Developmental Dysplasia of Spastic Hip in Children with Cerebral Palsy in Southern India

We studied the proportion of developmental dysplasia of spastic hip in children with cerebral palsy. Children with cerebral palsy aged 2-12 years were enrolled. Migration percentage was measured on pelvic radiographs. Hip dysplasia was seen in 15 (12.7%) children.

Keywords: *Complications*, *Gross Motor Function Classification, Hip displacement.*

Developmental dysplasia of spastic hip (DDSH) in cerebral palsy often causes severe sufferings including pain [1], reduced range of hip motion with associated sitting, standing and walking problems, if not detected early [2]. The incidence of spastic hip displacement in cerebral palsy is reported to vary from 1% to 75% [3,4]. This study was planned to find out DDSH in cerebral palsy, as there is a lack of Indian data on this aspect.

This descriptive study was conducted between May 2014 and January 2015 in children presenting with clinical features suggestive of cerebral palsy at a tertiary care teaching hospital. A total of 118 children with cerebral palsy between 2 to 12 years of age belonging to Gross motor function classification system (GMFCS) grade III-V were enrolled. History and clinical examination using systematically designed forms were taken. Radiograph of pelvis and hip joint with hip abducted in supine position was taken. The degree of hip displacement was measured by Reimer's Migration Percentage (MP) [1]. Hip migration between 33-80% was labeled subluxation, and over 80% as dislocation. Gross motor function in cerebral palsy was assessed according to GMFCS a five level ordinal scale [5]. This study was approved by the Institutional ethical committee of our institute.

DDSH was found in 15 (12.71%) children (hip subluxation 14, hip dislocation 1). All these children had spastic cerebral palsy.Sublaxation was seen in children (6.2%) with GMFCS grade III, 6 children (16.6%) with GMFCS grade IV and 6 children (12.0%) with GMFCS grade V. One child (2%) with GMFCS grade V had hip dislocation. No hip subluxation or dysplasia was found in dyskinetic cerebral palsy. One child (7.1%) with spastic hemiplegia and 2 children (14.2%) with spastic diplegia had subluxation. Eleven (12.2%) of the 90 children with spastic quadriplegia had subluxation. One child with spastic quadriplegia had hip dislocation.

Hip dysplasia in cerebral palsy is due to asymmetrical activity of the muscles surrounding the hip joint [6]. Previous studies described hip subluxation in 30-60% of children with cerebral palsy which is much higher then in our study [3,7]. The probable reason for less number of DDSH in current study is probably due to the positioning of younger kids on their mothers waist with hips widely abducted.

Soo, *et al*. [8], demonstrated a linear relationship between the rate of hip displacement and a child's GMFCS level, with hip displacement seen in 90% of GMFCS V children. They also reported more hip dysplasia in spastic quadriplegia than diplegia [8]. Our study showed similar results.

Limitations of the present study are a descriptive study, and not analyzing serial pelvis *X*-ray changes. Our hip dysplasia treatment protocol involves abduction brace, Botulinum toxin, abductor tenotomy and/or varus osteotomy based on severity of hip displacement. Early detection and surgical intervention for spastic hip displacement can prevent hip dislocation and need for more invasive surgery.

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Biologicals in Juvenile Idiopathic Arthritis

We share our experience with biological agents in children with juvenile idiopathic arthritis with an aim to highlight the adverse events and response to treatment. Out of a total of 10 children treated with biological agents, one patient had serious infection, all showed good response and none had tuberculosis. High cost was limiting factor for their use.

Keywords: *Arthritis, Etanercept, Treatment, Outcome.*

Better understanding of pathogenesis of juvenile idiopathic arthritis (JIA) have led to the use of a number of biological agents in last two decades [1]. Their high cost and potential adverse effects preclude them from being used as first-line agents in developing countries. Their most important adverse effect is infection, and in India, tuberculosis is of particular concern [2]. Aim of the present study was to evaluate the adverse events and response to biological agents in patients with JIA, and to provide description of challenges in way of treatment of JIA with biological agents.

We conducted chart review of all patients diagnosed to have JIA and treated with biological agents in our center. Diagnosis of JIA was based on International League of Associations for Rheumatology Criteria [3]. They were treated as per guidelines of American College of Rheumatology [4]. Selected patients who were refractory or had responded inadequately to conventional therapy, were explained the need of biological therapy. It was prescribed on an individual basis to those who could afford it. Screening for tuberculosis was done before the initiation of therapy. Patients were followed up every 4-12 weeks and the following information was extracted from their records: demographic profile, clinical phenotype, laboratory results, therapy, response and side effects. As available data might not suffice for standard outcome criteria [5,6], response to treatment was defined as complete response (no or minimal residual symptoms, with no requirement for supplementary agents to maintain clinical remission and normal laboratory study findings), no response (no or minimal clinical benefit), and partial response (intermediate between remission and absent response) [7].

Biological agents were given in 14 patients between March 2012 – July 2015. Four patients were excluded because total duration of follow up was less than 12 weeks in two, and discontinuation of therapy (because of financial constraints) in two patients. Before commencing biological agent, all 10 patients (7 males) were on disease modifying antirheumatoid drugs (DMARDS), and 7 were on systemic steroids. Mean (SD) age of study population at onset of disease and at commencing biological agents was 4.8 (2.7) and 7.3 (3.6) years, respectively. Median (range) duration of follow up following initiation of biological agents was 11 (range 4-41) months. Clinical profile of the patients who received biological agents are summarized in ***Table* I.**

Clinical response was seen in nine out of 10 patients. Eight patients achieved complete response, while one had partial response. Median (IQR) time to show response for systemic features was 15 (15,20) days, and for articular disease was 40 (30,75) days. Five out of 7 patients were free of steroids by three months. One patient suffered from bronchopneumonia necessitating systemic antibiotics, and another had minor reactions related to tocilizumab infusion. No case of tuberculosis, malignancy or death occurred while on treatment.

Experience with these agents in Indian patients is scant. With biological agents, substantial proportion of patients were able to discontinue systemic steroids. Efficacy to the biological agents in published literature (75-85%) is comparable to the present study [8-10]. Limitation of present set of data includes small number of patients, lack of standardized outcome criteria due to retrospective study design and use of biological agents in only those who could afford. Two patients had to stop treatment due to high cost. We conclude that biological agents can be used in children who fail conventional treatment without risk of increased incidence of infection.

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