PELVIC OSTEOTOMIES OF DYSPLASTIC HIP

Essay

Submitted for Partial Fulfillment of M. Sc. Degree in Orthopaedic Surgery

Presented by

Osama Farouk Abd El-Aziz

(M.B., B.Ch.)

617168

Supervisors

Prof. Dr. Ahmed Zaki El-Sobky
Professor of Orthopaedic Surgery
Faculty of Medicine

Ain Shams University

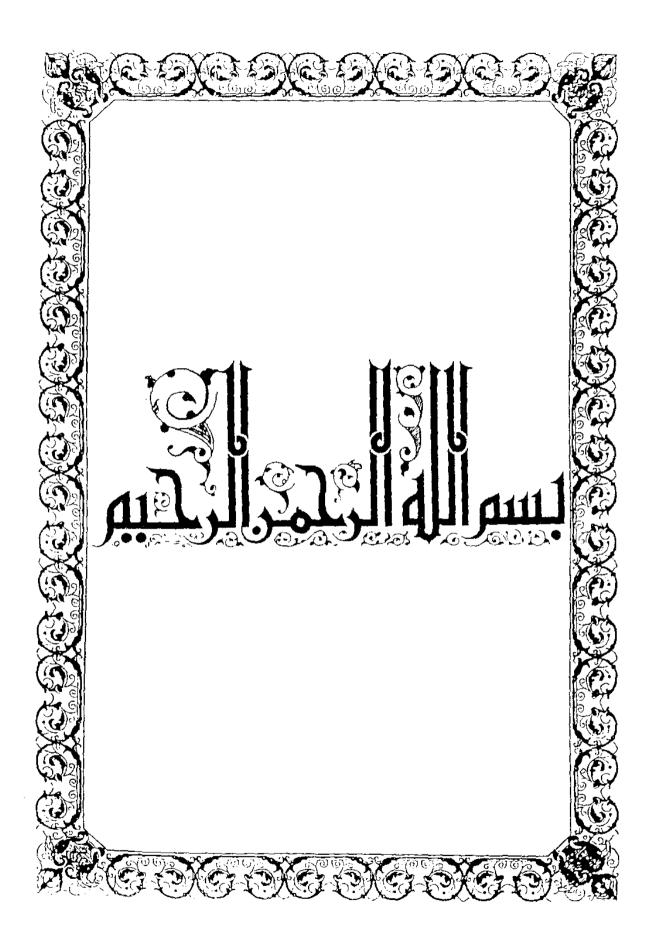
Dr. Tarek Mohamed Khalil

Lecturer Orthopaedic Surgery Faculty of Medicine Ain Shams University

Faculty of Medicine
Ain Shams University
1995

1990/1/64

92645





ACKNOWLEDGEMENTS

Words fail to express my deepest gratitude to *Prof. Dr. Ahmed*Zaki El-Sobky, Professor of Orthopaedic Surgery, Faculty of

Medicine, Ain Shams University, for offering me a lot of his support,

vast experience and fruitful criticism.

My deepest thanks to Dr. Tarek Mohamed Khalil, Lecturer of Orthopaedic Surgery, Faculty of Medicine, Ain Shams University, for his continuous help, advice and encouragement through every stage in this work.

LIST OF CONTENTS

- INTRODUCTION
- Developmental anatomy of the hip joint
- Pathology of hip dysplasia
- Radiography of hip dysplasia
- Pelvic osteotomies for dysplastic hip
[A] Acetabulum rotation osteotomies34
I- Surgery for immature pelvis
(1)Acetabuloplasty34
(a) The pericapsular acetabular osteotomy
of pemberton34
(b) Tectoplasty44
(2) Salter innominate osteotomy
II- Surgery for mature pelvis63
(1) The triple innominate osteotomy63
(2) The double innominate osteotomy
(3) The spherical osteotomies
(4) The "Dial" osteotomy of the acetabulum79
[B] Acetabulum displacement osteotomies
(1) Chiari innominate osteotomy81
(2) The dome osteotomy of the pelvis92
[C] Other pi cedures96
The shelf operation96
- Decision making choice of pelvic osteotomy in acetabular
dysplasia
- SUMMARY 112
-REFERENCES
- ARARIC SHMMARV

INTRODUCTION Central Library - Ain Shams University

INTRODUCTION

Hip dysplasia is generally defined as a condition of the hip characterized by abnormal sloping of the acetabular roof and shallowness of the acetabulum (Catterall,1982). Dysplasia may be congenital or acquired (Amstutz et al., 1991). Thus dysplasia is abnormal growth, molding or formation of the acetabulum (Amstutz et al., 1991). The deformity creates a secondary change or molding, of the head to conform to the acetabulum, generally resulting in increased anteversion of the femoral neck and abnormal muscle force with a shortened lever arm. As the child grows to an adult, these abnormalities cause fatigue and pain before there is roentgenographic evidence of degenerative arthritis. The contact area between the femoral head and the acetabulum is diminished, with the head being uncovered, leading to high stress concentration and inevitably osteoarthritis.

Hip dysplasia may also occur as a result of epiphyseal dysplasia of the femoral head and, by definition, also may occur with other conditions, eg., congenital coxa vara, Legg-Perthes disease, and slipped capital femoral epiphysis, with resultant abnormal molding of the head and acetabulum.

There is a spectrum of disease severity from mild dysplasia with slight acetabular sloping, to more severe dysplasia with a nearly uncovered femoral head, to complete dislocation. The most severe form of congenital hip dysplasia is congenital hip dislocation (Amstutz et al., 1991).

DEVELOPMENTAL ANATOMY OF THE

HIP JOINT

- (1)- Development of the hip joint.
- (2)- Normal ossification of the bones round the hip joint.
- (3)- Anatomy of the hip joint.

DEVELOPMENTAL ANATOMY OF THE HIP JOINT

(1) Development of the Hip Joint

Bone and joint development, which begins in the embryo, is regulated by genes and follows a predictable sequence. All elements of the hip differentiate in situ by growth and simple enlargement with minor changes in relationship between structures.

Four weeks after fertilization, the small lower limb buds begin on the anterior lateral body wall at the lumbar and first sacral segment levels. these buds contain mesenchyme which differentiates to cartilage, bone, synovium, ligaments, muscles and tendons (Fig. 1.1).

Mesenchymal cells form a central mass, the blastema. The central, avascular blastema forms the skeleton (Gardner, 1972). At 6 weeks the highly cellular blastema condenses to form the cartilage hip model. The primitive chondroblasts then differentiate: their nuclei separate as the cells secrete matrix material into the cytoplasm, and the club-shaped femur forms (Watanabe, 1974) (Fig. 1.2).

In older embryos, a shallow, saucer-shaped depression appears in the innominate blastema, proximal to the femoral head. This future acetabulum now forms 65 to 70 degrees of an arc (**Strayer**, **1971**) (Fig. 1.3).

The ligamentum teres and acetabular labrum appear as increased cell densities.

Hip joint cavitation begins in the late embryonic or early fetal period (Gardner, 1972) (Fig. 1.4).

The joint space forms along the femoral head periphery, gradually extending centrally (Watanabe, 1974).

When the acetabulum separates from the femoral head, it is a deep cavity almost totally enclosing the femoral head. As growth proceeds, the acetabulum depth continues to increase, but the extent to which it encloses the femoral head decreases, reaching a minimum at birth when it represents one third of a sphere (Ralis and Mckibbin, 1973).

The femoral head shape changes during prenatal development. In the embryo the femoral head represents 80% of a sphere, but decreases to 50% at birth. Postnatally the femoral head again becomes more spherical, but not to the same extent as in the embryo. Thus, femoral head coverage decreases during prenatal life from 100% at 12 weeks to a minimum of 65% at birth, after which coverage continues to increase gradually until development ceases (Ralis and Mckibbin, 1973).

The normal growth changes already described will leave the femoral head relatively uncovered during the early years of growth and



Fig. (1): Hip joint histologic features in the embryo.

- (1) Limb buds (4 weeks after fertilization).
- (2) Homogeneous skeletal blastema; muscular and osseous elements cannot be distinguished
- (3) The globular femoral heads are not congruent with the shallow acetabular depression.
- (4) The acetabulum, triradiate cartilage region, the acetabular labrum, joint space, and femoral head can be recognized in late embryonic period.

(Strayer, 1971).

this is reflected in the CE angle which increases with time from 22° to greater than 30° at maturity.

5

On the acetabular side the slope of the acetabular roof reduces with time, partly as a result of ossification of roof and partly by a change in orientation of the acetabulum. Radiologically this change in orientation is reflected by a progressive narrowing of the tear drop (Catteral, 1982).

Normal development of the hip joint following reduction of a dislocation is a progressive reduction in the slope of the acetabulum and its conversion to a dome-shape as the result of the appearance and later fusion of the secondary centers of ossification.

The tear drop progressively narrows during this time. By contrast, when this development fails to occur the acetabulum remains sloping and the tear drop remains wide. As the hip starts to decompensate there is break in Shenton's line and a progressive widening of the tear drop. A coxa magna with valgus in the femoral neck may be associated with these changes (Fig. 2).

* Abnormalities which may be associated with acetabular development:

Abnormalities may occur at a number of sites; the lateral acetabular epiphysis, the femoral head, the femoral neck, and the congruity of the hip joint itself (Catteral, 1992).

The lateral acetabular epiphysis:

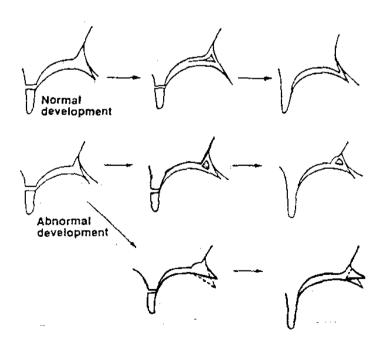
The epiphysis may fail to develop at the correct age, as a result of genetic or environmental influences such as loss of congruity of the joint. It may have been excised during the course of a limbectomy at the time of an open reduction. As a result the lateral part of the femoral head is unsupported and this produces abnormal mobility of the overlying acetabular articular cartilage. These stresses inhibit the normal ossification of the acetabular roof and result in a secondary acetabular dysplasia with an unstable lateral segment (Fig. 3). Secondarily the epiphysis may appear but fail to fuse. This again results in an unstable lateral segment. In both situations the abnormal mobility and pressures eventually result in a degenerative tear of the acetabular labrum (Dorrell and Catterall, 1986).

The femoral head and neck:

A number of changes occur in the femoral head and neck following treatment of dysplasia in the younger child. Operations on the upper femur particularly if repeated and associated with minor changes of avascular necrosis, may result in a growth disturbance in which overgrowth is the predominant feature. This leads to a coxa magna without associated enlargement of the acetabulum, producing uncovering of the femoral head anterolaterally. This overgrowth results in increasing valgus deformity of the femoral neck, this leads to long leg dysplasia.



Fig. (2): A-P roentgenogram of a patient with subluxation of the right hip and osteochondral loose bodies associated with the secondary degenerative process. The head neck junction lies 2.5 mm below the teardrop on the normal left hip. The abnormal right hip is 31 mm above the teardrop (Amstutz et al., 1991).



1. (3): The de dopment of the lateral acetabular epiphysis and its variants (Catterall, 1992).