STUDY ON PROFILE OF HEMIPLEGIA IN CHILDREN

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERSITY

in partial fulfillment of the regulations
for the award of the degree of

M.D. BRANCH - VII

PEDIATRICS

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERSITY

CHENNAI, INDIA

MARCH - 2008

CERTIFICATE

This is to certify that the dissertation titled "**STUDY ON PROFILE OF HEMIPLEGIA IN CHILDREN"** is the bonafide original work of **Dr.R.VANITHA,** in partial fulfillment of the requirements for **M.D., Branch - VII, (Pediatrics)** Examination of the Tamil Nadu Dr.M.G.R. Medical University to be held in March 2008.

DEAN
Govt. Stanley Medical College
and Hospital
Chennai - 600 001.

DIRECTOR
Department of Pediatric Medicine
Institute of Social Pediatrics,
Govt. Stanley Medical College and
Hospital,
Chennai - 600 001.

DECLARATION

I **Dr.R.VANITHA,** solemnly declare that this dissertation, "**STUDY ON PROFILE OF HEMIPLEGIA IN CHILDREN"** is a bonafide record of work done by me in the Department of Paediatrics, Institute of Social Pediatrics, Govt. Stanley Medical College and Hospital, Chennai, under the guidance of **Prof.Dr.SUJATHA SRIDHARAN, M.D., D.C.H.,** Director, Institute of Social Pediatrics, Govt. Stanley Medical College and Hospital, Chennai - 600 010.

This dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University regulations for the award of **M.D. Branch - VII, (Pediatrics)** Examination to be held in March 2008.

Place : Chennai

Dr.R.VANITHA

Date :

ACKNOWLEDGEMENT

I am extremely grateful to the Dean, **Dr.MYTHILI BHASKARAN, M.D.,** Govt. Stanley Medical College and Hospital for granting me permission to conduct this study at Institute of Social Pediatrics, Govt. Stanley Medical College and Hospital, Chennai.

I would like to express my sincere gratitude to my beloved Professor and Director, **Prof.Dr.SUJATHA SRIDHARAN, M.D., D.C.H.,** and **Prof.Dr.KARUNAKARAN, M.D., D.C.H.,** Institute of Social Pediatrics, Govt. Stanley Hospital, for their support and encouragement to conduct this study.

With extreme gratitude, I express my indebtedness to my beloved Professor, **Dr.STEPHEN ABRAHAM SURESH KUMAR, M.D., D.C.H., D.M. (Neurology),** Department of Neuromedicine, Institute of Social Pediatrics - Govt. Stanley Hospital, for his motivation, advice and valuable criticism, which enabled me to complete this work.

I offer my special thanks to my Asst. **Prof.Dr.M.ARAVIND, M.D., D.C.H.,** for his invaluable help and suggestions which gave a final shape to my study.

I would like to offer my gratitude to the registrar **Dr.C.N.KAMALARATHINAM, M.D., D.C.H.** for his kindness and guidance for my study.

I also thank my Assistant Professors, **Dr.J.GANESH, M.D., D.C.H., Dr.ELANGO, M.D., D.C.H., Dr.N.THIYAGARAJAN, M.D., (Pediatrics),** and **Dr.KUMAR, D.C.H.,** for their critical review and suggestions.

I also thank **Prof.Dr.JOHN SOLOMON, M.D., D.C.H.,** Assistant Professor, Department of Clinical Epidemiology, Institute of Social Pediatrics, Govt. Stanley Hospital, for

his valuable suggestions.

I also thank **Mr.A.VENKATESAN,** Lecturer in Statistics, Clinical Epidemiology Unit, Institute of Social Pediatrics, Govt. Stanley Medical College for his invaluable help in analysing the values.

I am greatly indebted to my family and friends who have been the greatest source of encouragement and support which enabled me to complete this work.

I am grateful to all the children in my study to their co-operation and patience.

CONTENTS

INTRODUCTION

“Kids have strokes

Stroke is not just an adult condition.

Strokes can even happen before birth.”

Hemiplegia in children is now recognised as an important cause of morbidity and mortality.There are fundamental developmental differences in hemiplegia in children compared with adults which make the recognition and treatment of children challenging.Hemiplegia in children is relatively rare and frequently results in a lack of recognition and delay of diagnosis.The etiologies of hemiplegia in children are legion, no single risk factor predominates.A wide variety of conditions predispose to cerebral infarct or hemorrhage in children and the underlying mechanism and cause in each individual can only be recognized with an informed and careful diagnostic approach.Increased awareness of hemiplegia will result in being brought to medical attention as rapidly as possible.With more rapid diagnosis,newer forms of thrombolytic and neuroprotective agents may become future treatment options for these children.Large multicenter collaborative intervention trials are necessary to determine the role of antithrombotic and other therapies in pediatric patients with hemiplegia.Although rare in children the effects of hemiplegia have a significant impact on childs development and lifelong burden of illness.Therefore the development of specific diagnosis and therapy for these conditions is of paramount importance.To define the service needs, population based studies of incidence, etiology, risk factors and outcome of childhood hemiplegia and cerebrovascular disease are needed.

This study was undertaken to analyse the epidemiological and clinical profile, the etiologic and risk factors of hemiplegia and the disability caused by hemiplegia in children.

REVIEW OF LITERATURE

Hemiplegia is paralysis of one side of the body.It denotes the involvement of pyramidal tract on the opposite side.It can be classified into congenital and acquired, acute and chronic.Congenital hemiplegia is otherwise known as hemiplegic cerebral palsy.Acute hemiplegia is a condition where weakness develops within a few hours.In chronic hemiplegia weakness evolves over days,weeks or months.

a. Acute hemiplegia

1. Vascular:
    - Thromboembolic occlusion of internal carotid artery
    - Cerebral infarct
    - Intracranial hemorrhage
2. Infection
    - Meningitis
    - Encephalitis
3. Trauma
    - Subdural hemorrhage
    - Extradural hemorrhage
    - Intracranial hemorrhage

b. Chronic hemiplegia

1. Cerebral palsy
2. Post meningitic \ Post encephalitic sequalae
3. Post traumatic residual weakness
4. Space occupying lesion
5. Cerebral abscess
6. AVmalformation
7. SturgeWeber syndrome

c. Recurrent hemiplegia:

1. Hemiplegic migraine
2. Moyamoya disease
3. Alternating hemiplegia of childhood
4. MELAS
5. Lipoprotein disorders
6. Todds paralysis

ACQUIRED HEMIPLEGIA

Acute infantile hemiplegia is characterized by the sudden onset of hemiplegia in infancy or childhood usually prior to 6 years of age[24]. In adult life, arterial occlusion is most frequently the consequence of arteriosclerosis of cerebral vasculature. In childhood, arterial occlusion usually results from congenital dysplasia of the vessels, cerebral arteritis or trauma. Since the anterior cerebral circulation is usually affected, acute hemiplegia results.

The syndrome of acute hemiplegia was first described in the nineteenth century by a number of authors including Freud[17], often under the term MarieStrumpell encephalitis, a nomenclature designed to stress its supposed relationship to Polio encephalitis.

Hemiplegia secondary to vascular disorders occur in children with an incidence of 1-3 per 100,000 per year[11].Schoenberg et al[39] showed that the combined incidence of all pediatric strokes is 2.5 to 2.7 per 100,000 per year.The incidence of pediatric hemorrhagic stroke is 1.9 per 100,000 per year.The incidence of pediatric ischaemic stroke is 1.2 per 100,000 per year.In the past two decades, the reported incidence of pediatric ischaemic stroke is changing and appears to be increasing. As Freuds[17] description, Ingram[22] and Lanska et al[28] found that acquired hemiplegia regardless of cause usually occurs before the age of 3years.deVeber et al[11] showed that there was a slight male predominance.

The pediatric causes of hemiplegia are distinctive compared with adult cases. The cause of hemiplegia in children is established in approximately 75% of cases.

CAUSES OF ACQUIRED HEMIPLEGIA IN CHILDREN

I. CARDIAC DISEASE

A. CONGENTIAL

1. Aortic stenosis
2. Mitral stenosis;mitral prolapse
3. Ventricular septal defect
4. Patent ductus arteriosus
5. Cyanotic congential heart disease involving right- to-left shunt

B. ACQUIRED

1. Endocarditis
2. Kawasaki disease
3. Cardiomyopathy
4. Atrial myxoma
5. Arrhythmia
6. Paradoxical emboli through patent foramen ovale
7. Rheumatic fever
8. Prosthetic heart valve

II. HEMATOLOGIC ABNORMALITIES

A. Hemoglobinopathies

1. Sickle cell disease

B. Polycythemia

C. Leukemia/lymphoma

D. Thrombocytopenia

E. Thrombocytosis

F. Disorders of coagulation

1. Protein C deficiency
2. Protein S deficiency
3. Factor V Leiden
4. Antithrombin III deficiency
5 Lupus anticoagulant
6. Oral contraceptive pill use
7. Pregnancy and the postpartum state
8. Disseminated intravascular coagulation
9. Paroxysmal nocturnal hemoglobinuria
10. Inflammatory bowel disease

III INFLAMMATORY DISEASE

A Meningitis

1. Viral
2. Bacterial
3. Tuberculosis

B Systemic infection

1. Viremia
2. Bacteremia
3. Local head and neck infection

C. Drug-induced inflammation

1. Amphetamine

2. Cocaine

D. Autoimmune disease

1. Systematic lupus erythematosus
2. Juvenila rheumatoid arthritis
3. Takayasu arteritis
4. Mixed connective tissue disease
5. Polyarteritis nodosum
6. Primary central nervous system vasculitis
7. Sarcoidosis
8. Behcet's syndrome
9. Wegener granulomatosis

IV METABOLIC DISEASE ASSOCIATED WITH STROKE

A. Homocystinuria

B. Pseudoxanthoma elasticum

C. Fabry disease

D. Sulfite oxidase deficiency

E. Mitochondrial disorders

1. MELAS
2. Leigh syndrome

F. Ornithine transcarbamylase deficiency

V INTRACEREBRAL VASCULAR PROCESSES

A. Ruptured aneurysm

B. Arteriovenous malformation

C. Fibromuscular dysplasia

D. Moyamoya disease

E. Migraine headache

F. Postsubarachnoid hemorrhage vasospasm

G. Hereditary hemorrhagic telangiectasia

H. Sturge-Weber syndrome

I. Carotid artery dissection

J. Post varicella angiopathy

VI TRAUMA AND OTHER EXTERNAL CAUSES

A. Child abuse

B. Head trauma/neck trauma

C. Oral trauma

D. Placental embolism

E. ECMO therapy

William et al[45] showed in their study the following causes for cerebrovascular occlusive disease,cyanotic heart disease in 8%,valvular heart disease in 1%,myoendocarditis in 2%,sickle cell disease in 17%, prothrombotic disorders in 5%, Moyamoya disease in 9%,vasculitis in 3%,miscellaneous in 5% and unknown in 31%.

Solomon et al[40] observed the following causes for acquired hemiplegia in 86 children as trauma in 11 patients, CNS infections(11), cardiac disease(10), sickle celldisease(5),

Arteriovenous malformation(4), documented occlusive vascular disease(16), unknown(25), miscellaneous(4).

deVeber et al[11] found the primary risk factors for AIS as cardiac disease(19%), coagulation disorders(14%), dehydration(11%), infection(6%), vasculitis in 7%,dissection (5%), cancer (4%), metabolic disorders (3%), Moyamoya disease (2%), sickle cell disease(2%), miscellaneous(4%).In 21% no risk factor was identifiable.

One of the most common identifiable cause for childhood ischemic stroke is complex congenital heart disease. Emboli formed in the heart can reach the cerebral circulation directly.Prosthetic heart valves are an important source of emboli.In the presence of ASD or VSD with intermittent right to left intracardiac shunting, systemic venous clots can reach the cerebral circulation[24].

DiTullio et al[13] observed small otherwise significant defects including PFO as risk factor for paradoxical embolism and stroke in young children.Webster et al demonstrated right to left shunting in 50% of patients by contrast echocardiography.

Fever and non specific viral infections are frequently present in children with cerebral infarction.Powell et al[36] observed arterial or venous stroke in 5% to 12% of children with meningitis. Meningitis should be considered in AIS with associated fever and selected cerebrospinal and serum studies for bacterial, tuberculous, and viral meningitis may be indicated.

Ganesan et al [18] reported that inherited or acquired coagulation disorders can predispose a child to AIS.Deficiencies in protein C or S appear to represent important risk factor for intracerebral occlusive disease in pediatric population.Sickle cell disease is the most common

hemoglobinopathy associated with cerebrovascular disease.Wood et al[46] found that 25% of sickle cell disease develop cerebrovascular complications.About 80% of such patients are children less than 15years of age.

Non infectious inflammatory vasculitis is associated with cerebral infarction in adults and can also be rarely the cause for AIS in children.Devinskey et al[12]observed SLE is often associated with AIS, but vasculitis as the etiology for stroke is rare.

Homozygous homocystinuria predisposes to arterial and venous thrombosis including cerebral infarcts.Bodensteiner et al[5] noted that thromboembolic manifestations can precede other features of the disease.

Glueck et al [19] stressed the importance of familial lipid and lipoprotein abnormalities in children with cerebrovascular occlusive disease.In particular low levels of high density lipoproteins and high levels of triglycerides can predispose to occlusive vascular disease.Additionally a high percentage of children with cerebrovascular occlusive disease have first degree relatives with histories of coronary heart disease and cerebrovascular accidents.

PATHOPHYSIOLOGY

In-situ thrombosis of the cerebral arteries can result from primary disorders pf the cerebral arteries or from acquired or congential prothrombotic states.Under physiologic circumstances,the endothelial lining of arteries provides an anticoagultant surface that helps maintain blood in a fluid phase.When the arterial wall is damaged,it becomes prothrombic and potentiates the formation of a thrombus through several mechanisms.Cerebral artery stenosis slows distal blood flow and potentiates nonlaminar blood flow that provides another mechanism for thrombosis.Embolic sources for AIS in childhood include structural disorders of

the heart,aorta,and cerebral arteries.These abnormalities result in cardiogenic embolism or artery-to-artery embolism.A further mechanism is the presence of intermittent right-to-left intracardiac shunting,such as with artial septal defects,patent foramen ovale and postoperative residual right to left shunts after surgery.This shunt provides a conduit for paradoxical emboli and effectively allows systemic venous thrombi to reach the cerebral circulation.

In AIS the severity of cerebral tissue damage and the resultant neurologic impairments are a function of the duration of ischemia,the size and location of brain structures supplied by the involved cerebral artery,the availability of collateral arterial blood supply,and the concurrent metabolic demands of the brain.

The delivery of substrates is impaired in degrees of severity that vary by the duration of vascular occlusion and the adequacy of collateral blood flow supplying the ischemic area.Depending on the severity of ischemia and the rate of neuronal metabolic activity,neuronal damage after arterial occlusion can be reversible or irreversible.In transient ischemic attacks (TIAs) the clinical deficit is usually extremely brief,typically lasting less than hour and even by MRI no permanent parenchymal lesion is seen,confirming that the damage has been fully reversible.In an arterial infarct the clinical deficit last longer,sometimes persisting for greater than 24 hours,and there is a radiographically confirmed infarct in the vascular territory appropriate to the neurologic syndrome.Even in AIS ,there is a central core zone ,which represent s irreversibly damaged brain and a surrounding penumbra zone which represents potentially viable brain tissue.The eventual volume of the ischemic infarct is the result of the balance between the rate of delivery of oxygen and glucose and the metabolic activity of the brain.

The metabolic effect of focal interruption of blood supply with focal brain ischemia are regional hypoxia and depletion of high-energy compounds, such as adenosine triphosphate

(ATP) and carbhohydratye stores. The metabolic rate of cerebral tissues is increased relative to other body tissues and there is a relative paucity of energy stores in the brain. The cerebral metabolic rate for oxygen is 3.5 ml per 100 gm of brain per minute. Since virtually no oxygen reserve is available, rapid loss of neuronal function results if the oxygen supply is interrupted.Brain glucose reserve is slightly greater, allowing for survival of brain tissue for up to 90 minutes if adequate oxygen is supplied The newborn can use lactate as a substrate for the production of energy, but this capability is lost quickly. when regional hypoxia develops during ischemia there is a shift from oxidative to glycolytic metabolism. Glucose is metabolized to lactate,which accumulates and results in acidosis that, in turn exacerbates hypoxic injury. Seizures,by increasing the neuronal metabolic rate,dramatically increase the extent of neuronal damage occuring during ischemia.

CLINICAL MANIFESTATIONS

The following is Freud's[17] description of an acute ischemic stroke.

"A child who has hitherto been well and with out hereditary predisposition is suddenly taken ill at an age between a few months to 3 years. The etiology of the disease either remains unexplained or is attributed to a concurrent infection .The presenting symptoms may be either stormy, with fever, convulsions, and vomiting, or in significant ,they may last 1 day or up to several weeks. The disease cannot be diagnosed with certainty in this initial stage. A hemiparesis may appear at this or not until later .It spreads in the usual matter : first face, then arm, then leg. At first it is a flaccid paresis, but very soon it becomes spastic, with increased reflexes and contractures. Partial or complete aphasia appear commonly, but are usually transient : hemianospia or paralyses of the ocular muscles are rare .The paresis may vanish or else recur in bouts with increasing severity : most commonly it is permanent. Improvement is more likely in the leg than the arm in such a case the child walks with circumduction of the

affected hip. Improvement of the paresis is very commonly associated with post hemiplegic chorea together with a greater or lesser degree of residual spasticity. Growth atropy of the affected limbs which is often extensive becomes apparent during the pubertal growth spurt. Impasired intelligence is seldom absent. Epileptic seizures may make their appearance at a variable interval after the initial illness. These are at first unilateral. Later they become generalized and severe. There is no time limit to the appearance of epileptic seizures.''

Lanska et al [28]observed that acute hemiplegia occurred before 3 years of age.Seizures at the onset of stroke are relatively frequent in children compared with adults.Ingram et al[22] found that 60% of the patients had unilateral or generalized seizures at the onset of hemiplegia.After the convulsion,neurologic function is lost or severely impaired.Headache is an important feature of basal arterial occlusion without telangiectasia.

DIAGNOSTIC EVALUATION IN A CHILD WITH HEMIPLEGIA

1. First line: Performed within first 48 hours of admission

a. CT scan of brain

b. MRI of brain

c. Complete blood count

d. PT \ PTT

e. Electrolytes

f. Calcium, magnesium, phosphorus

g. Sugar

h. LFT

i. Chest xray

j. ESR

k. ANA

l. Urine analysis

m. BUN, creatinine

n. Urine drug screen

o. ECG

2. Second line: Performed within first week as indicated

a. ECHO

b. Transcranial and carotid dopplers

c. MRA

d. Hypercoagulable state evaluation:

- Antithrombin III
- ProteinC, S
- Factor V leiden mutation
- Antiphospholipid antibody
- Anticardiolipin
- Lupus anticoagulant

e. Rheumatoid factor

f. Serum aminoacids

g. Urine for organic acids

h. Blood culture

i. Hemoglobin electrophoresis

j. Complement profile

k. Serum and urine homocystine

l. Lactate and pyruvate

m. Ammonia

n. CSF cell count,protein,glucose,lactate

o. Lipid profile

3.Third line: : Performed electively as indicated

a. HIV

b. Lyme titers

c. Mycoplasma titers

d. DNA testing for MELAS

e. Cerebral angiogram

f. Lepto meningeal biopsy

Neuroimaging

CT scan of the brain is often normal within the first 12 hours after ischemic stroke. In children CT findings of AIS are similar to those in adults. MRI is more sensitive than CT for the diagnosis of infarction within 24 hours, and is comparable for the diagnosis of hemorrhage27. In the brainstem and cerebellum, MRI is far superior to CT, since it avoids bone artifact. Modifications of MRI that have improved the early detection and specificity of ischemic injury include diffusion-weighted imaging. Perfusion MR imaging and proton MR spectroscopic imaging.

MRA can be performed at the same time as MRI and adds valuable information regarding the cerebral arteries. SPECT scanning has the ability to detect areas of hypoperfusion that occur earlier than other radiographically detected defects in AIS.

MANAGEMENT

The acute treatment of cerebral ischemia is largely supportive and requires an intensive care unit setting.Attention to oxygenation, fluid and electrolyte status,seizures and infections are critical.Treatment should be directed to the underlying cause if it is identifiable.Cerebral edema is maximal over the first 72 hours.Edema is usually effectively managed with hyperventilation and fluid restriction.In case of progressive deterioration, mannitol and steroids may be used[1].

The use of anticoagulation in pediatric ischemic stroke is controversial[1,24,27.] Anticoagulation is contraindicated in hemorrhagic infarct.Long term anticoagulation with warfarin is indicated in deficiency of protein C,S, antithrombin III and in presence of antiphospholipid antibodies.Low dose aspirin is a consideration though controlled studies in children have not been performed.Rehabilitation through aggressive physical,occupational and speech theraphy is essential for all patients.

OUTCOME

deVeber et al[11] observed a normal outcome in 50% of patients at 9 months average follow up interval,in population based studies of patients after neonatal AIS.The remaining patients had seizures or neurologic deficits that were usually mild in severity.Trauner et al[42] in his study showed that motor deficits have been reported in about 75%,cortical sensory deficits in one third, and seizure disorders in one third of neonates after AIS.

The improved outcome reported in more recent studies of childhood AIS may reflect the detection of milder forms of stroke enabled by MRI, an increasing trend to anticoagulation theraphy.

deVeber et a[l1]1 showed that mortality after AIS was 6%. Lanska et al[28] found that the mortality rate was 14%.Death is usually related to the underlying cause for stroke and less frequently results from the stroke itself.

CONGENITAL HEMIPLEGIA

It is otherwise known as hemiplegic cerebral palsy. It is characterized by a unilateral paresis that nearly always affects the upper extremity to a greater extent than the lower and that ultimately is associated with some spasticity and flexion contractures of the affected limbs. Males are commonly affected than females[14].Male: Female ratio is 3:2. Right side is more commonly affected than left side[25,27]. Right preponderance applies to both males and females.

ETIOLOGY

Of the various unilateral lesions seen in term infants with congenital hemiparesis, vascular infarcts are the most prominent. These are usually seen in the territory of the middle

cerebral artery with the left artery being more frequently affected than the right. A variety of causes have been impicated in the focal infarction. These include perinatal asphyxia, thromboembolism, polycythemia, dehydration, cocaine abuse, ECMO and a coagulopathy, notably a mutation in factor V, Leiden. Other causes for hemiparesis include PVL, Schizencephaly, porencephaly, pachygyria, unilateral hemimegalencephaly and focal dysplasias of the cerebral cortex[25].

Hagberg et al[21] observed that spastic hemiparesis accounted for 56% of term infants and 17% of preterm infants with cerebral palsy. Cohen et al[9] found pregnancy abnormalities in 5 cases, delivery abnormalities in 20 cases and no abnormalities in 27 cases.

CLINICAL MANIFESTATIONS

For unknown reasons ,the hemiparesis is only rarely documented at birth, although some subsequently hemiparetic infants present with focal seizures during the first few days of life.Byer et al[7] traced the evolution of hemiparesis from its appearance in the neonate to the spasticity seen in the older child. In older children, the extent of impaired voluntary function varies considerably from one patient to another.

Generally, fine movements of the hands are the most affected, notably the pincer grasp of thumb and fore finger, extension of the wrist, and supination of the forearm, proximal muscle power is well preserved, and function in the upper extremity relates to speed of movements and power in the distal musculature[25].In the lower extremity, dorsiflexion and eversion of the foot are impaired most frequently, with power in the proximal muscles being preserved. Increased flexor tone is invariable, leading to a hemiparetic posture, with flexion at the elbow, wrist and knees, and an equinus position of the foot. Despite these abnormalities, most children with pure hemiparetic cerebral plasy walk by 20 months of age. Deep tendon

reflexes are increased and Babinski and less often Hoffman reflexes can be elicited.In most children, the palmar grasp reflex persists for many years.

A large proportion of hemiparetic children has involuntary movements of the affected limbs. The involuntary movements are seen most clearly in the hand, where the patient demonstrates an avoidance response and athetotic posturing of the hand, producing overextension of the fingers and occasionally of the wrist, as the child attempts to hold an object. This type of posture is similar to that of patients with parietal lobe lesions. Before 10 years of age these movements are more evident in the unaffected hand; thereafter, they occur in both affected and unaffected hands these changes are believed to reflect callosal inhibition of the uncrossed motor pathways and the organizational changes of the pyramidal motor system.

Sensory abnormalities of the affected limbs are common. Stereognosis is impaired most frequently; less often, two point discrimination and position sense is defective. In general ,the severity of the sensory defect does not correlate with the severity of the hemiparesis. Brown et al[6] observed that stereognosis and graphesthesia are compromised to a varying degree.

Growth disturbances of the affected limbs are extremely common and like the sensory defects, probably reflect damage to the parietal lobes[25].Failure of growth, most evident in the upper extremities and particularly in the terminal phalanges and in the size of the nail beds is a result of under development of muscle and bone. Growth arrest is not always accompanied by sensory changes.

Black et al [4]observed homonymous hemianopia in 10% of patients with spastic hemiparesis. Between 17% and 27% of hemiparetic patients have homonymous hemianopia. Abnormalities in cranial nerve function are frequent and usually the result of a supranuclear involvement of the muscles innervated by the lower cranial nerves. Facial weakness is probably

the most common abnormality. Deviation of tongue and convergent strabismus are seen less often.

More than one half of hemiparetic patients develop seizures. Cohen and Duffner et al [9] observed that in 52%, seizures first appear before 18 months of age and only 8% of hemiparetic children suffer their first attack after age 10 years. For those who had experienced seizures during the neonatal period, the likelihood of recurrence is high. A paroxysmal EEG almost invariably indicates the presence of a seizure disorder.

Wiklund et al [44]showed that CT scan brain was normal in 29%, periventricular atrophy in 42%, maldevelopment in 17%, cortical-subcortical atrophy in 12%, and miscellaneous findings in 3%.

Approximately one half of the hemiparetic children have average IQ and 18% score above 100.Nearly all of the hemiparetic patients are educationally competitive and ultimately become atleast partially independent economically.Neither is there a consistent relationship between the extent of the lesion,the severity of hemiparesis and the functional outcome.

Porencephalic cyst

A porencephalic cyst[25] is a large intraparenchymal cyst that communicate with the ventricular system. Porencephaly results from infarction in the territory of a major artery, usually the middle cerebral artery,although at times it may be a sequel to a grade4 IVH that extends the ventricle lumen into the empty parenchymal space left by the reabsorption of the hematoma.It is not a watershed infarct. Porencephaly is usually limited to one hemisphere,and the clinical correlates are spastic hemiplegia, hemisensory deficits and often hemianopia.

AIM OF THE STUDY

1) To study the epidemiology, clinical features, etiological and risk factors of hemiplegia in children.

2) To assess the disability caused by hemiplegia in children and to follow up for improvement in disability in those children.

MATERIALS AND METHODS

Place of study

Institute of social Paediatrics,
Stanley Medical College and Hospital,
Chennai.

Study period

Two years.(September 2005- September 2007)

Study Design

Prospective cohort study.

Inclusion Criteria

All children aged 0 -12 years with hemiplegia, admitted at Institute of social Paediatrics for the first time are included.

Exclusion criteria

Children with Quadriplegia / monoplegia / diplegia are excluded.

Study Sample

All children with hemiplegia admitted at Institute of social Paediatrics during the study period were included.

Methodology

1. Patients admitted with hemiplegia at Institute of social Paediatrics during the study period were selected.

2. History regarding the type of weakness, onset, progress, involuntary movements, sensory, cranialnerves, speech, bladder / bowel, behavioural, sleep disturbances, seizures, altered sensorium were noted.

3. History suggestive of congenital / acquired heart disease, collagen vascular disease were noted.

4. Antenatal, natal, postnatal, developmental history, immunization history, family, contact history were obtained.

5. Physical examination, including general examination, neurocutaneous markers, carotid bruit, vital signs, anthropometry, detailed CNS examination was done.

6. Investigations like CT scan brain, CBC (hemogram), cardiac evaluation,lipid profile, metabolic studies, prothrombotic profile & collagen vascular disease screening, mantoux, carotid & vertebral Doppler study, CSF analysis, if possible & feasible MRI are done.

7. The patients were followed up for 6 months. The disability caused by hemiplegia was assessed at the time of presentation and 6 months later using modified Rankin scale.

MODIFIED RANKIN SCALE

Score	Description	Zero month	Six month
0	No symptoms at all.		
1	No significant disability despite symptoms, able to carry out all usual duties and activities.		
2	Mild disability, unable to carry out usual activities, but able to look after own affairs with assistance.		
3	Moderate disability, requiring some help, but able to walk without assistance.		
4	Moderately severe disability, unable to walk without assistance and unable to attend to own bodily needs without assistance.		
5	Severe disability, bedridden, incontinent and requiring constant nursing care and attention.		
6	Dead		

8. The observations and results were analysed using appropriate statistical scales.

STATISTICAL ANALYSIS

* Epidemologic variables are given in frequencies with their percentage.

* Significant difference of occurrence in epidemiologic variables are analysed using one sample chi square test.

* Difference on epidemiologic variables between acquired and congenital hemiplegia are analysed using Pearson chi square test.

* MRS score at the time of presentation and follow up for 6 months was analysed using student paired t-test.

* MRS score between0 months and 6 months was (analysed) compared using McNemars chi square test.

* Influence of epidemiological variables on MRS score was analysed using student t-test.

* Influence of etiological factors on MRS score was analysed using oneway ANOVA F-test.

* Socio demographic factors influencing the outcome was identified by multivariate logistic regression.

* $P<0.05$ was taken as significant.

OBSERVATION AND RESULTS

Total number of patients admitted in our paediatric ward during the study period was 15580. Among these cases those admitted with hemiplegia was 60 which accounts for 0.39% of total admissions. In our set up 1 out of 260 cases of admission was a hemiplegia case. Among 60 cases 30 were congenital and remaining 30 were acquired. Out of 60 cases 26 (43.3%) were in the age group of 0-3 years, 18 cases (30%) in 3-6 years age group,9 cases (15%) in the 6-9 years age group and 7 cases (11.7%) in the 9-12 years age group. Among congenital hemiplegia17 cases(56.7%) were in the 0-3 age group, 12 cases (40%) in 3-6 years age group, Only 1 case (3.3%) in more than 6 years age group. Among acquired hemiplegia 9 cases (30%) were in 0-3 years age group, 6 cases (20%) in 3-6 years age group,8 cases (26.7%) in 6-9 years age group,7 cases (23.3%) in 9-12 years age group. Maximum number of cases were in the 0-3 years age group which is statistically significant.

In our study total male children with hemiplegia were 35 (58.3%), total female children with hemiplegia were 25(41.7%). Among congenital hemiplegia number of males were 19 (63%) and females were 11 (36.7%). Among acquired hemiplegia number of males were 16(53.3%) and females were 14 (46.7%)

TABLE: I
EPIDEMIOLOGICAL PROFILE OF HEMIPLEGIA

Factors		No. of children	%	One sample chi square test
Age	<=3 yrs	26	43.3%	$\chi 2$=15.33 P=0.002 significant
	3-6 yrs	18	30.0%	
	6-9 yrs	9	15.0%	
	9-12 yrs	7	11.7%	
Sex	Male	35	58.3%	$\chi 2$=1.66 P=0.20 not significant
	Female	25	41.7%	
Place	Urban	29	48.3%	$\chi 2$=0.07 P=0.79 not significant
	Rural	31	51.7%	
Education of Father	Illiterate	6	10.0%	$\chi 2$=38.4 P=0.001 significant
	Literate	54	90%	
Education of Mother	Illiterate	10	16.7%	$\chi 2$=49.3 P=0.001 significant
	Literate	50	83.3%	
Occupation of Father	Unskilled	36	60%	$\chi 2$=2.4 P=0.12 not significant
	Skilled	24	40%	
Income of Father	<2000	50	83.4%	$\chi 2$=26.6 P=0.001 significant
	>2000	10	16.6%	

In our study about 29 cases (48.3%) were residing in the urban area and 31 cases (51.7%) were residing in the rural area. Regarding the education of parents, 6 fathers (10%) were illiterate and 54 fathers (90%) were literate. Among mothers 10 (16.7%) were illiterate and 50 (83.3%) were literate.Regarding the occupation of the father 36(60%) were unskilled and 24 (40%) were skilled. Analysing the income of the father the per capita income was <

2000 in 50 (83.4%) and > 2000 in 10 (16.6%).

TABLE: II

EPIDEMIOLOGICAL PROFILE OF CONGENITAL AND ACQUIRED HEMIPLEGIA

		Hemiplegia				Significance
		Acquired hemiplegia		Congenital hemiplegia		
		n	%	n	%	
Age	<=3 yrs	9	30.0%	17	56.7%	$\chi 2$=16.91 P=0.01 significant
	3-6 yrs	6	20.0%	12	40.0%	
	6-9 yrs	8	26.7%	1	3.3%	
	9-12 yrs	7	23.3%			
Sex	Male	16	53.3%	19	63.3%	$\chi 2$=0.62 P=0.43 not significant
	Female	14	46.7%	11	36.7%	
Place	Urban	17	56.7%	12	40.0%	$\chi 2$=1.66 P=0.99 not significant
	Rural	13	43.3%	18	60.0%	
Education of Father	Illiterate	2	6.7%	4	13.3%	$\chi 2$=0.74 P=0.38 not significant
	Literate	28	93.3%	26	86.7%	
Education of Mother	Illiterate	3	10.0%	7	23.3%	$\chi 2$=1.92 P=0.17 not significant
	Literate	27	90%	23	76.7%	
Occupation of Father	Unskilled	16	53.3%	20	66.7%	$\chi 2$=1.11 P=0.29 not significant
	Skilled	14	46.7%	10	33.3%	
Income of Father	<2000	21	70.0%	29	26.7%	$\chi 2$=8.56 P=0.05 significant
	>2000	9	30.0%	1	3.3%	

TABLE:III

CLINICAL PROFILE

		N	%
Side of Hemiplegia	Right	36	60.0%
	Left	24	40.0%
Weakness	Limbs alone	59	98.3%
	Limbs and trunk	1	1.7%
Involuntary Movements	Present	2	3.3%
	Absent	58	96.7%
Sensory Disturbances	Absent	60	100.0%
Cranial Nerve Disturbances	Present	11	18.3%
	Absent	49	81.7%
Bladder & bowel disturbances	Absent	60	100.0%
Sleep Disturbances	Absent	60	100.0%
seizures	Present	41	68.3%
	Absent	19	31.7%
Increased ICT	Present	5	8.3%
	Absent	55	91.7%

In our study hemiplegia occurred in right side in 36 cases(60%) and left side in 24 cases(40%).Among both congenital and acquired hemiplegia, 18(60%) in right side and 12(40%) in leftside in each category.The onset is acute in 17 cases(28.3%) and subacute in 13(21.7%) in acquired hemiplegia, and chronic in 30(50%) in congenital hemiplegia.

Among 60 cases 2(3.3%) had involuntary movements,11 cases (18.3%) had cranial nerve disturbances,5(8.3%) had increased intracranial tension. Seizures was present in 41cases(68.3%).No cases had sensory,bladder & bowel and sleep disturbances.

TABLE: IV

CLINICAL PROFILE

General Examination	carotid bruit	1	1.7%
	Anemia	13	21.7%
	others	30	50.0%
	Malnutrition	16	26.7%
Higher Functions	Normal	21	35.0%
	Abnormal	39	65.0%
Motor-bulk	Normal	41	68.3%
	wasting	19	31.7%
tone	Normal	3	5.0%
	Hypertonia	57	95.0%
power	2	1	1.7%
	3	50	83.3%
	4	9	15.0%
dtr	Brisk	48	80.0%
	Exaggerated	12	20.0%
Plantar Reflex	Expensor	60	100.0%
Sensory system	Normal	60	100.0%
Cerebellum	Normal	60	100.0%
Meningeal signs	Normal	50	83.3%
	Abnormal	10	16.7%
Spine & Cranium	Normal	60	100.0%

On general examination of 60 cases,13(21.7%) had anemia, 16(26.7%) had malnutrition. only 1case(1.7%) had carotid bruit. On CNS examination, higher functions was abnormal in 39 cases(65%) and normal in 21(35%).Wasting was noted in 19 cases(31.7%) .Hypertonia was noted in 57 cases(95%).Regarding the power, 50 cases (83.3%) had a power of grade3 and 9(15%) had a power of grade4. Deep tendon reflexes were brisk in 48 cases(80%) and exaggerated in 12(20%).Plantar reflex was extensor in all cases. Sensory system, cerebellar system, spine& cranium was normal in all cases. Meningeal signs of irritation was found in10(16.7%).

TABLE: V

RISK FACTORS FOR CONGENITAL HEMIPLEGIA

		Congenital Hemiplegia	
		n	**%**
AN Uteroplacental insufficiency	Present	7	23.3%
	Absent	23	76.7%
Gestation	Full term	29	96.7%
	Preterm	1	3.3%
Delivery	Normal	22	73.3%
	LSCS	5	16.7%
	Forceps	3	10.0%
Birth asphyxia	Present	23	76.7%
	Absent	7	23.3%
Birth weight	<1.5	1	3.3%
	1.5-2.0	18	60%
	2.0-3.0	10	33.3%
	>3.0	1	3.3%
Neonatal seizures	Present	4	13.3%
	Absent	26	86.7%
Neonatal Jaundice	Present	2	6.7%
	Absent	28	93.3%

In our study, in congenital hemiplegia, uteroplacental insufficiency was found in cases (23.3%).Among 30 cases of congenital hemiplegia 29(96.7%) were born by full term delivery and 1(3.3%) by preterm delivery. Regarding the mode of delivery 22(73.3%) were delivered by normal vaginal route, 5(16.7%) by LSCS, 3(10%) by forceps delivery. Birth asphyxia was found in 23 cases (76.7%) and absent in 7(23.3%). On analyzing the birth weight it was <1.5kg in 1 case (3.3%), 1.5-2kg in18 (60%), 2-3kg in10 (33.3%), >3kg in1 case (3.3%).Neonatal seizures was found in 4(13.3%). Neonatal jaundice was found in 2 cases (6.7%).

TABLE: VI

RISK FACTORS FOR ACQUIRED HEMIPLEGIA.

Acquired hemiplegia

	No. of Cases	Percentage
Heart Disease	8	26.7%
Acyanotic CHD	3	10%
Cyanotic CHD	3	10%
Acquired heart disease	2	6.7%
Vasculitis	4	13.3%
CNS TB	10	33.4%
Pyogenic meningitis	3	10%
Encephalitis	4	13.3%
Metabolic disorder	1	3.3%

Among 30 acquired hemiplegia cases, heart disease was found in 8(26.7%), vasculitis in 4(13.3%), CNS TB in 10(33.4%), pyogenic meningitis in 3(10%), encephalitis in 4(13.3%), metabolic disorder in 1 case(3.3%).

INVESTIGATIONAL PROFILE

Analysing the CT scan brain findings in congenital hemiplegia, cerebral atrophy was found in 21 cases(70%), porencephalic cyst was found in 8(26.7%), arachnoid cyst was found in 1 case(3.3%).Among acquired cases, infarct was found in 14(46.7%), haemorrhage in 1(3.3%), tuberculoma in 5(16.1%), cerebral abscess in 1(3.3%).

TABLE: VII

	Hemiplegia			
	Acquired hemiplegia		Congenital hemiplegia	
	n	%	n	%

CT Scan brain	Hemorrhage	1	3.3%		
	Infarct	14	46.7%		
	Cerebral atrophy			21	70.0%
	Porencephalic cyst			8	26.7%
	Tuberculoma	5	16.7%		
	Others	10	33.3%	1	3.3%

Analysing the hemogram of the cases, anemia was found in 14 cases (23.3%), leucocytosis in 3 cases (5%) and other findings like thrombocytopenia, thrombocytosis in 3 cases (5%).Hemogram was normal in 40 cases(66.7%).TB screening was positive in 2 cases (3.3%).Cardiac evaluation was normal in 52 cases (86.7%).Acyanotic CHD was present in 3 cases (5%), Cyanotic CHD in 3 cases (5%) and acquired heart disease in 2 cases (3.3%). Metabolic screening was positive in 1 case (1.7%).Vasculitis profile was positive in 4 cases (6.7%). In doppler study resistance in blood flow in carotid and vertebral arteries was noticed in 4 cases accounting for 6.7%.CSF analysis was abnormal in 12 cases (20%).

TABLE:VIII

INVESTIGATIONAL PROFILE OF HEMIPLEGIA

		n	**%**
CT Scan brain	Hemorrhage	1	1.7%
	Infarct	14	23.3%
	Cerebral atrophy	21	35.0%
	Porencephalic cyst	8	13.3%
	Tuberculoma	5	8.3%
	Others	11	18.3%
hemogram	Normal	40	66.7%
	Anemia	14	23.3%
	Leucocytosis	3	5.0%
	others	3	5.0%

TB Screening	Positive	2	3.3%
	Negative	58	96.7%
Cardiac Evaluation	Normal	52	86.7%
	Acyanotic CHD	3	5.0%
	Cyanotic CHD	3	5.0%
	Acquired HD	2	3.3%
Lipid Profile	Normal	60	100.0%
Metabolic screening	Normal	59	98.3%
	Abnormal	1	1.7%
Prothrombotic profile	Normal	60	100.0%
Vasculitis profile	Normal	56	93.3%
	Abnormal	4	6.7%
Doppler study	Normal	56	93.3%
	Abnormal	4	6.7%
CSF analysis	Normal	48	80.0%
	Abnormal	12	20.0%

TABLE:IX

MODIFIED RANKIN SCALE

	N	**Mean**	**Std. Deviation**	**Student Paired t-test**
MRS 0 months	60	3.73	.762	t=10.68
MRS 6 months	59	2.75	.975	P=0.001significant

The above table shows the mean score of MRS at 0 and 6 months which is statistically significant.

TABLE: X

MRS O MONTHS			**MRS 6 MONTHS**							
			1		**2**		**3**		**4**	
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
2	6	10%	6	100.0%						
3	9	15%	4	44.5%	5	55.5%				
4	39	65%			23	48%	26	52%		
5	5	8.3%					2	40.0%	3	60.0%
6	1	1.7%								

χ^2_{MC} = 222.1 P =0.001

At the time of presentation,6 cases(10%) were in the score 2, after 6 months all the 6 had improvement and had a score1. 9 cases(15%) were presented with a score3 initially and after 6 months, all had improvement with a score 2 in 5(55.5%) and score1 in 4(44.5%). 39 cases(65%) were presented with moderately severe disability, after 6 months, 23(48%) had improvement with a score 2, 26(52%) had score3. 5 cases(8.3%) were presented with severe disability with score5 and after 6 months, all had improvement,2(40%) with score 3 and 3(60%) with score4. Only 1 child(1.7%) died after an attack of hemiplegia.

TABLE: XI

		Mean	**SD**	**Student Paired t-test**
Acquired hemiplegia	MRS 0 months	3.66	1.010	t=15.7 P=0.001 significant
	MRS 6 months	2.17	.889	
Congenital hemiplegia	MRS 0 months	3.80	.407	t=5.37 P=0.001 significant
	MRS 6 months	3.30	.702	

The above table shows the mean score of MRS at 0 and 6 months for congenital and acquired hemiplegia. Among acquired hemiplegia, 6 cases (20%) were presented with mild disability and all had improvement with score1. 3 cases(10%) were presented with moderate disability and all had improvement with score2. 15 cases(50%) were presented with a score4 and after 6 months,12 cases(80%) had score2, 3 cases(20%) had score3.5 cases (16%) had severe disability initially and after 6 months all had improvement with a score 3 in 2 cases (40%) and score 4 in 3 cases (60%).

TABLE: XII

Acquired hemiplegia										
MRS O MONTHS			MRS 6 MONTHS							
			1		2		3		4	
	N	%	N	%	N	%	N	%	N	%
2	6	20%	6	100.0%						
3	3	10%			3	100.0%				
4	15	50%			12	80.0%	3	20.0%		
5	5	16%					2	40.0%	3	60.0%
6	I	3.3%								

Among congenital hemiplegia, 6 cases(20%) were presented with moderate disability and after 6 months 4(66.7%) had score 1 and 2 cases (33.3%) had a score 2.Remaining 24 cases were (80%) were presented with moderately severe disability and after 6 months all had improvement as 11(45.8%) with a score 2 and 13 (54.2%) had a score 3.

TABLE : XIII

Congenital hemiplegia										
MRS O MONTHS			MRS 6 MONTHS							
			1		2		3		4	
	N	%	N	%	N	%	N	%	N	%
2										
3	6	20%	4	66.7%	2	33.3%				
4	24	80%			11	45.8%	13	54.2%		
5										
6										

TABLE : XIV

Association between epidemiological variables and modified rank in scale

		n	Mean	SD	Significance
Age	<=3 yrs	26	.73	.78	**t=2.69P=0.05 significant**
	3-6 yrs	18	1.06	.64	
	6-9 yrs	9	1.33	.50	
	9-12 yrs	7	1.33	.52	
Sex	Male	35	.91	.67	t=0.90 P=0.37 not significant
	Female	25	1.08	.76	
Place	Urban	29	1.14	.65	t=1.67P=0.09 not significant
	Rural	31	.84	.73	
Education of Father	Illiterate	6	.83	.98	t=0.54P=0.59 not significant
	Literate	54	1.00	.68	
Education of Mother	Illiterate	10	.90	.88	t=0.41P=0.68 not significant
	Literate	50	1.00	.68	
Occupation of Father	unskilled	36	.89	.67	t=1.28P=0.20 not significant
	skilled	24	1.13	.76	
Income of Father	<2000	50	.86	.68	**t=3.27 P=0.02 significant**
	>2000	10	1.60	.52	

The improvement in MRS score was analysed with the epidemiological variables using student t-test.

TABLE: XV

Association between epidemiological variables and modified rankin scale

	No. of children	Significance	Odds ratio	95% CI	
				Lower	Upper
AGE < 3 yrs >3 yrs	26 34	.051	3.14	1.05	10.36
SEX Male Female	35 25	.846	1.130	.330	3.87
PLACE Urban Rural	29 31	.811	0.84	.223	3.23
EDUF Illiterate Literate	6 54	.753	0.62	.033	11.87
EDUM Illiterate Literate	10 50	.342	3.48	.266	45.55
OCCU Unskilled Skilled	36 24	.398	1.85	.443	7.72
INCOME <2000 >2000	50 10	.021	17.41	1.54	195.91

On Multivariate logistic regression analysis of the socio demographic factors that influence the improvement in the disability,those gained statistical significance are age of the child and income of the father.

TABLE:XVI

Association between etiological factors an and modified rank in scale

	N	**Mean**	**Std. Deviation**	**ANOVA F-test**
Hemorrhage	1	1.0000		**F=10.69 P=0.001 Significant**
Infarct	14	1.6923	.48038	
Cerebral atrophy	21	.4762	.51177	
Porencephalic cyst	8	.6250	.51755	
Tuberculoma	5	1.0000	.00000	
Others	11	1.3636	.67420	
Total	60	.9831	.70690	

The improvement in the MRS score was analysed with various etiological factors using ANOVA F-test.

DISCUSSION

In this tertiary paediatric hospital 0.39% of total admissions was due to hemiplegia. In the present study, the incidence of congenital and acquired hemiplegia is same. On analysing the incidence of hemiplegia in different paediatric age group, 43.3% occurred in 0-3years age group, followed by 30% in 3-6years age group and least in more than 9years age group(11.7%).It is found that significantly high incidence of hemiplegia was in 0-3years age group and it is true for both congenital and acquired hemiplegia. In congenital hemiplegia the mean age of seeking medical advice was 3.53years. In acquired hemiplegia mean age of presentation was 6.4years.

Study	**Mean age**
Michael et al(1995-2005)[30]	7.7 years
Chung B et al(1998-2001)[8]	5.6 years
Salih MA et al(2001-2003)[38]	1.5 years
Josiane et al(2002-2003)[26]	2.5 years
PRESENT STUDY(2005-2007)	6.4 years

The above table shows that mean age for acquired hemiplegia is similar to other studies. In our study hemiplegia is more common in males (58.3%) than females (41.7%) and it is similar in congenital and acquired hemiplegia. Male to female ratio in congenital hemiplegia is 1.7:1 which is in comparable with Peter et al[34] study(1.7:1). Male to female ratio in acquired hemiplegia is 1.1:1.

Study	Male to female ratio
Raghu Raman et al(1994-1995)[37]	1.1:1
Michael et al(1995-2005)[30]	1.07:1
Chung B et al(1998-2001)[8]	1.27:1
Salih MA et al(2001-2003)[38]	1.08:1
Josiane et al(2002-2003)[26]	0.92:1
PRESENT STUDY(2005-2007)	1.1:1

The above table shows that Male to female ratio in acquired hemiplegia is similar to other studies. The incidence of hemiplegia was found to be little higher in rural population (51.7%) than urban population(48.3%) which has no statistical significance. On analysing the education of parents, 90% of the fathers and 83.3% of the mothers were literate which is statistically significant. This shows that education of parents has a role in recognizing and seeking medical advice in hemiplegia. Significantly high incidence of hemiplegia occurred in low income group, <2000 percapita income in83.4%.

In our study hemiplegia occurred more commonly in the right side(60%) than left(40%) which holds true for both categories.

Study	Right: left
Raghu Raman et al(1994-1995)[37]	2.3:1
Josiane et al(2002-2003)[26]	1.3:1
Peter et al(1985-1998)[34]	0.86:1
PRESENT STUDY(2005-2007)	1.5:1

The above table shows right preponderance of hemiplegia which is in parallel with

other studies except for Peter et al[34] who observed slight preponderance to left side.

Anemia was found in 21.7% and malnutrition in 26.7% which is attributable to the illness and poor food intake.On analysing the clinical profile,seizures were the most common associated finding(68.3%).Incidence of seizures was more in acquired hemiplegia (76.7%) than in congenital hemiplegia (60%).

Study	**Seizures**
Michael et al(1995-2005)[30]	27%
Chung B et al(1998-2001)[8]	52%
Josiane et al(2002-2003)[26]	43.5%
PRESENT STUDY(2005-2007)	76.7%

The above table shows incidence of seizures in various studies.On analysing the CNS involvement higher functions was abnormal in 65%, cranial nerve disturbances was found in 18.3%, wasting in 31.7%,involuntary movements in 3.3%, increased ICT in 8.3%, meningeal signs of irritation in16.7%. On analysing the risk factors for acquired hemiplegia, Intracranial infections (56.7%) are the most common risk factors followed by heart disease (26.7%).Among intracranial infections, tuberculosis of CNS was found in 33.4% followed by encephalitis in 13.3%, pyogenic meningitis in10%.

Raghu Raman et al[37] observed tuberculous meningitis in 17.5%, pyogenic meningitis in 7.5%, heart disease in17.5%.Anisur Rehman et al[3] observed intracranial infections (56.09%) are the most common risk factors followed by heart disease(9.75%).Josiane et al[26] observed intracranial infections in 31% followed by heart disease in 17.4%.The above mentioned

studies showed that the most common risk factors for acquired hemiplegia are intracranial infections followed by heart disease which is at par with the present study.

Chung B et al[8] observed heart disease and Salih MA et al[38] observed hematological disorders as the most common risk factor in their studies.The higher incidence of intracranial infections in hemiplegia in the present study is attributed to the low socioeconomic status and unhygienic environment.

Study	Intracranial infections
Raghu Raman et al(1994-1995)[37]	25%
Chung B et al(1998-2001)[8]	12%
Salih MA et al(2001-2003)[38]	17.3%
Josiane et al(2002-2003)[26]	31%
Anisur Rehman et al[3]	56.09%
PRESENT STUDY(2005-2007)	**76.7%**

The above table shows incidence of intracranial infections in various studies.On analysing the risk factors for congenital hemiplegia , uteroplacental insufficiency was found in23.3%.Birth asphyxia was present in76.7% which is found to be an important risk factor.Prematurity was noticed in 3.3

Study	UP Insufficiency	Birth asphyxia	Prematurity
Uvebrant et al(1969-1978)[43]	42%	16%	24%
Peter et al(1985-1998)[34]	75.6%	24.4%	29.3%

PRESENT STUDY(2005-2007)	**23.3%**	**76.7%**	**3.3%**

.

The above table shows incidence of the risk factors for congenital hemiplegia in various studies.In our study the most common CT scan finding in acquired hemiplegia was infarct (46.7%), followed by tuberculoma (16.7%), hemorrhage(3.3%).

Chung B et al[8] observed infarct in 72% and hemorrhage in 28%. In our study the most common CT scan finding in congenital hemiplegia was cerebral atrophy (70%), followed by porencephalic cyst (26.7%) and arachnoid cyst (3.3%).

Uvebrant et al[43] observed cerebral atrophy in 36% and porencephalic cyst in 20%. Peter et al[34] observed cerebral atrophy in 5% and porencephalic cyst in 27%.In the above mentioned studies cerebral atrophy is a less common finding when compared to the present study, but the incidence of porencephalic cyst is at par with the present study.

On analysing the hemogram it was found to be normal in 66.7%, anemia in 23.3% followed by leucocytosis in 5%.Analysis of other investigations correlate with the risk factors.

On analysing the disability in acquired hemiplegia moderately severe disability was noticed in 50% ,mild disability in 20%,severe disability in 16%,moderate disability in 10%.All of them had improvement but with varying degree of disability. On analysing the disability in congenital hemiplegia moderate disability was noticed in 20% ,moderately severe disability in 80%.After 6 months almost all of them showed some degree of improvement.

The improvement in disability is better in acquired hemiplegia when compared to

congenital hemiplegia. In acquired hemiplegia, the improvement is better in mild and moderate disability group. In congenital hemiplegia, the improvement is better in moderate disability group.

On analysing the influence of epidemiological factors over improvement in disability it was found that age of the child and income of the father have significant association.The improvement is better in children more than 3 years age group and high income group had a better improvement.

On analysing the association between the etiological factors and improvement in the disability it was found that those with cerebral atrophy and porencephalic cyst had minimal improvement when compared to other factors.In our study the mortality rate was 1.7% which occurred in the acquired hemiplegia group.

Study	**Mortality rate**
Raghu Raman et al(1994-1995)[37]	4%
Chung B et al(1998-2001)[8]	18%
Michael et al(1995-2005)[30]	0%
Josiane et al(2002-2003)[26]	4%
PRESENT STUDY(2005-2007)	**1.7%**

The above table shows the mortality rate due to hemiplegia in children in various studies which is comparable with the present study.

SUMMARY AND CONCLUSION

1. The incidence of hemiplegia in this study was 0.39%.

2. The commonest age group affected was 0-3 years of age (43.3%).

3. Mean age of seeking medical advice in congenital hemiplegia was 3.53 years and mean age of presentation of acquired hemiplegia was 6.4 years.

4. Right side hemiplegia (60%) is more common than left side.

5. The most common associated CNS finding was seizures (68.3%),followed by cranial nerve disturbances (18.3%).

6. The most common risk factor for acquired hemiplegia in our study was intracranial infections (56.7%) followed by heart disease (26.7%).

7. The most important risk factor for congenital hemiplegia was birth asphyxia (76.7%).

8. The most common finding in CT scan brain in acquired hemiplegia was infarct (46.7%) followed by tuberculoma (16.7%) and in congenital hemiplegia was cerebral atrophy (70%) followed by porencephalic cyst (26.7%).

9. Majority of congenital hemiplegia (80%) and acquired hemiplegia (50%) had moderately severe disability.

10. Almost all cases showed some degree of improvement. Improvement is better in acquired hemiplegia than congenital hemiplegia.

11. Improvement in disability is better in children more than 3 years and high income group.

12. Improvement in disability is poor in cerebral atrophy and porencephalic cyst.

13. The mortality rate in children due to hemiplegia in the present study was 1.7%.

BIBLIOGRAPHY

- Abram,MD; Childhood strokes : Evaluation and management. Jax-medicine;Nov,1998.
- Aicardi J,Amsili J, Chevrie JJ.Acute hemiplegia in infancy and childhood. Dev Med Child Neurol 1969;11:162-173.
- 3.Anisur Rehman,Tahir Saeed Siddiqui,Basharat Ahmed et al ;Etiology of strokes and hemiplegia in children presenting at AyubTeaching Hospital,Abbottabad,J Ayub Med Coll Abbottabad 2006 ;18 (2).
- Black PD, ocular defects in children with cerebral palsy.Br Med J 1988;281:487.
- Bodensteiner et al.hyperhomocystinemia; a newly recognized risk factor for vscular disease.Neth J Med 45:34,1994.
- Brown JK,Van RensburgF, Walsh G, et al. A neurological study of hand function of hemiplegic children. Dev Med Child Neurol 1987;29:287.
- Byers RK. Evolution of hemiplegias in infancy. Am J Dis Child 1941;61: 915-927.
- Chung B Wong V et al; Pediatric stroke among Hong Kong Chinese subjects. Pediatrics.2004 Aug;114(2):e206-12.
- Cohen ME, Duffner PK, et al; Prognostic indicators in hemiparetic cerebral palsy.Ann Neurol.1981 Apr;9(4);353-7.
- Crothers B,Paine RS. The natural history of cerebral palsy, Cambridge: Harvard University Press,1959.
- de Veber et al;Cerebrovascular disease in children; Paediatric Neurology, Principles and Practice;Text Book, third edition.
- Devinsky O, Petito CK,Alonso DR ; Clinical and neuropathological findings in

Systemic lupus erythematosis; the role of vasculitis,heart emboli and thrombotic thrombocytopenic purpura. Ann Neurol 23;380, 1988.

- Di Tullio et al;Patent foramen ovale and the risk of ischemic stroke in a multiethnic population; Paediatric Neurology,Principles and Practice;Text Book, third edition.
- E.M.Brett,D.Scrutton; Cerebral palsy, Perinatal injury to the spinal cord; ; Paediatric Neurology, Text Book, third edition.
- FJ Kirkam; Stroke in childhood.Arch Dis Child 1999;81:85-89(July).
- Ford FR. Cerebral birth injuries and their results.Medicine 1926; 5 : 121- 194
- Freud S.Die infantile cerebrallahmung. In ; Nothnagel H,ed. Spezielle Pathologic and therapie, Vol-9,pt.3. Vienna: Holder,1987.
- Ganesan et al ; Investigations of risk factors in children with arterial ischemic stroke.Ann Neurol 2003,53 : 167-173.
- Glueck CJ, et al. Pediatric victims of unexplained stroke and their families : familial lipid and lipoprotein abnormalities. Pediatrics 1982; 69: 308-316.
- Gold AP,Hammill JF,Carter S. Cerebrovascular diseases. In : Farmer TW, Pediatric Neurology, third edition, Philadelphia; JB Lippincott 1983.
- Hagberg B et al, The panorama of cerebral palsy in Sweden.The birth year period 1979-82,Acta Paediat Scand 1989:78:283-290.
- Ingram TT.Paediatric aspects of cerebral palsy.Edinburgh,Scotland:ES Livingstone, 1964.
- James H.Menkes Jr.MD et al; Prognosis of acute hemiplegia estimated by Transcranial Doppler ultrasonography;Stroke 1988;19: 648-649.
- John H.Menkeys and Harvey B.Sarnath ; Cerebrovascular disorders;Child Neurology Text Book,sixth edition.

- John H.Menkeys and Harvey B.Sarnath ; Perinatal Asphyxia and Trauma; Child Neurology Text Book;sixth edition.
- Josiane Ranzan,Neura Tellechea Rotta et al;Ischemic stroke in children.A study of the associated alterations Arq Neuropsiquiatr 2004; 62 (3-A) : 618-625.
- Kenneth F.Swaiman MD; Paediatric Neurology Principles and Practice, Textbook; second edition; volume-2
- Lanska MJ,et al.Presentation,clinical course and outcome of childhood stroke, Pediatr Neurol 1991; 7:333-341.
- Mc Nutt SJ. Apoplexia Neonatorum.Am J Obstet 1885: 18: 73-81.
- Michael D.Bowen,MD, Christopher R.Burak MD; Todd D.Barron,MD et al;
- Chidhood Ischemic stroke in a nonurban population.J Child Neurol.2005;20(3):195-197.
- Michaelis R,Rooscheez B,Dopfer R.Prenatal origin of congenital spastic hemiparesis.Early Human Development 1980; 4:243-255.
- Molteni B et al.Relationship between CT patterns,clinical findings and etiological factors in children born at term affected by congenital hemiparesis, Neuropediatrics 1987;18:75-80.
- Nelson KB.Prenatal origin of hemiparetic cerebral palsy; how often and why? Pediatrics 1991;88:1058-1062.
- Peter Humphreys,Sharon Whitings ,and Ba' pham et al; Hemiparetic cerebral palsy; Clinical pattern and imaging in prediction of outcome;Can.J.Neurol .Sci 2000;27:210-219
- Perlstein MA,Hood PN.Infantile spastic hemiplegia, Incidence. Pediatrics 1954;14:436-441.
- Powell HF,Puck J, Fernandez F. Cerebral infarction associated with mycoplasma

pneumoniae. Pediatrics 1981;67:373-375.

- Raghu Raman,Surendran,Gupta et al; Hemiplegia; Indian Pedaitr 1997,34:55-58.
- Salih MA,Abdel-Gader AG,Hassan HH,Bahakim HM et al; Stroke in Saudi Children Epidemology,Clinical features and risk factorsSaudi Med.J 2006 Mar;27 Suppl 1;S12-20.
- Schoenberg BS,Mellinger JF,Schoenberg DG.Cerebrovascular disease in infants and children. A study of incidence, clinical features and survival. Neurology 1978:28:763-768.
- Solomon GE,Hilal SK,Gold AP,Carter S. Natural history of acute hemiplegia in childhood.Brain 1970,93:107-120.
- Tal'vik TA,Tomberg TA,Tolpats VA et al; Computerised tomographic and clinical examination of children with hemiparesis;Article in Russian; Neuropatol psikhiatr Im SS Korsakova,1987;87(3):359-365.
- Trauner DA,Manuino FL et al; Neuro developmental outcome after neonatal cerebrovascular accident. J Pediatr 1986;108:459.
- Uvebrant P et al; Hemiplegic Cerebral palsy,Aetiology and outcome; Acta Paediatr Scand 1988;suppl 345:1-100.
- Wilklund LM et al;Computed tomography as an adjunct in etiological analysis of hemiplegic cerebral palsy; Neuropaediatrics 1991;22:50.
- 45. Williams LS et al. Subtypes of ischemic stroke in children and young adults.Neurology 1997:49:1541-1545.
- Wood DH. Cerebrovascular complications of sickle cell anemia. Stroke 1978;9:73-75.

PROFORMA

SERIAL NUMBER :

NAME :

AGE :

SEX :

I.P No :

PAED NEURO No :

ADDRESS :

EDUCATION OF THE FATHER :

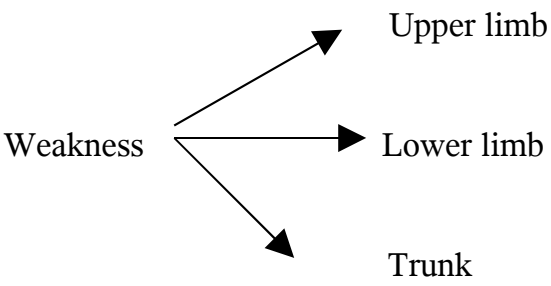
EDUCATION OF THE MOTHER :

OCCUPATION OF THE FATHER :

INCOME OF THE FATHER :

COMPLAINTS :

HISTORY :



Onset -

Progress -

H/O Stiffness / flialness of limbs -

H/O Twitching of musles -

H/O Involuntary movements –

Sensory disturbances –

Cranial nerve disturbances –

Bladder / Bowel disturbances –

Speech disturbances –

Behavioural disturbances –

H/O Altered sensorium –

H/O Seizures –

H / S / O Increased intracranial tension –

H / S / O Congenital / acquired heart disease –

H / S / O Collagen vascular disease –

H/O Ear discharge –

H/O Trauma –

H/O Dog bite –

H/O Vaccination –

H/O Exanthematous illness –

H/O Drug intake –

PAST HISTORY :

ANTENATAL / NATAL / POST NATAL HISTORY

DEVELOPMENTAL HISTORY:

IMMUNISATION HISTORY :

FAMILY HISTORY :
CONTACT HISTORY :

EXAMINATION :

GENERAL EXAMINATION :

Conscious -
Oriented –
Febrile –
Anaemia –
Jaundice –
Cyanosis –
Clubbing –
Pedal edema –
Lymphadenopathy –
Neuro cutaneous markers –
Carotid bruit –
Vital signs – PR
RR
BP
Anthropometry –

CNS EXAMINATION :

1.HIGHER FUNCTIONS :

Conscious –
Oriented –
Speech –
Intelligence –
Memory –
Behaviour –

2.CRANIAL NERVES EXAMINATION :

3.MOTOR SYSTEM : **Upper limb** **lower limb**

Rt Lt Rt Lt

Bulk –
Tone –
Power –
DTR –
Biceps –
Triceps –
Supinator –
Knee –
Ankle –
Superficial reflexes –

Corneal –

Conjunctival –
Abdominal –
Cremasteric –
Plantar –

4.SENSORY SYSTEM :

5.CEREBELLUM :

6.GAIT :

7.BLADDER :

8.SPINE & CRANIUM :

9.MENINGEAL SIGNS :

OTHER SYSTEM EXAMINATION

CVS –
RS –
Abdomen –

DIAGNOSIS :

INVESTIGATIONS :

1.CT Scan brain : → plain
→ contrast

2.Complete hemogram :
Blood hb % -

Tc –
Dc –
ESR –
PCV –
Platelet count –
Peripheral smear study –

3. Mantoux test –

4.Cardiac evaluation :
X-ray chest –
ECG –
Echo –

5.Lipid profile –
6.Serum lactate & pyruvate –

7.Urine for homocysteine –
8.Prothrombotic profile :

Anti-phospholipid Antibody –
Protein C , S –
Anti-thrombin lll –

Leiden factor –
Lupus anticoagulant –

9.Vasculitis & Collagen vascular disease screening :

ANA –

Anti-ds DNA –
Anti Smith –
RA factor –
ASO titre –

10.Carotid & vertebral Doppler study :

11. MRI / MRA :

12.CSF ANALYSIS :

13.Other Investigations

MODIFIED RANK IN SCALE

Score	Description	Zero month	Six month
0	No symptoms at all.		
1	No significant disability despite symptoms, able to carry out all usual duties and activities.		
2	Mild disability, unable to carry out usual activities, but able to look after own affairs with assistance.		
3	Moderate disability, requiring some help, but able to walk without assistance.		
4	Moderately severe disability, unable to walk without assistance and unable to attend to own bodily needs without assistance.		
5	Severe disability, bedridden, incontinent and requiring constant nursing care and attention.		
6	Dead		

KEY TO MASTER CHART

1. S.NO
2. Age
3. Sex ;Male=1; Female=2
4. Place; Urban=1; Rural=2
5. Education of Father; Illiterate=1; Literate=2
6. Education of Mother; Illiterate=1; Literate=2
7. Occupation of Father; Unskilled=1; Skilled=2
8. Income of Father;<2000=1; >2000=2
9. Side of Hemiplegia;Right=1; Left=2
10. Weakness; Limbs alone=1; Limbs and trunk=2
11. Progress; Progressive=1; Improving=2; Nonprogressive=3
12. Involuntary Movements; Present=1; Absent=2
13. Sensory Disturbances; Present=1; Absent=2
14. Cranial Nerve Disturbances; Present=1; Absent=2
15. Bladder & bowel disturbances; Present=1; Absent=2
16. Sleep Disturbances; Present=1; Absent=2
17. Seizures; Present=1; Absent=2
18. Increased ICT; Present=1; Absent=2
19. Heart Disease; Present=1; Absent=2
20. Collagen vascular Disease; Present=1; Absent=2
21. Ear Discharge; Present=1; Absent=2
22. Trauma; Present=1; Absent=2
23. Recent Vaccination; Present=1; Absent=2
24. Past History ; Present=1; Absent=2
25. AN Uteroplacental insufficiency; Present=1; Absent=2
26. AN Drug intake; Present=1; Absent=2
27. IU Infections ; Present=1; Absent=2
28. Recurrent abortion; Present=1; Absent=2
29. Natal-Gestation; Full term=1; Preterm=2
30. Delivery; Normal=1; Forceps=2; LSCS=3
31. Birth asphyxia; Present=1; Absent=2
32. Birth weight; <1.5kg=1;1.5-2kg=2; 2-3kg=3;>3kg=4
33. Neonatal Jaundice; Present=1; Absent=2
34. Neonatal seizures; Present=1; Absent=2
35. Developmental Delay; Present=1; Absent=2
36. Immunization;Given=1; Not given=2

37. Family History; Present=1; Absent=2
38. Contact History; Present=1; Absent=2
39. General Examination;Neurocutaneous markers=1;Carotid bruit=2;Anemia=3;Others=4;Malnutrition=5
40. Higher Functions;Normal=1; Abnormal=2
41. Motor-bulk;Normal=1; Abnormal=2
42. Tone ;Normal=1;hypotonia=2; hypertonia=3
43. Power ;0-5 grades
44. DTR;Absent=0;present=1;brisk=2;exaggerated=3
45. Plantar Reflex;Flexor=1;Extensor=2
46 Sensory system;Normal=1; Abnormal=2
47. Cerebellum;Normal=1; Abnormal=2
48. Meningeal signs;Normal=1; Abnormal=2
49. Spine & Cranium;Normal=1; Abnormal=2
50. CT Scan brain;Hemorrhage=1;Infarct=2;cerebral atrophy=3;porencephalic cyst=4;Tuberculoma=5; others=6
51. Hemogram; Normal=1; Anemia=2;leucocytosis=3; leucopenia=4;thrombocytopenia=5; others=6
52. TB Screening ;positive =1; negative=2
53. Cardiac Evaluation; Normal=1; Acyanotic CHD=2; cyanotic CHD =3;Acquired HD=4
54. Lipid Profile;Normal=1; Abnormal=2
55. Metabolic screening;Normal=1; Abnormal=2
56. Prothrombotic profile;Normal=1; Abnormal=2
57. Vasculitis profile;Normal=1; Abnormal=2
58. Doppler study;Normal=1; Abnormal=2
59. CSF analysis ;Normal=1; Abnormal=2
60. MRS 0 months;0-6
61. MRS 6 months;0-6