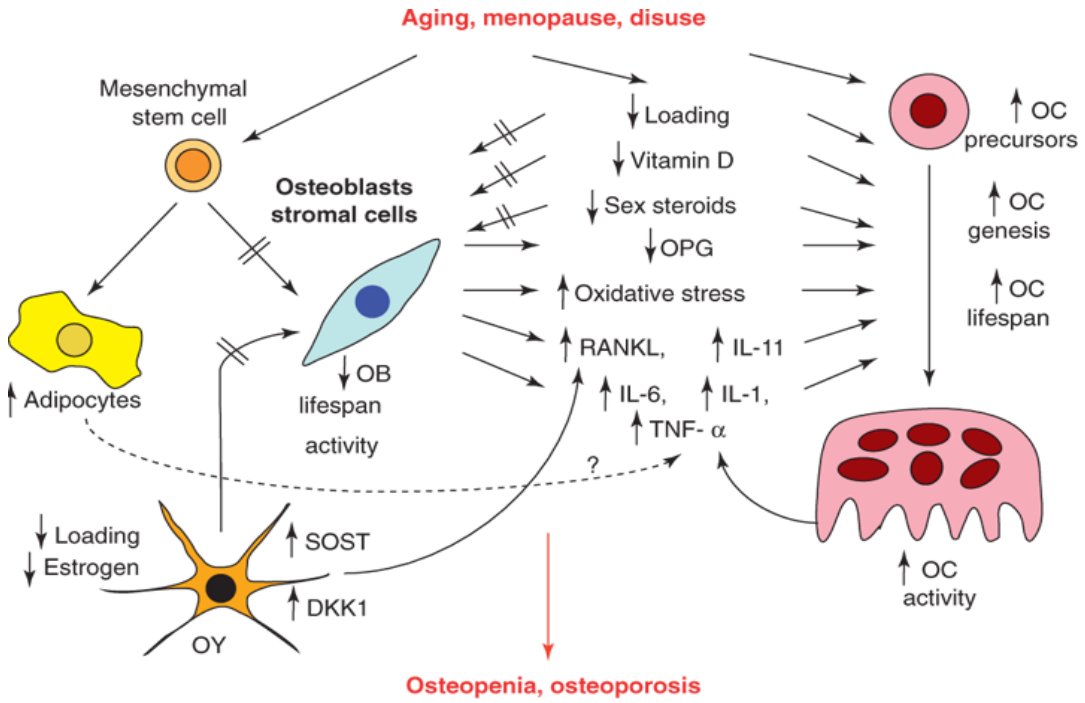


Figure 15: Pathophysiology of Osteoporosis

Mechanical, neurological and hormonal factors play a role in osteoporosis. Immobility leads to absence of mechanical stress on

the bone resulting in bone loss. Reduced testosterone and sympathetic response enhances the risk of osteoporosis

4.6.16 Psychological Issues in SCI

In acute stage nonspecific distress and shock of recent event is common. Psychological problems encountered in SCI are

- Depression [45]
- Post-traumatic stress disorder
- Alcohol and substance abuse
- Psychogenic paralysis

Depression

- It is present with the incidence of 20 to 30%
- Prior history of depression, family history of depression or suicide or associated traumatic brain injury or non-modifiable risk factors.
- Chronic pain, alcohol and substance abuse and reduced participation of modifiable risk factors.
- Risk of suicide is 3 to 5 times more than the general population
- Incidence of suicide is higher with complete paraplegia.
- Early morning awakening, fatigue in the morning are characteristic of depression.

Post-Traumatic Stress Disorder

- Diagnostic criteria for post traumatic stress disorder are
- History of exposure to traumatic event
- Negative alteration in cognition and mood that begin event after traumatic event,
- Alteration in arousal and reactivity
- Sleep disturbance
- All these symptoms should present for more than one month with functional impairment

Alcohol and substance abuse

- Incidence is higher than general population.
- Associated impaired judgment or cognition may lead to pressure ulcer
- It can lead to impulsivity and unsafe behavior.
- Increased risk of depression
- Negative impact on physical as well as psychological functioning

Dysfunctional behavior

- It involves anger and hostility
- Excess dependence on others
- Non-compliance

4.6.17 Socioeconomic Consequences and Quality of Life

SCI is associated with high cost both to individual and society.

This is attributed to improved life expectancy and increased cost of care overtime.

Rehospitalization add significant cost to the individual

Quality of life and life satisfaction has been shown to relate positively to social participation, social support and to perceived control over one's life.

4.7 EVALUATION OF SCI

Comprehensive evaluation includes

a) Assessment in emergency department

b) Detailed history

c) General medical evaluation

d) Neurological assessment

e) Functional ability assessment

f) Assessment of potential complications

g) Psychological assessment

h) Assessment regarding mobility aids

i) Home, work place and environment modifications.

4.8 NEUROLOGICAL ASSESSMENT IN SCI

International standard examination is used for neurological classification of SCI and it has sensory and motor components along with neurological rectal examination [36, 37,38].

American Spinal Injury Association (ASIA) is used for assessment of acute SCI. Below table describes the ASIA scoring mechanism.

TABLE - 9

ASIA Score definition

Grade	Definition
A	Complete. No sensory or motor function is preserved in the sacral segments S4-S5
B	Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5
C	Incomplete. Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3 (Grades 0-2).
D	Incomplete. Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade greater than or equal to 3
E	Normal. Sensory and motor functions are normal.

4.9 FUNCTIONAL OUTCOME IN SCI

International Classification of Functioning (ICF) developed by World Health Organization (WHO) includes 3 domains such as body functions and structure, activity and participation.

Most pertinent measures for 3 domains of ICF are as follows

a) Body function and structure

   a. International standards for neurological classification of SCI

b) Activity

   a. Functional Independence Measure Score (FIMS) [39]

   b. Spinal Cord Independence Measure

   c. Modified Barthel Index

   d. Quadriplegia Index of Function

   e. Specific Measures of Walking (6 mins walk test, 10 m walk test, timed up and go)

   f. Specific Measures for Upper Extremity Function (GRASS P test).

c) Participation

   a. Craig Handicapped Assessment and Reporting Technique (CHART)

Factors affecting functional outcomes

a) Motor Function

b) Age

c) Co-morbid condition

d) Pain

e) Spasticity

f) Psycho-social and environmental factors

5. Materials and Methods

Study Center

Government Institute of Rehabilitation Medicine,

KK Nagar,

Madras Medical College,

Chennai.

Duration of the Study

March 2017 to September 2018.

Study Design

Cross-sectional study.

Sample Size

100 cases of Spinal Cord Injury

Inclusion Criteria

- Age > 20 Years
- All traumatic case of SCI
- Duration < 1 year
- Traumatic Spinal Cord Injury without any endocrine pathology

Exclusion Criteria

- Known case of Diabetes Mellitus – Type II Diabetes
- Past history of CAD
- Past history of dyslipidemia
- Spinal Cord Lesion – Non traumatic causes
- Other endocrine problems

Methodology

After obtaining informed consent in patient's comfortable language, venous blood samples drawn after an overnight fasting for doing

- Fasting lipid profile,
- HbA1C
- Oral Glucose Tolerance Test

75g of anhydrous glucose is mixed with 250 ml of water and orally administered to patients. In case of patients who have

sensation of vomiting, addition of lemon juice lessens the vomiting sensation.

Recent ADA guideline and National cholesterol education project Adult Treatment Panel – III guidelines are used for diagnosing glucose intolerance and dyslipidemia respectively.

TABLE - 10

ADA Guidelines

FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.*
OR
2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.*
OR
A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.*
OR
In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).
*In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing.
FPG 100 mg/dL (5.6 mmol/L) to 125 mg/dL (6.9 mmol/L) (IFG)
OR
2-h PG during 75-g OGTT 140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L) (IGT)
OR
A1C 5.7–6.4% (39–47 mmol/mol)
*For all three tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at the higher end of the range.

Below table provides the ATP III guidelines for diagnosis of dyslipidemia

LDL Cholesterol – Primary Target of Therapy	
<100	Optimal
100-129	Near optimal/above optimal
130-159	Borderline high
160-189	High
≥190	Very high
Total Cholesterol	
<200	Desirable
200-239	Borderline high
≥240	High
HDL Cholesterol	
<40	Low
≥60	High

TABLE 11

ATP III Guidelines

Risk Category	LDL Goal (mg/dL)	Non-HDL Goal (mg/dL)
CHD and CHD Risk Equivalent (10-year risk for CHD >20%)	<100	<130
Multiple (2+) Risk Factors and 10-year risk ≤20%	<130	<160
0-1 Risk Factor	<160	<190

LDL is calculated using the below formula

LDL = Total Cholesterol - HDL - (Triglycerides/5)

Non-HDL is calculated using below formula

Non-HDL = Total Cholesterol - HDL

Values considered as abnormal

TABLE - 12

Out of bound range for risk parameters

S. No	Parameters	Values
1	Fasting Blood Sugar	< 70 and > 100
2	Post Prandial (2 Hour)	> 140
3	LDL	> 100
4	TGL	> 170
5	HDL	< 40
6	Non HDL	> 130
7	A1C	> 6.5

All these tests were repeated after 3 months of admission.

All biochemical analysis is performed by same laboratory.

Patients considered for this study are categorized using Kuppusamy classification which is as shown below.

TABLE - 13

Kuppusamy Classification

Total score	Socioeconomic class
26–29	Upper class
16–25	Upper middle
11–15	Lower middle
5–10	Upper lower
Below 5	Lower

Rehabilitation Program

All the patients are started on comprehensive rehabilitative program

It includes

- Functional re-education program
    - Rolling, sitting, Balance training in sitting, strengthening of upper limbs for paraplegics, bed transfer and transfer bed to wheel chair and back
- Bracing
- Gait training

Template 1:

Below template shows patients performing rehab cycling.

Template – 2

Below template shows SCI patient doing arm ergometry

Template - 3

Below patient undergoing therapeutic Walking

Template – 4

Balancing exercise

Template – 5

Therapeutic Standing

Analysis

Analysis was done using standard statistical software.

Sponsorship

No

Ethical Issues

Institutional ethical committee approval obtained.

6. ANALYSIS AND RESULTS

6.1 ANALYSIS

Standard statistical software was used to analyze the results. Basic statistical analysis was done in terms of mean, standard deviation, range in terms of minimum and maximum values. Next stage of analysis was performed using Chi square test to test the hypothesis. P values of less than 0.05 was significant and thereby proving the hypothesis to be correct.

Demographic Analysis

Factors considered here are age, sex, marital status, BMI and socio-economic factors.

Lesion Type

Sample population is segregated based on the type of lesion – paraplegia or tetraplegia.

Etiology

Here also the population is categorized based on either Road Traffic Accident (RTA) or Falls.

Neurological Level

Based on the ASIA impairment scale, the population are classified as having either complete (ASIA – A) or incomplete (ASIA – B) injury

Complications

% of occurrence of complications among the study population is analyzed.

6.2 RESULTS

Based on basic statistical analysis, below tables depicts the values.

TABLE - 14

Summary of Population Distribution

		Count	%
Gender	Male	84	84.0%
	Female	16	16.0%
ASIA	A	60	60.0%
	B	40	40.0%
Marital Status	Unmarried	31	31.0%
	Married	69	69.0%
Mode of Injury	Fall	64	65.3%
	RTA	34	34.7%
Lesion	Tetraplegia	34	34.3%
	Paraplegia	65	65.7%
Smoking	Yes	36	36.0%
	No	64	64.0%
Alcohol	Yes	48	48.0%
	No	52	52.0%
Pressure Ulcer	Yes	33	33.0%
	No	67	67.0%
DVT	Yes	6	6.0%
	No	94	94.0%
HO	Yes	3	3.0%
	No	97	97.0%
Family History	Yes	0	0.0%
	No	100	100.0%

TABLE - 15

Basic statistical Analysis of Population

	N	Mean	Std. Deviation	Minimum	Maximum
Age	100	36.210	10.9888	20.0	64.0
FIMS_126	100	60.860	11.8219	44.0	92.0
FBS	100	73.880	13.7314	51.0	128.0
PPBS	100	112.650	33.6339	78.0	284.0
HBA1C	100	5.385	.7277	4.5	8.7
MBG	100	113.194	23.4280	83.0	232.0
Total Cholesterol	100	195.870	29.6350	136.0	277.0
TGL	100	174.480	41.7280	101.0	303.0
HDL	100	46.120	4.3094	34.0	55.0
LDL	100	113.940	27.4928	56.0	190.0
VLDL	100	35.220	8.6860	20.0	61.0
Non HDL	100	147.700	34.5924	46.0	239.0

Age Distribution

Below table provides the summary of the population distribution according to the age.

TABLE - 16

Age Distribution

S.No	Age Range	Count
1	20 – 40	74
2	41 – 60	22
3	61 – 80	4

This data is represented in the pie chart

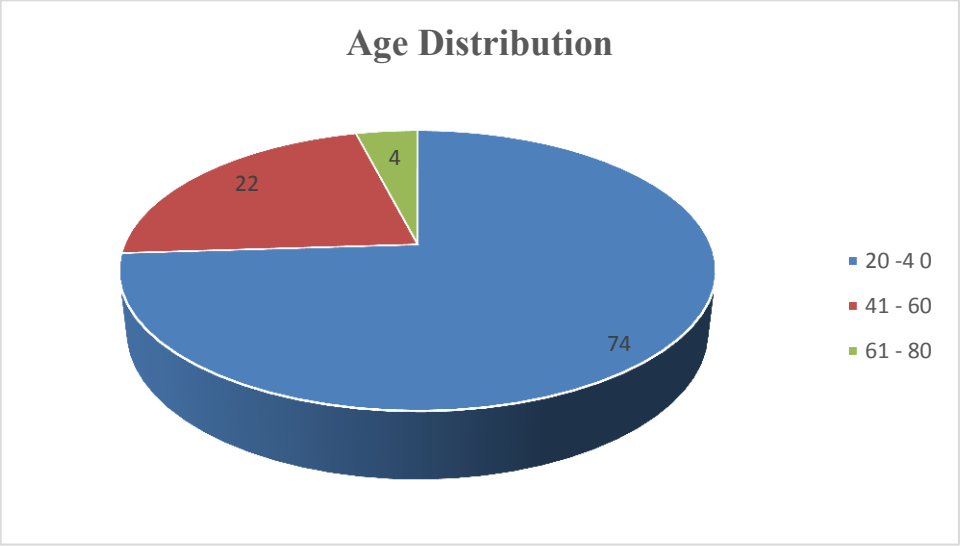


Chart 1: Age Distribution

Average age of the impacted population is around 39 years.

Gender Distribution

Below table shows the gender distribution

TABLE - 17

Gender Distribution

Gender	Count
Male	84
Female	16

Males are predominantly affected as seen from the analysis.

Below is the pictorial distribution of gender.

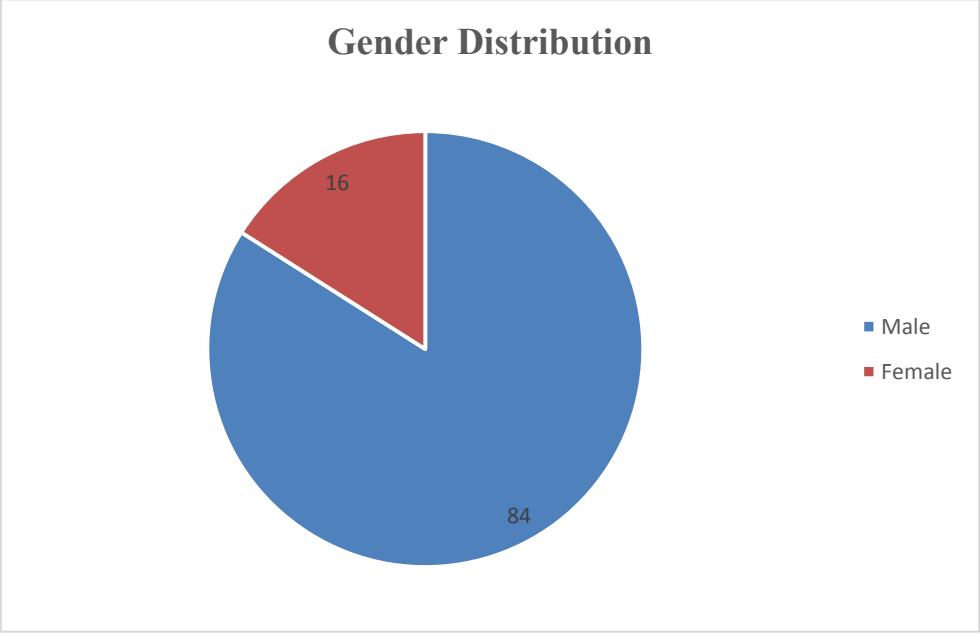


Chart 2: Gender Distribution

Marital Status

Among the total population 69% are married signifying the socio-economic impact and same is shown below.

Below table shows the marital population distribution

TABLE -18

Marital Status distribution

Marital Status	Count
Married	69
Unmarried	31

Chart depicting the marital status distribution

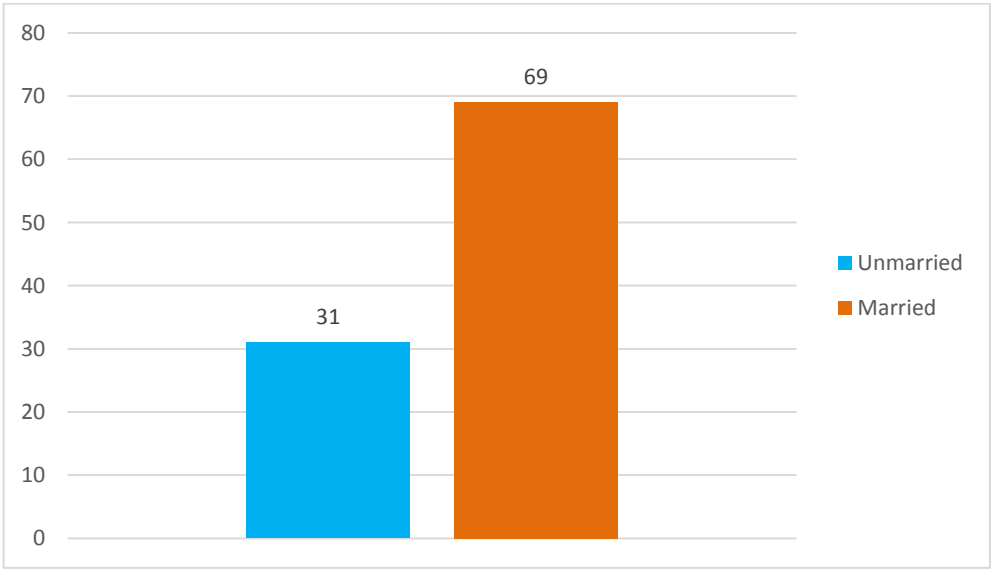


Chart 3: Marital Status

Lesion Type

Paraplegia (65%) is more common among the study population than Tetraplegia (35%).

Table showing the lesion distribution

TABLE - 19

Lesion type distribution

Lesion Type	Count
Tetraplegia	35
Paraplegia	65

Below chart depicts the lesion distribution

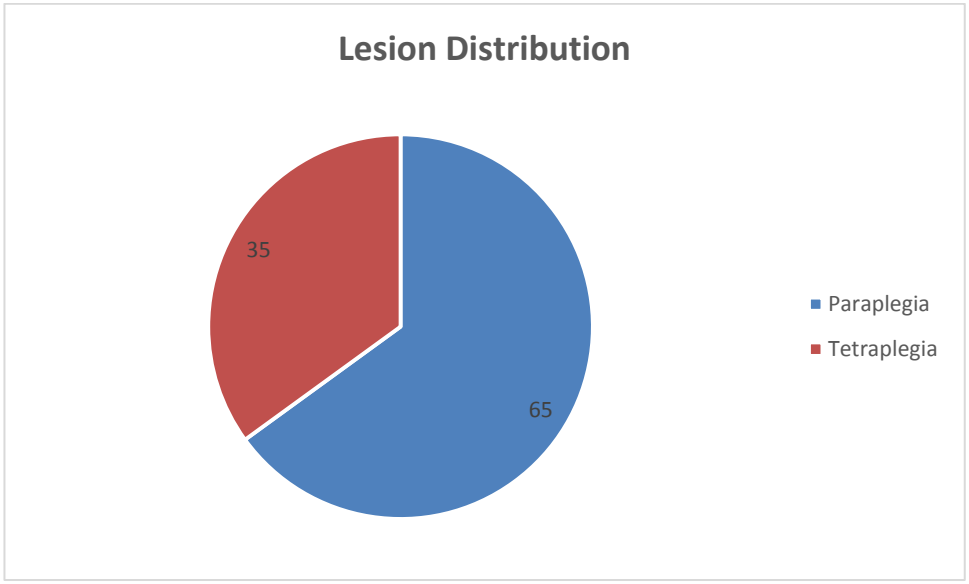


Chart 4: Lesion type

Traumatic Etiology

Falls is more common mode of injury (65%) when compared to RTA (35%).

Table depicts the etiology distribution

TABLE - 20

Etiology Distribution

Etiology Type	Count
Fall	65
RTA	35

Below chart provides the distribution of etiology among the sample population

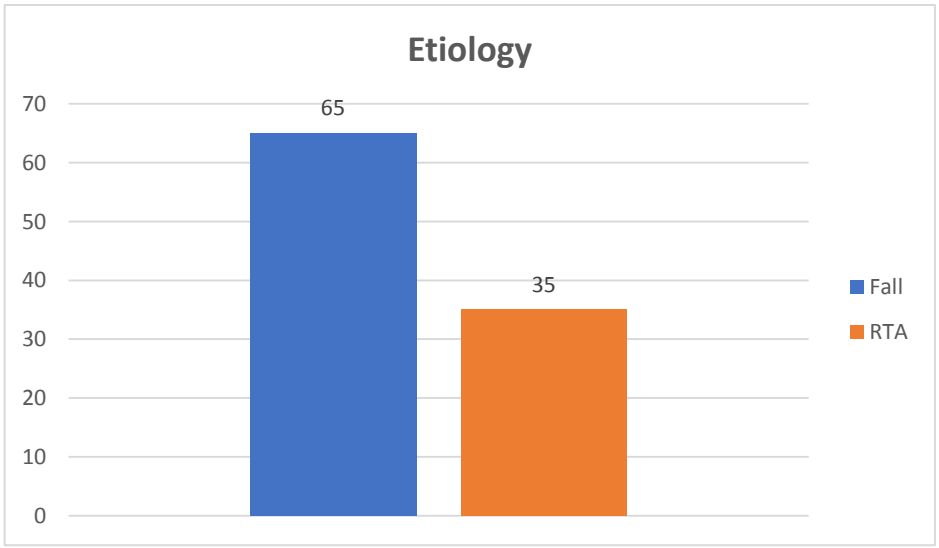


Chart 5: Etiology Distribution

Neurological Level

Complete injury (ASIA – A) more common than incomplete injury (ASIA – B)

Neurological Level	Count
ASIA A	40
ASIA B	60

TABLE - 21

Neurological Level

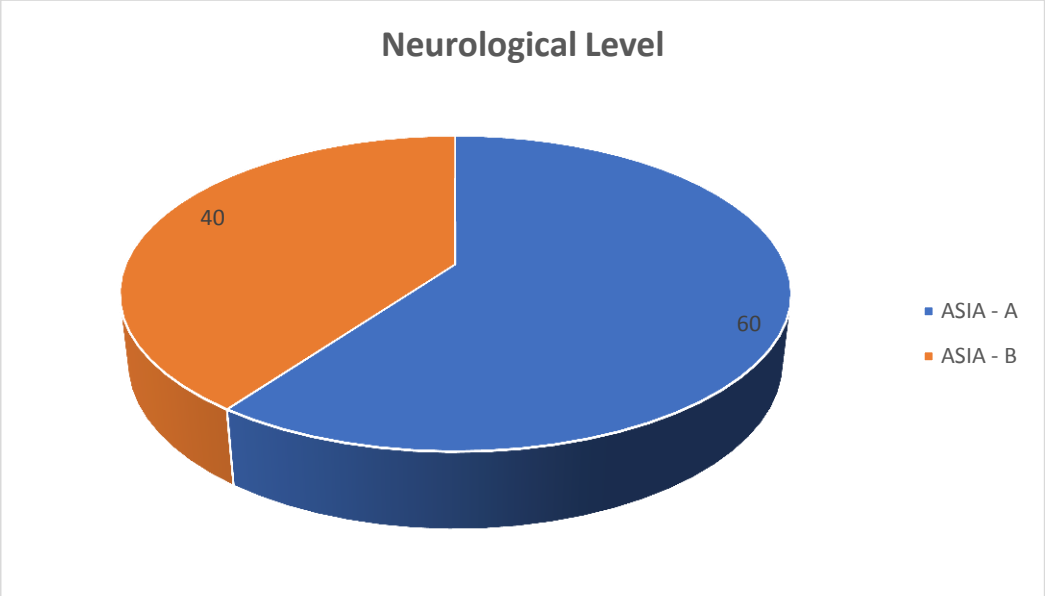


Chart 6: Neurological Level Distribution

Risk Factors

Dyslipidemia and hypo and hyper glycemia enhancing the cardio vascular risk.

Below table summarizes the overall risk factors among the demographic population.

TABLE - 22

Metabolic variable among gender

		Gender			
		Male		Female	
		Count	%	Count	%
GLYCEMIA_FBS	HYPOGLYCEMIA	40	88.9%	5	11.1%
	NORMAL	44	80.0%	11	20.0%
GLYCEMIA_HBA1C	HbA1C > 6.5	3	75.0%	1	25.0%
	HbA1C < 6.5	81	84.4%	15	15.6%
GLYCEMIA_PPBS	HYPERGLYCEMIA	14	73.7%	5	26.3%
	NORMAL	70	86.4%	11	13.6%
DYSLIPIDEMIA	YES	68	85.0%	12	15.0%
	NO	16	80.0%	4	20.0%

When further analyzed the risk factors with respect to dyslipidemia, below table shows the distribution of abnormal values for the risk factors considered.

TABLE - 23

Risk factor Distribution

	FBS	PPBS	LDL	Non-HDL	TGL
Male	40	14	55	61	43
Female	5	5	10	9	5

Same is depicted pictorially to show the distribution of risk factors among the gender.

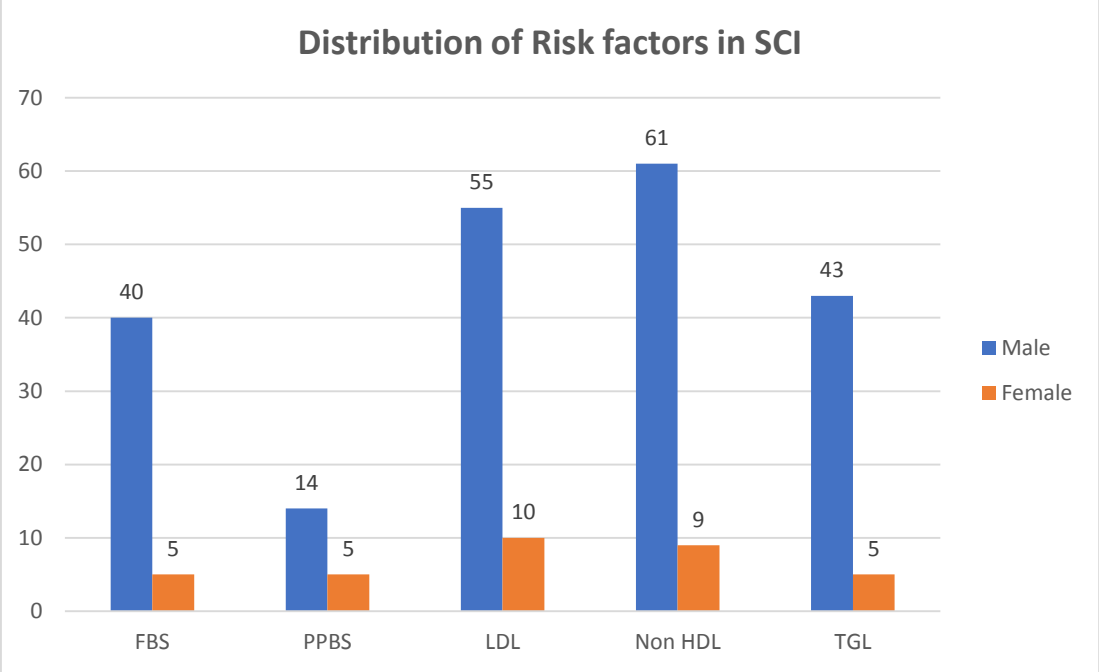


Chart 7: Distribution of Risk Factors in SCI

Analysis was also performed for prevalence of risk factors among complete and incomplete injury and same is shown in the below table.

TABLE - 24

Risk factors distribution Vs Neurological Level

		ASIA			
		A		B	
		Count	%	Count	%
GLYCEMIA_FBS	HYPOGLYCEMIA	28	62.2%	17	37.8%
	NORMAL	32	58.2%	23	41.8%
GLYCEMIA_HbA1C	HbA1C > 6.5	1	25.0%	3	75.0%
	HbA1C < 6.5	59	61.5%	37	38.5%
GLYCEMIA_PPBS	HYPERGLYCEMIA	10	52.6%	9	47.4%
	NORMAL	50	61.7%	31	38.3%
DYSLIPIDEMIA	YES	47	58.8%	33	41.3%
	NO	13	65.0%	7	35.0%

Risk factors are more prevalent in complete injury Further analysis of risk factors was undertaken with respect to lesion type and below is the table for this distribution.

TABLE - 25

Risk factors distribution Vs Lesion Type

		Lesion			
		Quadriplegia		Paraplegia	
		Count	%	Count	%
GLYCEMIA_FBS	HYPOGLYCEMIA	15	34.1%	29	65.9%
	NORMAL	19	34.5%	36	65.5%
GLYCEMIA_HbA1C	HbA1C > 6.5	3	75.0%	1	25.0%
	HbA1C < 6.5	31	32.6%	64	67.4%
GLYCEMIA_PPBS	HYPERGLYCEMIA	10	52.6%	9	47.4%
	NORMAL	24	30.0%	56	70.0%
DYSLIPIDEMIA	YES	26	32.9%	53	67.1%
	NO	8	40.0%	12	60.0%

Below table 26 and 27 shows the hypothesis that hypo glycemia, hyper glycemia and dyslipidemia are having significant impact on cardiovascular risk.

TABLE - 26

Hypothesis – 1

	Hypoglycemia	**Hyperglycemia**	**Marginal Row Total**
Abnormal	44	18	62
Normal	56	82	138
Marginal Column Total	100	100	200

Analysis was performed on sample population and it showed that carbohydrate and lipid abnormality had strong association with cardio vascular risk.

The p value was found to be significant and less than 0.05 at 95% CI

TABLE - 27

Hypothesis – 2

	BS Abnormal	**Dyslipidemia**	**Marginal Row Total**
Abnormal	62	80	142
Normal	38	20	58
Marginal Column Total	100	100	200

TABLE - 28

Hypothesis– 3

	FBS	PPBS	LDL	Non HDL	TGL	Row Total
Abnormal	44	18	65	70	48	245
Normal	64	82	35	30	52	263

7. DISCUSSION

Based on the analysis, mean age of the population is 36 but among impacted population the median age is 39 years. As per the various studies [40], it is found out that the incidence of SCI is more prevalent in young, active reproductive age group.

Also, from the study it is found that the males are more commonly affected (84%) than female (16%) as the males are causal labors who are involved in high risk job. Also, average Body Mass Index is found between 19 and 24.

Most of the individual are belonging to the low socio-economic group and they are the sole bread winners of the family.

Considering the traumatic etiology fall from height is the predominant mode of injury. This implies the lack of fall preventive measures while involving themselves in high risk jobs. Paraplegia is the most common lesion type among the study population.

Analyzing the complication of SCI, it is found that the 19% of individual found to have hyper glycemia, which is in line with other studies published [41,42]. At the same time, 45% of individual had hypo glycemia in this study which is not seen in previous studies. This can be attributed to malnutrition as the study population belonging to low socio-economic status and it may be due to deficiency of anabolic hormones like growth hormone and

testosterone or may be due to underlying infection. Not only hyper glycemia but also hypo glycemia has adverse effects on cardio vascular system.

Below picture shows impact of hypo glycemia on CVS

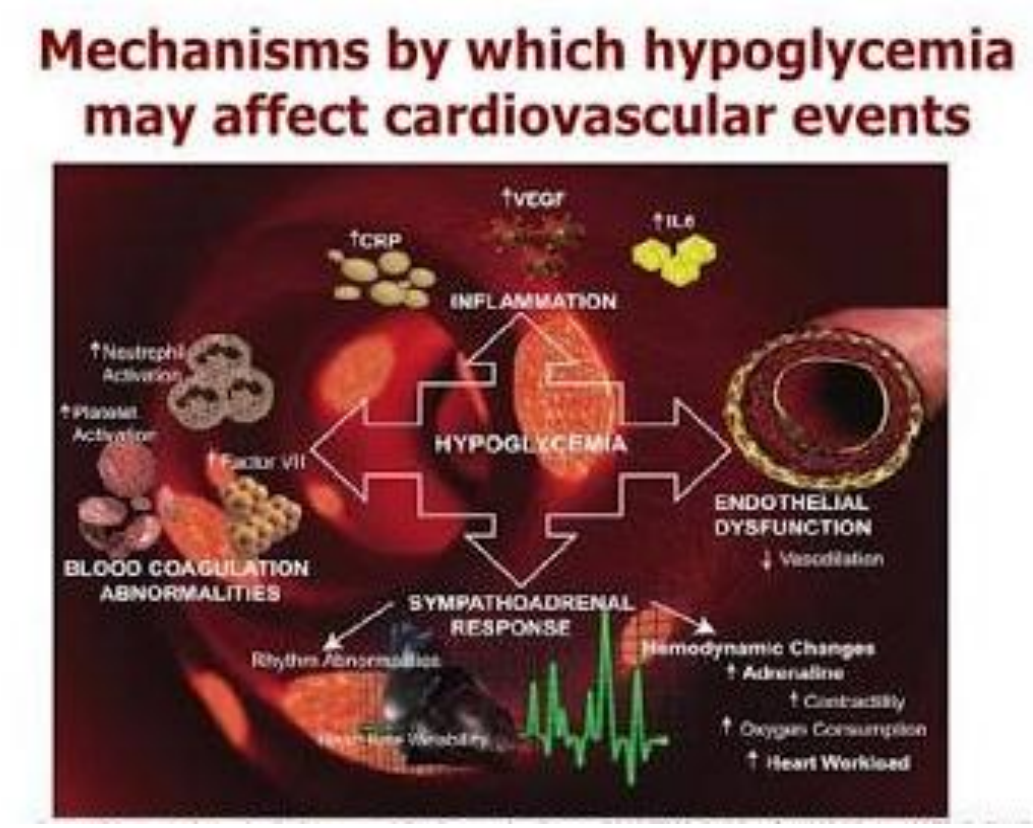


Figure 16: Effects of Hypoglycemia on CVS

Observed hyper glycemia in the study population is due to insulin resistance, sedentary life style and loss of lean body mass.

80% of individual had dyslipidemia like elevated LDL, elevated TGL and Non-HDL. This may be because of increased adiposity. In this study, only 6% had low HDL and it may be attributed to the nature of work prior to injury. Non-HDL is included in this study as one of the risk factors since it is found to

be an emergent risk factor [43] for CAD. Six individuals developed Deep Vein Thrombosis (DVT) and 2 died due to CAD.

After the institution of Comprehensive rehabilitation program, blood sugar values repeated after 3 months found to be in near normal range for those individuals with abnormal values found in the first sample after the admission.

8. CONCLUSION

As per the study, dyslipidemia and both hypo and hyper glycemia increases the cardiovascular risk in cases of spinal cord injury by accelerating the atherosclerosis.

Study Showed BS and lipid abnormalities acts as multiplier effect for cardiovascular risk.

RECOMMENDATIONS

1. Periodic monitoring of blood sugar and lipid parameters along with other risk factors.
2. Appropriate Medical Management & life style modifications.
3. Institution of early rehabilitation measures.

9. LIMITATION

Below is some of the limitation of the study

a) Single institution-based study.

b) Study population belonging to low socio-economic status

c) Focusing on few risk factors

d) Small population

e) No age matched control group

f) Short duration of the study

10. FUTURE SCOPE

This study can be enhanced by considering,

- Extending the study to wider geographic area
- Multi-center study
- Longer duration of study period
- Including all socio-economic group
- Including population in different occupational category
- Age matched control
- Additional risk factors like fasting insulin level, HS – CRP, serum troponin level.

11. REFERENCES

1. Castelli WP, Lead A, Identification and assessment of cardiac risk an overview, Cardiol Clin 1985; 3:171-178.

2. William H. Donovan, MD, J.Spinal Cord Med. 2007;30 (2:85-100).

3. M.D.Schiller, R. J. Mobbs, Historical Evolution of Management of Spinal Cord Injury, https://doi.org/10.1016/j.jocn.2012.03.002.

4. Gray's Anatomy for Students, III edition, 2013.

5. Kang Y, Ding H, Zhou HX, Weiz J,Li UL, Pan D Y, The epidemiology of traumatic spinal cord injury in British, Colombia, Canada. Spine (Phla Pa 1976). 2012; 37(4) 321 – 329.

6. Spinal Cord Injury: Facts and Figures at a glance, NSCISC, 2018 SCI data sheet.

7. Epidemiology of Traumatic SCI in Asia: Systematic review, J. Spinal Cord Med. 2012; 35(4) 229 – 239.

8. Braddom's Physical Medicine & Rehabilitation, V edition, 2016.

9. Kwon BK, Tetzlaff W, Grauer JN, Beiner J, Vaccaro AR, Pathophysiology and Pharmacologic treatment of acute spinal cord injury, Spine J. July – August 2004;4(4):451-464.

10. Semin Vet, The pathophysiology of spinal cord injury and its clinical implications, Medsurg (small Anom). 1996 Nov;11(4) 201 – 207.

11. Bauman WA, Korsten MA, Radulovic M, Schilero GJ, Secondary medical consequences of Spinal Cord Injury. Top Spinal Cord Inj. Rehabil. 2012;18(4): 355 – 378 .

12. Bauman WA, Spungen AM, Adkins RH, Kemp BJ, Metabolic and Endocrine changes in persons aging with Spinal Cord Injury. Assist Technol. 2001;11:88 – 96.

13. Mestrl H, Alkon T, Salazar S, Ibarra. Spinal Cord Injury Sequelae alter drug pharmaco kinectics:: An overview. Spinal Cord. 2011;49(9): 955 – 960 .

14. Bauman WA, Spungen AM, Metabolic changes in person after Spinal Cord Injury. Phys Med Rehab Clin North AM. 2000; 11: 102 – 140 .

15. Atlanta, GA American Spinal Cord Association. International Standards to document remaining autonomic function after Spinal Cord Injury (ISAFSCI). 2012.

16. Franga DL, Hawkins ML, Medeiros RS, Adewumi D. Recurrent asystole resulting from high cervical Spinal Cord Injury. Amsurg. 2006;72(6); 525 – 529 .

17. Ellen Merete Hagen, Tiina Rekand, Marit Gronning, Cardiovascular Complications of Spinal Cord Injury 15 May, 2012.

18. Consortium for Spinal Cord Medicine. Acute management of autonomic dysreflexia: Individuals with Spinal Cord Injury Presenting to Health care facilities. 2nd Ed. Washington DC: Paralysed Veterans of America; 2001.

19. Sabharwal S. Cardiovascular dysfunction in Spinal Cord Disorders. In: Linv. Ed. Spinal Cord Medicine. 2nd Ed. New York NY: Demos Publishing: 241 – 255 .

20. Bauman WA, Spungen AM. Coronary Heart Disease in individuals with Spinal Cord Injury: Assessment of risk factors. Spinal Cord. 2008;46(7):466 – 476 .

21. Teasell RW, Arnold MO, Karassioukov A, et. Al. Cardio vascular consequences of loss of supra spinal control of sympathetic nervous system after Spinal Cord Injury. Arch Phys Med Rehabil. 2008; 81(4): 506 – 516.

22. Carison KF, Wilt TJ, Taylor BC, et. Al. Effect of exercise on disorders of carbohydrates and lipid metabolism in adults with Traumatic Spinal Cord Injury; systematic review of the evidence. J Spinal Cord Med. 2009;32(4): 361 – 378 .

23. Wrong SL, Shemk, Crew J. specialized respiratory management for acute cervical spinal cord injury: a retrospective analysis. Top spinal cord inj. Rehabil. 2012 ; 18(4) : 283-290.

24. Langton G.E, Buchholz AC, Martin Ginis KA. Lowering body mass index cutoffs better identifies obese persons with spinal cord injury spinal cord 2009 ; 47(10) : 757-762.

25. Gater DR. obesity after spinal cord injury. Phys Med Rehabil clin North Am. 2007 ; 18(2) : 333 – 351.

26. Dhallss, Hadley MN, Aarabi B, et al., Deep venous thrombosis and thromboembolism in patients with cervical spinal cord injuries. Neurosurgery, 2013 ; 72 (suppl. 2) : 244 – 254.

27. Price MJ. Thermoregulation during exercise in individuals with spinal cord injuries. Sports med. 2006; 36(10) : 863 – 879.

28. Ebert E. Gastrointestinal Involvement in Spinal Cord injury; a Clinical perspective. J.Gastrointestin liver Dis.2012; 21(1) : 75-82.

29. SamsonG, Cardenas DD. Neurogenic Bladder in Spinal cord injury. Physmed Rehabil Clin N Am.2007; 18(2): 255-274.

30. D’ ttondt F, Everaert K.Urinary tract infections in patients with spinal cord injuries, Curr Infect Dis, Rep.2011;13(6): 544-551.

31. Reddy M, Gillss, Kalkar SR, WuW, Anderson PJ. Rochon PA. Treatment of Pressure Ulcer: a Systematic review. Jama, 2008; 300 (22): (2647-2662).

32. Elliott SL. Problems of Sexual function after Spinal Cord Injury. Prog Brain Res.2006; 152: 387-399.

33. Goldstein B. Musculoskeletal Conditions after Spinal Cord Injury. Phys Med Rehabil Clin N.A.M.2000; 11(1) : 91-108.

34. Finnerup NB, Baestrop C. Spinal Cord Injury Pain: Mechanism and Management. Curr Pain Headache Rep.2012:16(3):207-216.

35. Charmetant C, Praner V, Condemine A, Calmels, P. Diagnosis and Treatment of Osteoporosis in Spinal Cord Injury Patients : a literature review. Ann Phys Rehabil Med. 2010; 53(10): 655-668.

36. Zariffa J, Kramer JL, Jones LA, et al., Sacral Sparing in SCI: beyong the S4-S5 and anorectal examination. Spine J. 2012;12(5) 389-400.

37. Van Middendrop JJ, Hosman AI, Donders Ar, et al., A. Clinical Prediction rule for ambulation outcomes after traumatic spinal cord injury: a longitudinal cohort study. Lancet.March 19, 2011:377 (9770) :1004-1010.

38. American Spinal Association: International Standards for Neurological and Functional Classification of spinal cord injury, revised 2011, Atlanta GA.

39. Coaters RL; Adkins R, YakuraJ, Siei.Donal Munro Lecture: Functional and neurological recovery following acute SCI.J. Spinal Cord Med. 1998; 21(3) :195-199).

40. Epidemiology of Worldwide Spinal Cord Injury Kurtzke JF. Neurol pisquiat, 1977.

41. Duckworth WC, Jallepalli, Solomon SS, Glucose Intolerance in Spinal Cord Injury, Arch Phys Med Rehabil, 1983 Mar, 64(3), 107-10.

42. V. Bhuvshtein, A. D. Korczyn I. Pinhas et al, Insulin Resistance in tetraplegia but not in mid thoracic paraplegia: Is the mid-thoracic spinal cord involved in glucose regulation?, Spinal Cord (2011) 49, 648-652.

43. Jiang R. Schulze MB, LiJ, Rifain, S, Non – HDL Cholesterol and Apo lipoprotein B, Predict Cardiovascular diseases among men with Type 2 Diabetics.

44. Adiponectin :a Key adipocytokinin in MS Yoshimosa, Okamotu, Tohru, Yujimassuzawa, Peter libby Clinical Evidence Feb.10, 2006, 110 (13) 267-278.

45. Consortium for Spinal Cord Medicine Clinical practice Guideline, Depression following Spinal Cord Injury: a clinical practice guidline for primary care physician J Spinal Cord Med: 200124 (Suppl. 1) (S40-S101).

ANNEXURES

A1 - MASTER DATA

SAMPLE - 1

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
1	Mani	60	M	89	166	6.1	140	178	141	53	97	28	125
2	Masilamani	40	M	62	117	5.6	122	202	184	45	120	37	157
3	Ranjith	22	M	64	91	4.9	97	211	130	49	136	26	162
4	Sivakumar	40	M	69	101	5.3	111	259	173	48	177	34	211
5	Amul Raj	23	M	51	89	4.6	87	178	201	50	88	40	128
6	Veeran	32	M	75	108	4.9	97	215	179	49	130	36	166
7	Srinivasan	23	M	60	88	4.7	90	200	128	51	123	26	149
8	Jayaraman	35	M	65	104	5.1	104	218	153	49	139	30	169
9	Arumugam	60	M	77	158	8.3	104	218	153	49	139	30	189
10	Venu	39	M	83	101	5	101	202	141	49	125	28	153
11	Samikannu	37	M	69	109	4.9	97	231	159	47	152	32	184
12	Chinna Durai	35	M	72	98	5.2	108	181	147	46	106	29	135
13	Murugesan	51	M	76	101	5.8	130	207	141	48	131	28	159
14	Vinod	22	M	78	106	5	101	178	155	50	97	31	128
15	Ammachi	39	F	77	95	5.2	108	173	122	55	94	24	118
16	Abhirama Sundari	23	F	76	99	5.2	108	173	122	55	94	24	118
17	Selvi	33	F	71	148	6.1	140	255	159	44	179	58	211

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
18	Ravi	35	M	128	284	8.4	221	189	266	43	93	53	146
19	Rajendran	36	M	66	83	5.2	107	194	168	49	111	34	145
20	Priya	23	F	75	93	5.2	107	191	188	43	110	38	148
21	Anthony Raj	30	M	89	145	6.2	136	213	286	38	118	57	175
22	Ponnusamy	62	M	84	157	6.8	164	175	188	45	92	38	130
23	Prem Raj	44	M	92	140	6.1	140	224	205	40	143	41	184
24	Meganathan	26	M	79	101	5.4	115	172	163	50	89	33	122
25	Gnanavel	38	M	57	126	5.6	122	188	303	34	93	61	154
26	Maya Kannan	28	M	73	96	5	100.7	144	183	52	56	36	92
27	Sasi kumar	22	M	65	134	5.6	122	184	177	45	104	35	139
28	Venkatesan	45	M	52	109	5.6	122	219	198	44	136	39	175
29	Hariharan	38	M	68	85	5.1	104	241	206	42	158	41	199
30	Mani	56	M	56	95	5.4	114	235	219	40	151	44	195
31	Nagappan	63	M	55	81	5.1	104	277	247	38	190	49	239
32	SivaLingam	60	M	62	99	5.6	122	207	193	42	127	38	165
33	Krishna Kumar	28	M	79	91	5.1	104	246	133	47	173	26	199
34	Nagappan	61	M	74	131	5.7	125	202	131	48	128	26	154
35	Charan	20	M	71	113	4.8	94	195	141	49	118	28	146
36	Kannan	32	M	63	101	5	100.7	192	101	51	121	20	141
37	Prasanth	25	M	68	85	4.9	97	188	119	52	77	59	136
38	Velu	62	M	67	131	6.1	140	267	124	48	134	25	219
39	Sampath	47	M	76	126	6	136	242	161	46	164	32	196
40	Selvi	35	F	67	120	4.9	97	176	127	48	103	25	128

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
41	Arul	29	M	73	97	5.1	104	208	193	47	123	38	161
42	Manikantan	25	M	51	88	4.9	97	173	166	50	90	33	123
43	Kannadasan	45	M	68	82	5.2	107	191	186	48	106	37	143
44	Chitra	28	F	75	142	5.6	122	169	155	47	91	31	122
45	Kasturi	40	F	85	165	6.4	150	221	262	41	128	52	180
46	Madu	28	M	61	96	4.9	97	166	221	44	78	44	122
47	Kumaran	43	M	69	84	5	100.7	225	258	41	133	51	184
48	Sekar	43	M	78	101	4.9	94	145	119	53	68	24	92
49	Srinivasan	33	M	61	88	4.8	94	158	129	51	81	26	107
50	Damodaran	38	M	98	180	6.1	140	184	193	47	99	38	137
51	Nagarajan	29	M	81	138	5.8	129	221	196	48	134	39	173
52	Sathish	35	M	54	88	5.1	104	185	142	47	110	28	138
53	Yasodha Krishnan	36	M	81	116	5.6	122	265	162	48	125	32	217
54	Sesuraj	34	M	69	91	5	100	221	193	45	138	38	176
55	Prabhakaran	26	M	69	81	4.5	83	142	169	48	60	34	94
56	Sudhakaran	25	M	69	97	5	101	155	171	45	76	34	110
57	Parathasarathy	25	M	79	94	4.9	97	188	165	46	109	33	142
58	Murugan	30	M	83	117	5.6	122	183	196	44	100	39	139
59	Sayeed Ibrahim	28	M	59	80	4.9	97	136	122	50	62	24	86
60	Kiran	20	M	57	78	4.6	91	189	144	50	110	29	139
61	Kanaga sabapathy	26	M	65	84	4.9	97	225	178	47	143	35	178
62	Ramesh	30	M	66	94	5.1	104	196	192	46	112	38	150
63	Prabhu	35	M	66	101	5.3	111	199	204	44	114	41	155

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
64	Tirupathi	27	M	62	89	4.5	83	165	189	45	82	38	120
65	Ravi	45	M	55	89	5	101	203	210	48	113	42	155
66	Saravanan	30	M	69	87	4.9	97	202	165	51	118	33	151
67	Raghupathy	40	M	85	158	5.9		197	185	48	112	37	149
68	Raja	35	M	67	92	4.9	97	241	181	45	160	36	196
69	Lakshmi	32	F	71	98	4.8	94	221	175	47	139	35	174
70	Babu	25	M	76	99	5.1	115	160	151	49	81	30	111
71	Moovendan	29	M	79	164	5.4	115	164	176	47	82	35	117
72	Sakthivel	27	M	56	81	4.6	87	157	135	47	83	27	110
73	Karthik	23	M	82	109	5.1	104	166	112	48	96	22	118
74	Rahmat Nisha	44	F	73	91	5.1	104	166	151	45	91	30	121
75	Manimegalai	40	F	69	109	5.4	115	171	135	46	98	27	125
76	Yuvarani	23	F	69	103	5.3	115	183	105	49	113	21	134
77	Balamurugan	31	M	70	98	5	101	141	105	52	68	21	89
78	Magesh	24	M	71	92	4.9	97	144	136	50	67	27	94
79	Vijayan	45	M	86	103	5.2	108	161	159	47	82	32	114
80	Mohan	39	M	64	87	4.9	97	148	129	48	74	26	100
81	Velayudham	35	M	77	92	5.1	104	183	142	51	114	28	132
82	Shanti	42	F	85	165	6.4	150	221	262	41	128	52	180
83	Kumaravel	40	M	69	84	5	100.7	225	258	41	133	51	184
84	Rajendran	35	M	73	106	5.3	111	191	154	44	116	31	147
85	Gopi	33	M	78	91	5.3	111	195	186	50	108	37	145

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
86	Vedamma	60	F	88	211	8.7	232	231	196	48	144	39	183
87	Vadivel	42	M	106	148	6.1		212	178	40	137	35	172
88	Venkatesan	45	M	75	107	5	100	164	149	52	82	30	112
89	Venkatesan	35	M	61	89	4.9	97.7	183	196	45	99	38	138
90	Mythili	35	F	85	112	5.2	107.8	195	138	50	117	28	145
91	Anthony	42	M	82	146	6.4	150	223	191	47	138	38	176
92	Kumar	38	M	79	93	4.9	97.1	201	226	48	108	45	153
93	Anbalagan	47	M	102	189	6.2	149	208	250	35	135	40	173
94	Lenin	38	M	124	200	6.1	145	201	230	35	138	38	166
95	Chidambaram	42	M	82	112	5.3	116	195	186	40	108	37	155
96	Durai	26	M	73	106	5.1	109	225	258	41	103	51	184
97	Pongavanam	40	F	69	84	5	105	191	154	38	116	31	153
98	Vasantha kumar	40	M	116	158	6	142	198	170	35	138	35	163
99	Kannan	45	M	85	120	5.4	120	180	150	40	135	30	140
100	Pugalendhi	22	M	78	116	5.2	113	190	170	42	138	35	148

SAMPLE - 2

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
1	Mani	60	M	89	136	6.1	140	178	141	53	97	28	125
2	Masilamani	40	M	51	89	4.6	87	178	201	50	88	40	128
3	Ranjith	22	M	69	101	4.9	97	211	130	49	136	26	162
4	Sivakumar	40	M	64	99	5.3	111	259	173	48	177	34	211
5	Amul Raj	23	M	51	89	4.6	87	178	201	50	88	40	128
6	Veeran	32	M	75	108	4.9	97	215	179	49	130	36	166
7	Srinivasan	23	M	65	104	5.6	104	202	184	45	120	37	157
8	Jayaraman	35	M	62	117	5.1	122	218	153	49	139	30	169
9	Arumugam	60	M	77	138	6.3	122	218	153	49	139	30	189
10	Venu	39	M	83	101	5	101	202	141	49	125	28	153
11	Samikannu	37	M	69	109	4.9	97	231	159	47	152	32	184
12	Chinna Durai	35	M	72	98	5.2	108	181	147	46	106	29	135
13	Murugesan	51	M	76	101	5.8	130	207	141	48	131	28	159
14	Vinod	22	M	78	106	5	101	178	155	50	97	31	128
15	Ammachi	39	F	77	95	5.2	108	173	122	55	94	24	118
16	Abhirama Sundari	23	F	76	99	5.2	108	173	122	55	94	24	118
17	Selvi	33	F	71	128	6	140	255	159	44	179	58	211
18	Ravi	35	M	108	180	7.2	221	189	266	43	93	53	146
19	Rajendran	36	M	66	83	5.2	107	194	168	49	111	34	145

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
20	Priya	23	F	75	93	5.2	107	191	188	43	110	38	148
21	Anthony Raj	30	M	89	135	6.1	136	213	286	38	118	57	175
22	Ponnusamy	62	M	84	130	6.2	164	175	188	45	92	38	130
23	Prem Raj	44	M	92	140	6.1	140	224	205	40	143	41	184
24	Meganathan	26	M	79	101	5.4	115	172	163	50	89	33	122
25	Gnanavel	38	M	57	126	5.6	122	188	303	34	93	61	154
26	Maya Kannan	28	M	73	96	5	100.7	144	183	52	56	36	92
27	Sasi kumar	22	M	65	134	5.6	122	184	177	45	104	35	139
28	Venkatesan	45	M	52	109	5.6	122	219	198	44	136	39	175
29	Hariharan	38	M	68	85	5.1	104	241	206	42	158	41	199
30	Mani	56	M	56	95	5.4	114	235	219	40	151	44	195
31	Nagappan	63	M	55	81	5.1	104	277	247	38	190	49	239
32	SivaLingam	60	M	62	99	5.6	122	207	193	42	127	38	165
33	Krishna Kumar	28	M	79	91	5.1	104	246	133	47	173	26	199
34	Nagappan	61	M	74	131	5.7	125	202	131	48	128	26	154
35	Charan	20	M	71	113	4.8	94	195	141	49	118	28	146
36	Kannan	32	M	63	101	5	100.7	192	101	51	121	20	141
37	Prasanth	25	M	68	85	4.9	97	188	119	52	77	59	136
38	Velu	62	M	67	131	6.1	140	267	124	48	134	25	219
39	Sampath	47	M	76	126	6	136	242	161	46	164	32	196
40	Selvi	35	F	67	120	4.9	97	176	127	48	103	25	128

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
41	Arul	29	M	73	97	5.1	104	208	193	47	123	38	161
42	Manikantan	25	M	51	88	4.9	97	173	166	50	90	33	123
43	Kannadasan	45	M	68	82	5.2	107	191	186	48	106	37	143
44	Chitra	28	F	75	120	5.6	122	169	155	47	91	31	122
45	Kasturi	40	F	85	135	6	150	221	262	41	128	52	180
46	Madu	28	M	61	96	4.9	97	166	221	44	78	44	122
47	Kumaran	43	M	69	84	5	100.7	225	258	41	133	51	184
48	Sekar	43	M	78	101	4.9	94	145	119	53	68	24	92
49	Srinivasan	33	M	61	88	4.8	94	158	129	51	81	26	107
50	Damodaran	38	M	98	140	6.1	140	184	193	47	99	38	137
51	Nagarajan	29	M	81	138	5.8	129	221	196	48	134	39	173
52	Sathish	35	M	54	88	5.1	104	185	142	47	110	28	138
53	Yasodha Krishnan	36	M	81	116	5.6	122	265	162	48	125	32	217
54	Sesuraj	34	M	69	91	5	100	221	193	45	138	38	176
55	Prabhakaran	26	M	69	81	4.5	83	142	169	48	60	34	94
56	Sudhakaran	25	M	69	97	5	101	155	171	45	76	34	110
57	Parathasarathy	25	M	79	94	4.9	97	188	165	46	109	33	142
58	Murugan	30	M	83	117	5.6	122	183	196	44	100	39	139
59	Sayeed Ibrahim	28	M	59	80	4.9	97	136	122	50	62	24	86
60	Kiran	20	M	57	78	4.6	91	189	144	50	110	29	139
61	Kanaga sabapathy	26	M	65	84	4.9	97	225	178	47	143	35	178

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
62	Ramesh	30	M	66	94	5.1	104	196	192	46	112	38	150
63	Prabhu	35	M	66	101	5.3	111	199	204	44	114	41	155
64	Tirupathi	27	M	62	89	4.5	83	165	189	45	82	38	120
65	Ravi	45	M	55	89	5	101	203	210	48	113	42	155
66	Saravanan	30	M	69	87	4.9	97	202	165	51	118	33	151
67	Raghupathy	40	M	85	130	5.9		197	185	48	112	37	149
68	Raja	35	M	67	92	4.9	97	241	181	45	160	36	196
69	Lakshmi	32	F	71	98	4.8	94	221	175	47	139	35	174
70	Babu	25	M	76	99	5.1	115	160	151	49	81	30	111
71	Moovendan	29	M	79	134	5.4	115	164	176	47	82	35	117
72	Sakthivel	27	M	56	81	4.6	87	157	135	47	83	27	110
73	Karthik	23	M	82	109	5.1	104	166	112	48	96	22	118
74	Rahmat Nisha	44	F	73	91	5.1	104	166	151	45	91	30	121
75	Manimegalai	40	F	69	109	5.4	115	171	135	46	98	27	125
76	Yuvarani	23	F	69	103	5.3	115	183	105	49	113	21	134
77	Balamurugan	31	M	70	98	5	101	141	105	52	68	21	89
78	Magesh	24	M	71	92	4.9	97	144	136	50	67	27	94
79	Vijayan	45	M	86	103	5.2	108	161	159	47	82	32	114
80	Mohan	39	M	64	87	4.9	97	148	129	48	74	26	100
81	Velayudham	35	M	77	92	5.1	104	183	142	51	114	28	132
82	Shanti	42	F	85	125	6	150	221	262	41	128	52	180

S.No	Name	Age	Gender	OGTT		HBA1C	MBG	Fasting Lipid Profile					
				Fasting	Post Prandial			Total Cholesterol	TGL	HDL	LDL	VLDL	Non HDL
83	Kumaravel	40	M	69	84	5	100.7	225	258	41	133	51	184
84	Rajendran	35	M	73	106	5.3	111	191	154	44	116	31	147
85	Gopi	33	M	78	91	5.3	111	195	186	50	108	37	145
86	Vedamma	60	F	88	150	6.9	232	231	196	48	144	39	183
87	Vadivel	42	M	96	136	6.1		212	178	40	137	35	172
88	Venkatesan	45	M	75	107	5	100	164	149	52	82	30	112
89	Venkatesan	35	M	61	89	4.9	97.7	183	196	45	99	38	138
90	Mythili	35	F	85	112	5.2	107.8	195	138	50	117	28	145
91	Anthony	42	M	82	138	6.2	150	223	191	47	138	38	176
92	Kumar	38	M	79	93	4.9	97.1	201	226	48	108	45	153
93	Anbalagan	47	M	95	136	6.2	149	208	250	35	135	40	173
94	Lenin	38	M	102	140	6.1	145	201	230	35	138	38	166
95	Chidambaram	42	M	82	112	5.3	116	195	186	40	108	37	155
96	Durai	26	M	73	106	5.1	109	225	258	41	103	51	184
97	Pongavanam	40	F	69	84	5	105	191	154	38	116	31	153
98	Vasantha kumar	40	M	98	138	6	142	198	170	35	138	35	163
99	Kannan	45	M	85	120	5.4	120	180	150	40	135	30	140
100	Pugalendhi	22	M	78	116	5.2	113	190	170	42	138	35	148

LEVEL OF INJURY AND COMPLICATION

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
1	Amul Raj	23	M		1	1		1		1		C5	0	0	1			0	49/126
2	Ramesh	30	M		1	1		1			1	D12	0	0	0	0	0	0	47/126
3	Prabhu	35	M	1			1	1		1		C5	1	1	1	0	0	0	44/126
4	Sathish	35	M	1			1		1	1		C2, C3, C4	1	1	1	1	0	0	55/126
5	Prabhakaran	26	M	1		1			1	1		C6	0	0	1	0	0	0	54/126
6	Gnanavel	38	M		1	0	1	0	1	1	0	C4, C5	0	1	1	0	0	0	55/126
7	Sesu raj	34	M	1	0	1	0	1	0	0	1	D12	0	1	0	0	0	0	64/126
8	Kanaga sabapathy	26	M	0	1	1	0	1	0	0	1	L1 Burst fracture	1	1	0	0	0	0	91/126
9	Nagappan	63	M	0	1	0	1	0	1	1	0	C5, C6	0	0	0	0	0	0	63/126
10	Maya kannan	28	M	1	0	1	0	1	0	1	0	C5	0	1	1	0	0	0	48/126
11	Parathasarathy	25	M	0	1	1	0	1	0	0	1	L1 Burst fracture	0	1	0	0	0	0	69/126
12	Yosudha Krishnan	36	M	0	1	1	0	1	0	0	1	L2	0	0	0	0	0	0	69/126
13	Venu	39	M	0	1	0	1	1	0	1	0	C3, C4	0	1	0	0	0	0	48/126
14	Ponnusamy	62	M	1	0	0	1	1	0	1	0	D3, D4	0	0	0	0	0	0	70/126
15	Meghanathan	26	M	0	1	1	0	1	0	0	1	D12	1	1	0	0	0	0	68/126
16	Veeran	32	M	1	0	0	1	0	1	0	1	D11, D12	0	0	1	1	0	0	69/126
17	Sasikumar	22	M	0	1	1	0	0	1	1	0	C5	0	0	0	0	0	0	68/126

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
18	Premraj	44	M	0	1	0	1	1	0	0	1	D12, L1	1	1	0	0	1	0	67/126
19	Srinivasan	23	M	1	0	1	0	0	1	1	0	C4, C5	1	1	0	0	0	0	48/126
20	Sudhakaran	25	M	1	0	1	0	1	0	0	1	D6	1	0	1	0	0	0	68/126
21	Tirupathi	27	M	1	0	1	0	1	0	0	1	D12	1	1	0	0	0	0	73/126
22	Murugan	30	M	1	0	1	0	1	0	0	1	L1 Burst fracture	0	1	0	0	0	0	77/126
23	Kiran	20	M	0	1	1	0	0	1	0	1	L1 Burst fracture	0	0	1	0	0		68/126
24	Kumaran	43	M	1	0	0	1	0	1	0	1	L1 Burst fracture	1	1	0	0	0	0	55/126
25	Raja	35	M	0	1	0	1	0	1	0	1	D11, D12	1	0	0	0	0	0	48/126
26	Srinivasan	33	M	1	0	0	1	1	0	1	0	C4, C5	0	1	0	0	0	0	48/126
27	Raghupathy	40	M	1	0	0	1	1	0	0	1	D12	0	0	0	0	0	0	44/126
28	Sekar	43	M	0	1	0	1	0	1	1	0	C3, C4	0	0	1	0	0	0	48/126
29	Selvi	35	F	1	0	0	1	1	0	0	1	D8	0	0	0	0	0	0	60/126
30	Manimegalai	40	F	0	1	0	1	1	0	1	0	C4	0	0	0	0	0	0	90/126
31	Yuvarani	23	F	0	1	0	1	0	1	0	1	D9	0	0	0	0	0	0	90/126
32	Arumugam	60	M	0	1	0	1	1	0	1	0	C2 - C6	0	0	0	0	0	0	48/126
33	Sami kannu	37	M	1	0	0	1	1	0	0	1	D12	0	1	0	0	0	0	55/126
34	Saravanan	30	M	0	1	0	1	1	0	0	1	L1 Burst fracture	1	1	0	0	0	0	77/126

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
35	Ranjith	22	M	1	0	1	0	0	1	0	1	L1 Burst fracture	0	0	0	0	0	0	84/126
36	Priya	23	F	0	1	0	1	1	0	0	1	L1 Burst fracture	0	0	0	1	0	0	68/126
37	Lakshmi	32	F	1	0	0	1	0	1	0	1	D12	0	0	0	0	0	0	55/126
38	Kasturi	40	F	0	1	0	1	1	0	1	0	C6	0	0	0	0	0	0	66/126
39	Maadu	28	M	1	0	0	1	1	0	0	1	D11, D12	0	1	0	0	0	0	68/126
40	Nagaraj	29	M	0	1	0	1	1	0	0	1	D12	0	1	1	0	0	0	66/126
41	Damodaran	38	M	1	0	0	1	1	0	1	0	C3 - C7	0	1	1	0	0	0	48/126
42	Magesh	24	M	0	1	1	0	1	0	0	1	D12	0	1	1	0	0	0	60/126
43	Vijayan	45	M	1	0	0	1	1	0	1	0	C5	0	1	0	0	0	0	48/126
44	Prasanth	25	M	1	0	1	0	0	1	1	0	C3 - C7	0	0	0	0	0	0	57/126
45	Karthik	23	M	1	0	1	0	1	0	0	1	D10	0	1	0	0	0	0	66/126
46	Chandra	64	F	0	1	0	1	1	0	1	0	C2-C6	0	0	0	0	0	0	47/126
47	Chitra	28	F	1	0	0	1	0	1	1	0	C5	0	0	0	1	0	0	47/126
48	Velayudham	35	M	0	1	0	1	1	0	1	0	C4	1	1	0	0	0	0	48/126
49	Moovendan	29	M	0	1	1	0	1	0	1	0	C6	0	1	1	0	0	0	54/126
50	Syed Ibrahim	28	M	1	0	1	0	1	0	0	1	D10-D11	0	0	0	0	0	0	56/126
51	Ravi	45	M	1	0	0	1	1	0	0	1	D11	0	0	1	0	0	0	62/126
52	Mani	56	M	1	0	0	1	1	0	0	1	D12	0	1	1	0	0	0	54/126
53	Vadivelu	42	M	0	1	0	1	1	0	0	1	D12	0	1	1	0	0	0	83/126

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
54	Kannan	45	M	1	0	0	1	0	1	0	1	D12	1	1	0	0	0	0	52/126
55	Babu	25	M	1	0	1	0	0	1	0	1	D12	1	1	1	0	0	0	64/126
56	Vasantha kumar	40	M	1	0	0	1	1	0	0	1	L1 Burst fracture	1	0	1	0	0	0	52/126
57	Mohan	39	M	1	0	0	1	1	0	0	1	L2	1	1	0	0	0	0	90/126
58	Sakthivel	27	M	1	0	0	1	1	0	0	1	D12	1	1	0	0	0	0	72/126
59	Mani	60	M	0	1	0	1	0	1	1	0	C5	0	0	0	0	1	0	68/126
60	Masilamani	40	M	1	0	0	1	1	0	0	1	D12	1	0	0	0	0	0	58/126
61	Sivakumar	40	M	1	0	0	1	1	0	0	1	D8	1	1	0	0	0	0	52/126
62	Jayaraman	35	M	0	1	0	1	1	0	1	0	C3-C4	0	0	1	0	0	0	47/126
63	Chinna durai	35	M	1	0	0	1	1	0	0	1	L1 Burst fracture	1	0	1	0	0	0	60/126
64	Murugesan	51	M	1	0	0	1	0	1	0	1	D12	0	0	0	0	0	0	52/126
65	Vinoth	22	M	1	0	1	0	1	0	0	1	L1 Burst fracture	1	1	1	0	0	0	70/126
66	Ammactchi	39	F	0	1	0	1	0	1	0	1	L2	0	0	0	0	0	0	86/126
67	Abirama sundari	23	F	1	0	1	0	0	1	0	1	D8	0	0	1	0	0	0	48/126
68	Selvi	35	F	0	1	0	1	0	1	0	1	D12	0	0	0	1	0	0	52/126
69	Ravi	35	M	0	1	0	1	1	0	0	1	L2	1	0	0	0	0	0	72/126
70	Rajendran	36	M	1	0	0	1	0	1	0	1	D12	1	0	0	0	0	0	66/126
71	Anthony Raj	30	M	1	0	0	1	1	0	0	1	D12	1	1	1	0	0	0	52/126
72	Venkatesan	45	M	1	0	0	1	1	0	0	1	D12	0	1	0	0	0	0	60/126

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
73	Hariharan	38	M	1	0	0	1	0	1	0	1	D12	0	0	0	0	0	0	58/126
74	Sivalingam	60	M	1	0	0	1	1	0	0	1	D12	0	1	0	0	0	0	60/126
75	Krishnakumar	28	M	1	0	1	0	0	1	0	1	D12	0	0	1	0	0	0	52/126
76	Nagappan	61	M	1	0	0	1	1	0	0	1	L1 Burst fracture	1	0	0	0	0	0	60/126
77	Charan	20	M	0	1	1	0	0	1	1	0	C5	0	0	1	0	0	0	60/126
78	kannan	32	M	1	0	0	1	0	1	0	1	L1 Burst fracture	1	1	0	0	0	0	62/126
79	Velu	62	M	1	0	0	1	1	0	0	1	D12	0	1	1	0	0	0	58/126
80	Sampath	47	M	0	1	0	1	1	0	1	0	C5	1	0	0	0	0	0	70/126
81	Arul	29	M	1	0	0	1	1	0	0	1	L1 Burst fracture	0	1	1	0	0	0	68/126
82	Manikandan	25	M	1	0	1	0	0	1	1	0	C3	0	0	1	0	1	0	47/126
83	Kannadasan	45	M	0	1	0	1	1	0	0	1	D12	1	1	0	0	0	0	87/126
84	Poongavanam	40	F	0	1	1	0	1	0	0	1	L1 Burst fracture	1	0	0	0	0	0	92/126
85	Pugalendhi	22	M	0	1	0	1	0	1	1	0	C5	0	0	0	0	0	0	47/126
86	Rehamath Nisa	44	F	0	1	0	1	1	0	0	1	L2	0	0	1	0	0	0	68/126
87	Balamurugan	31	M	1	0	1	0	1	0	0	1	D12	1	1	0	0	0	0	52/126
88	Durai	26	M	1	0	1	0	0	1	0	1	D12	0	0	0	0	0	0	58/126
89	Shanti	42	F	1	0	0	1	0	1	0	1	D12	0	0	0	0	0	0	50/126
90	Kumaravel	40	M	1	0	0	1	1	0	0	1	D12	1	1	0	0	0	0	60/126

S. No	Name	Age	Sex	ASIA Score		Martial Status		Mode of Injury		Lesion		Level of Injury	Smo-king	Alcohol	Complications			Family History	FIMS
				A	B	U	M	Fall	RTA	Q	P				Pressure Ulcer	DVT	H.O		
91	Rajendran	35	M	1	0	0	1	1	0	0	1	L1 Burst fracture	1	1	0	0	0	0	58/126
92	Gopi	33	M	1	0	1	0	0	1	0	1	D12	0	0	0	0	0	0	58/126
93	Vedamma	60	F	0	1	0	1	1	0	1	0	C5	0	0	1	0	0	0	60/126
94	Venkatesan	35	M	0	1	0	1	1	0	1	0	C5	1	1	0	0	0	0	58/126
95	Mythili	35	F	1	0	0	1	0	1	1	0	D12	0	0	0	1	0	0	58/126
96	Anthony	42	M	0	1	0	1	0	1	1	0	C5	1	1	1	0	0	0	58/126
97	Kumar	38	M	1	0	0	1	1	0	0	1	L1 Burst fracture	0	1	0	0	0	0	57/126
98	Anbalagan	47	M	1	0	0	1	0	1	1	0	C4	0	0	1	0	0	0	47/126
99	Lenin	38	M	1	0	0	1	1	0	0	1	D8	1	1	0	0	0	0	58/126
100	Chidambaram	42	M	1	0	0	1	1	0	0	1	D12	1	1	0	0	0	0	60/126

A2 - Study Proforma

Below is the study proforma

1	Name	
2	Age	
3	Sex	
4	BMI	
5	Occupation	
6	Socio-Economic status	
7	Level of injury	
8	Duration of Injury	
9	Mechanism of Injury	
10	Previous History of Dyslipidemia	
11	Previous History of CAD	
12	Personal History	
	Investigations	
13	OGTT	
14	HbA1C	
15	Fasting lipid profile	

A3 – Consent Form in Tamil

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

முதுகு தண்டுவடத்தில் அடிப்பட்ட நோயாளிகளுக்கு இரத்தத்தில் சர்க்கரை மற்றும் கொழுப்பு சத்தின் அளவை பரிசோதனை செய்வதன் மூலம் இருதய பாதிப்பை மதிப்பிட வருங்கல ஆய்வு.

பெயர் : வயது : தேதி : பங்கேற்பாளர் எண் :

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முறையாக மருத்துவரிடம் கேட்டு அறிந்து கொண்டேன். எனது சந்தேகங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன் பங்கு கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்து கொள்ளலாம் என சம்மதிக்கிறேன்.

இந்த ஆய்வில் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ வெளியிடப்பட்டாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்று கொண்டேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்றும் தெரிவிக்கிறேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்றும் தெரிந்து கொண்டேன்.

பங்கேற்பாளர் கையொப்பம் தேதி :

ஆய்வாளர் கையொப்பம் தேதி :

தகவல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

முதுகு தண்டுவடத்தில் அடிப்பட்ட நோயாளிகளுக்கு இரத்தத்தில் சர்க்கரை மற்றும் கொழுப்பு சத்தின் அளவை பரிசோதனை செய்வதன் மூலம் இருதய பாதிப்பை மதிப்பிட வருங்கல ஆய்வு.

ஆய்வாளர் : **மரு. ராம காமாட்சி,**
முதலாம் ஆண்டு பட்டமேற்படிப்பு மாணவி,
மருத்துவம் மற்றும் மறுசீரமைப்பு உயர்நிலைத் துறை,
சென்னை மருத்துவக் கல்லூரி,
சென்னை-600003.

முதுகு தண்டுவடத்தில் காயம் ஏற்படுவதால் கை மற்றும் கால்கள் செயலிழந்து போய்விடுகிறது. இதனால் அன்றாட வேலைகளை செய்வதற்கு கூட மற்றவர்களின் உதவி தேவைப்படுகிறது. உடற்பயிற்சி குறைவு, மற்றும் அதிக நாட்கள் படுக்கையிலேயே இருக்க நேரிடுவதால் தசைகள் செய்நலிவு ஏற்படுகிறது. இரத்தத்தில் சர்க்கரை மற்றும் கெட்ட கொழுப்பு சத்தின் அளவு அதிகமாகிறது. நல்ல கொழுப்பு சத்து குறைகிறது. இருதய பாதிப்புகள் ஏற்படுவது அதிகமாகிறது. இதனை முன்னரே கண்டறிந்தால் இருதய பாதிப்பு ஏற்படுவதை தவிர்க்கலாம்.

இந்த ஆய்வையொட்டி எந்த விதமான சந்தேகங்களுக்கும் விளக்கம் பெற பங்கேற்பாளர்களுக்கு உரிமை உள்ளது.

இந்த ஆய்வில் ஆகும் அதிகப்படியான செலவிற்கு நோயாளிகளிடமிருந்து பணம் பெற்றுக்கொள்ளப்படமாட்டாது.

இந்த ஆய்வின் முடிவுகள் இறுதியில் பிரசுரிக்கப்படும். இந்த ஆய்வை பற்றிய சந்தேகங்கள் முழுமையாக தங்களுக்கு விளக்கப்படும். தொடர்பு கொள்ள வேண்டியவர் : மரு. ராம காமாட்சி, செல் : 9442032177

ஆய்வாளர் கையொப்பம்

தேதி :

பங்கேற்பாளர் கையொப்பம் /
இடதுகை பெருவிரல் ரேகை

தேதி :

A5 – Patient Information Sheet

A study titled "To Assess Cardiovascular Risk in cases of Spinal Cord Injury by screening Impaired Glucose Tolerance and Dyslipidemia-Cross sectional Study" is being conducted at Government Institute of Rehabilitation Medicine, KK Nagar, Chennai 600 083.

The purpose of this study is to assess the cardiovascular risk in patients with SCI.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Place: Chennai

Date:

A6 – Previous Presentations and Publications

Poster 1:

Effective Rehabilitation of Traumatic Paraplegia with multiple bony injuries.

Poster 2:

Effective Rehabilitation of Neurofibromatosis -1 with congenital Scoliosis and Paraplegia.

Poster 3:

Comparative efficacy of Hydrogel metal coated catheter with Regular Foley's catheter in UTI reduction.

Paper 1:

Correlation of Blood Group Among Cases of Dysvascular amputation

Paper 2:

Assessment Cardiovascular risk in cases of Spinal Cord Injury.

Paper 3: (Published SMJ Journal)

Comparative analysis of Spinal Cord Injury among male and female population.