



The aetiology of protrusio acetabuli Literature review from 1824 to 2006

Samuel VAN DE VELDE, Ramona FILLMAN, Suzanne YANDOW

From Shriners Hospitals for Children, Honolulu, HI, USA

Protrusio acetabuli is a hip joint deformity in which the medial wall of the acetabulum invades into the pelvic cavity, with associated medial displacement of the femoral head. The gradual deepening of the acetabular cavity is caused by primary idiopathic and secondary neoplastic, infectious, metabolic, inflammatory, traumatic, and genetic disorders. Due to this variety of causes, there was a considerable speculation regarding the aetiology of the hip deformity in the early literature.

Keywords : protrusio acetabuli ; acetabular protrusion ; arthrokataclasis ; hip joint deformity.

INTRODUCTION

The first recorded case of intrapelvic protrusio acetabuli, a deformity of the hip joint in which the medial wall of the acetabulum invades into the pelvic cavity, with associated medial displacement of the femoral head, was published by Adolph William Otto (37), a German pathologist, in 1824. He described in detail the pelvis of an adult woman, seen in the Natural History Museum of Breslau, Poland, of which “*the right acetabulum protrudes into the pelvis like half an orange*”. The chief characteristic of the pelvis, which has since come to bear Otto’s name, was “*the very deep insertion of both femoral heads in the acetabula, so that the floors of the latter protrude far into the pelvis and are at the same time imperfectly ankylosed*”.

Secondary protrusio acetabuli

Early literature speculated that protrusio acetabuli was a disease entity that was caused by a specific underlying process. Otto attributed protrusio acetabuli – translated by Doub (8) from the German ‘Intrapelviner Pfannenvorwölbung’ – to abnormal gout, even though the inner aspect of the acetabulum and the head of the femur were ‘smooth, both devoid of cartilaginous covering’, a description sounding like degenerative arthritis (48). In 1854, Gurlt (18) blamed acetabular fractures as the cause of the deformity, referring to it as ‘a coxalgia with acetabular fracture’. Metastatic malignancy caused a unilateral protrusio acetabuli in a 40-year-old woman, who had been operated on for a carcinoma of the breast, according to Thompson (49).

■ Samuel Van de Velde, MD, Doctoral student.

Department of Orthopaedic Surgery, Catholic University of Leuven – Faculty of Medicine, Leuven, Belgium/Massachusetts General Hospital, Boston, MA, USA.

■ Ramona Fillman, MHA, Clinical data coordinator.
Shriners Hospitals for Children, Honolulu, HI, USA.

■ Suzanne Yandow, MD, Professor.
JABSOM - University of Hawaii, Honolulu, HI, USA/Shriners Hospitals for Children, Honolulu, HI, USA.

Correspondence : Samuel Van de Velde, Department of Orthopaedic Surgery, University Hospital Pellenberg, Weligerveld 1, 3212 Pellenberg, Belgium.

E-mail : samuel.vandevelde@student.kuleuven.ac.be.

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1. Infectious diseases

In 1903, Eppinger's study (11) of four anatomical specimens attributed the deformity to a "disturbance in the acetabular development, of the nature of a chondritis or chondrodystrophy. The persistent cartilage does not fuse, but is projected into the pelvic cavity". Unaware of Otto's description of the condition 79 years before, Eppinger suggested the term 'Coxarthrolysis-Becken' or 'Chrobak-pelvis' – a birthday present to Rudolph Chrobak, professor of obstetrics and gynaecology. Even though 3 of the 4 cases he described had a history of tuberculosis, Eppinger disregarded any causal relationship.

It was Valentin and Müller (51) who established tuberculosis as an aetiological factor. Gonococcus was found at autopsy in the hip joint of a female patient (45), and despite the fact that protrusio acetabuli was a rare finding in gonococcal arthritis, Breus was convinced it was the only bacterium capable of producing all the changes and sequelae typical for protrusio acetabuli (6). Echinococcus cysts were found at operation by Réczey (43) and Trendelenburg (50). It has been stated that echinococcus involvement first occurs in the soft tissue of the joint and secondarily in the bone (28). Staphylococcus and streptococcus (12) completed the list of infectious causes of protrusio acetabuli.

2. Metabolic diseases

Several authors have stressed the role played by metabolic diseases. Although a generalised deformity of the pelvis and femur, such as ilium varus, is expected, protrusio acetabuli was found in osteogenesis imperfecta (56), ochronosis (36), acrodysostosis (31), osteoporosis (4), hyperparathyroidism, pseudo-pseudohypoparathyroidism (48) and 25% of the patients with Paget's disease (17). Verral (55) undoubtedly had not read the German literature when he described the 34-year-old woman complaining of stiff hips, caused by a "sinking in of both acetabula, so that femora are gripped round their necks". He suggested the name 'arthrokatady-sis' (subsidence of a joint) and ascribed the deformity to a localised osteomalacia. Hertzler (22) thought it seemed questionable whether 'deformi-

ties due to specific infections and to malignant growth should be admitted to this disease group'. He excluded cases due to acute osteomyelitis, tuberculosis, nerve disease and tumours and favoured the opinion of Kuliga (29). The latter disregarded Eppinger's theory of chondrodystrophy and considered the deformity osteoarthritis, causing his patient to die of haemorrhage following delivery.

3. Inflammatory diseases

Protrusio acetabuli is well recognised in rheumatoid arthritis (21). Hastings (20), in an evaluation of 694 patients with rheumatoid arthritis, reported protrusio acetabuli occurring in 14% of the cases with rheumatoid hip disease. In particular, his paper is of interest, since it bears on the significant role played by steroids in exacerbating the aetiology of protrusio acetabuli. Steroids lessen the number of osteoblasts and osteoclasts in rheumatoid arthritis most likely by inhibition of mesenchymal cell differentiation, and also affect collagen synthesis (9). The association of steroid intake and protrusio acetabuli was missing in juvenile rheumatoid arthritis. Protrusio acetabuli was found to occur in juvenile rheumatoid arthritis in 12% of the patients, associated with a greater age of disease onset and lower frequency of extra-articular manifestations (19). Ankylosing spondylitis (10), psoriatic arthritis, acute idiopathic chondrolysis (35) and Reiter's syndrome all have been verified to be aetiological factors in protrusio acetabuli, as has osteolysis following hip replacement.

4. Genetic diseases

Several genetic disorders are associated with protrusio acetabuli. Trisomy 18 (41), Ehlers-Danlos syndrome and sickle cell disease (32) all have been reported with protrusio acetabuli. Acetabular changes have been reported in the trichorhinophalangeal syndrome (13), most notably the development of protrusio acetabuli. Beals (3) found a developmental form of more severe bilateral protrusion in females with hereditary arthro-ophthalmopathy (Stickler syndrome). Interestingly, a recent study also found protrusio acetabuli in patients with

Congenital Contractural Arachnodactyly (57), an autosomal dominant disorder of connective tissue, originally described by Beals (2).

In 1978, Steel (47) linked Marfan syndrome to the development of protrusio acetabuli. It was suggested that the same abnormal mesenchymal tissue that predisposes the patients to scoliosis was related to protrusio acetabuli (56). A recent cross-sectional study demonstrated a radiographic prevalence of protrusio acetabuli of 27% in 173 Marfan patients (46). Because protrusio acetabuli resulted only occasionally in severe pain and degenerative changes in the hip joints of Marfan patients (46, 48, 58), it was advised to perform hip surgery in patients with Marfan syndrome and protrusio acetabuli on an individual basis and based on both clinical and radiographic parameters (46). A standard anteroposterior radiograph of the pelvis at first consultation to assess the presence of protrusio acetabuli and one at a follow-up consultation to rule out possible progression of the deformity, reinforced with a well-documented family history of symptomatic protrusio acetabuli, was recommended in all patients with Marfan syndrome (52). The treatment of choice is age-specific. In skeletally immature Marfan patients with the triradiate physis of the acetabulum still open, closure of the triradiate physis is recommended. For the older Marfan patients, valgus intertrochanteric osteotomy and eventually total hip arthroplasty are the suggested treatment options (53).

5. Neoplastic disorders

As mentioned earlier, metastatic malignancy was found to cause protrusio acetabuli (49). Protrusio acetabuli is a common finding in patients with neurofibromatosis (26), with a prevalence of 20%. Radiation provokes osteonecrosis and pathological fractures, but has rarely been reported to cause protrusio acetabuli (27).

Primary Protrusio Acetabuli

In 1932, Pomeranz (39) reviewed the 79 cases reported up until then in literature and added 6 of his own. He regarded the attempts by various authors to consider protrusio acetabuli a disease

entity as fallacious. He concluded that “*the condition may be produced by any process which involves essentially the acetabulum and leaves the femoral head intact [so that] the femoral head maintains its boring qualities ... the destructive process must not be too severe and must permit bone regeneration*”. This process could either be an acute infection involving mainly the acetabulum or a chronic hip lesion occurring as an incident in general osseous diseases like osteomalacia and Paget’s disease.

Schaap (44) agreed with Pomeranz on the acute form, but in the chronic type he denied the connection of protrusio acetabuli with any disease. By observing a female predominance, the large number of bilateral protrusions and the uniformity of presentation, the link with congenital luxation of the hip joint was expressed. Schaap believed the primary cause was “*a [congenitally] too deep acetabulum, resulting in a too thin acetabular floor*”. This floor could not withstand the pressure exerted on it by the femoral head, with protrusio acetabuli as effect. Rechtman (42) stated this congenitally deep acetabulum was the result of overgrowth of some elements of the acetabulum (fig 1). The acetabulum develops from three primary centers, one for each of the three main bones of the pelvis. These appear between the third and the sixth foetal month (38). The bones of the acetabulum are joined by a Y cartilage, the os acetabuli, which has a secondary center of ossification appearing at about the age of 11 years. At that age the head of the femur grows very rapidly, and there come into view the secondary centers of ossification for the acetabular rim, one for the anterior, superior and posterior parts (33). The rim gives added support to the rapidly enlarging head. If equal overgrowth occurs, a large, deep acetabulum is created. Rechtman concluded “*intrapelvic protrusion was an acquired defect superimposed on a deep acetabulum. The symptoms of discomfort began only after trauma due to stress and strain of function, infection or injury had so aggravated the condition as to cause a clinical picture of arthritis or of a derangement in the hip joint*”. He was the first to recognize the familial nature of the deformity, which has been confirmed by many (7, 30, 54).

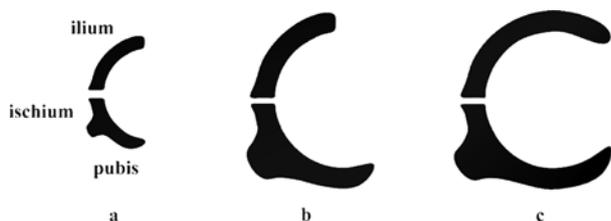


Fig. 1. — Sagittal view at the level of the triradiate cartilage of the normal and abnormal acetabular development (drawings based on Rechtman).

- a. Normal acetabulum at birth. The structure is divided into 2 equal parts, the iliac and the ischiopubic elements, in a ratio of 1:1.
- b. Normal acetabulum at later stage, when the ratio of the iliac to the ischiopubic elements is 2:3.
- c. A deep acetabulum produced by overgrowth of the iliac and of the ischiopubic elements and overgrowth of the rim, with persistence of the infantile ratio, 1:1.

Golding (16) proposed a classification 'based on radiological findings': an osteochondritis group of non-inflammatory origin in early life, following Eppinger's theory (for which the term 'arthrokatadysis' was suitable); a rheumatic group with specific (gonococcal), non-specific infections and metabolic arthritides; and finally a 'heterogeneous collection of little interest', including many varieties due to gross destructive disease, such as tuberculosis, syphilis, echinococcal disease and neoplasm.

Gilmour (15) divided the cases of protrusio acetabuli into two categories: a secondary group, accidental in nature and irregular in type, resulting from destructive diseases of the hip joint, injury of the acetabular floor or bone diseases in which softening occurs; and a primary group, caused by a 'premature acceleration of primary epiphysial ossification'. Alexander (1) granted this consideration and stated that primary protrusio acetabuli was "unrelated to any pathological process in the joint, the adjacent bone, or the Y-cartilage [and] that it is the direct result of the normal stress of weight-bearing on the normal Y-cartilage". A significant shear stress operates across the Y-cartilage during growth, resulting in medial deviation of the Y-cartilage epiphysis and producing a general pelvic deformity. He concluded that primary protrusio acetabuli develops when the correction by remodelling of the juvenile Y-cartilage 'beaking' —

associated with deeper than average acetabula — prior to fusion, is incomplete. Morton (34) observed an inward bulge in the region of the acetabula of the children of pre-puberty age, remodelled with subsequent growth. Nevertheless, the theory of 'premature fusion of the Y-cartilage resulting in protrusio acetabuli' could not withstand the test of time, given the outstanding results of closure of the triradiate epiphysis in arresting the deformity (47).

The literature now tended to focus on this primary group of protrusio acetabuli. Gilmour explained the female predominance by the fact that — together with the wider female pelvis, making the acetabular epiphysis more susceptible to deforming influences — ossification and epiphysial fusion in females preceded those in males by 1 or 2 years (15). That a wider pelvis is more subject to increased joint reaction forces is comprehensible; early epiphysial fusion, in contrary, has to be seen as a protective rather than as causative agent in the development of the deformity. Although protrusio acetabuli was generally regarded as a disease of adulthood (15), it is stated that the frequency of primary protrusio acetabuli as a bilateral condition in infants is almost as great as in adults (5). It was questioned whether primary protrusio acetabuli, almost fully present at an early age, was not in reality a childhood affliction (14). The protruded acetabulum in childhood is asymptomatic until later life when osteoarthritic symptoms indicate a need for radiographic examination — even then frequently overlooked because of the symmetry and regularity of the deformity. Hooper and Wyn-Jones (23) first described a group of teenagers in which protrusio acetabuli progressed rapidly. The steadily progressive development throughout life was revealed by the evaluation of serial roentgenograms (47); although some reports stuck to the idea that protrusio acetabuli does not increase in adults (25). The relationship between primary protrusio acetabuli and idiopathic thoracolumbar scoliosis was suggested (5), based on the findings that an epiphysial plate lesion — a condition linked to protrusio acetabuli (24) — and idiopathic thoracolumbar scoliosis may be "due to loss of cohesion of the cartilage matrix, caused by an alteration of the chemical composition of the ground substance" (40).

Table I. — Aetiology of protrusio acetabuli

<u>Primary form</u>
<u>Secondary form</u>
<i>Neoplastic</i>
Metastatic carcinoma
Neurofibromatosis
<i>Infectious</i>
M. Tuberculosis
Gonococcus
Echinococcus
Staphylococcus
Streptococcus
<i>Metabolic</i>
Paget's disease
Osteogenesis imperfecta
Ochronosis
Acrodysostosis
Osteoporosis
Hyperparathyroidism
Pseudo-pseudohypoparathyroidism
Osteomalacia
<i>Inflammatory</i>
Rheumatoid arthritis
Juvenile rheumatoid arthritis
Ankylosing spondylitis
Psoriatic arthritis
Acute idiopathic chondrolysis
Reiter's syndrome
<i>Genetic</i>
Trisomy 18
Ehlers-Danlos syndrome
Sickle cell disease
Arthro-ophthalmopathy
Trichorhinophalangeal syndrome
Congenital contractural arachnodactyly
Marfan syndrome
<i>Traumatic / Iatrogenic</i>
Acetabular fracture
Radiation-induced osteonecrosis
Osteolysis following hip replacement
Medial acetabular component placement (THA)

CONCLUSION

A review of the literature on protrusio acetabuli reveals a long history of speculation regarding the aetiology of the hip deformity, due to the widespread variety of causes (table I). An agreement in current literature is seen to divide protrusio

acetabuli into a primary idiopathic and a secondary group, caused by neoplastic, infectious, metabolic, inflammatory, traumatic, and genetic disorders.

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