Bladder Extrophy In An Adult: Management By Tensor Fasciae Latae Flap.

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Citation

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Abstract

Extrophy of the bladder is a congenital defect during the phase of organogenesis. This leads to a complex form of defect involving urogenital and musculoskeletal system. It usually is repaired during early life because of the simplicity of the procedure and in order to bring the kid in the main stream of society, but it may be neglected as in our case leading to complexities both for the patient and for the surgeon.

INTRODUCTION

This case represents a delayed presentation of bladder extrophy (at the age of 52 years) with squamous cell carcinoma of the bladder. Extrophy of the bladder is an anomaly during the organogenesis phase of intra uterine life. It is a form of urogenital defect in which there are failure of closure of the bladder and the anterior abdominal wall as well as epispadias and various other anomalies. Usually it is not complicated by malignancy, but if there is any malignant change, it is usually adenocarcinoma or transitional carcinoma of the bladder. Our case is different as it presented late and had squamous cell carcinoma.

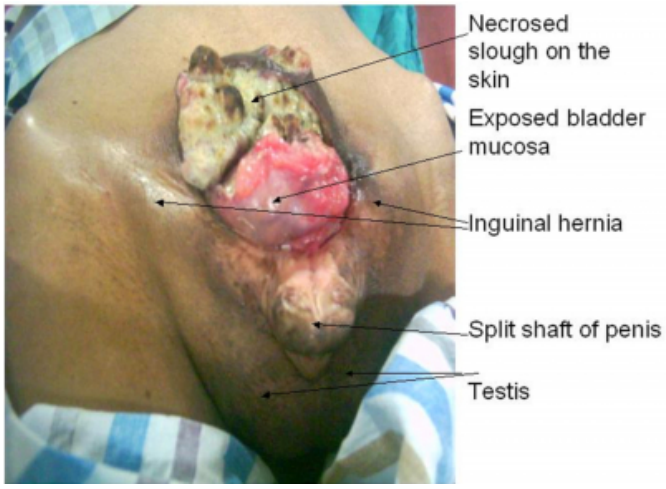
Management of these anomalies is a challenge to surgery as there are various defects to be corrected, with stabilization of the pelvis being the most vital. Because of the delayed presentation, the pelvis in this patient had stabilized due to ankylosis and the defect of the anterior abdominal wall was managed by tensor fasciae latae flap. No case is reported of such a management.

CASE REPORT

History: A 52-year-old male presented to us with the chief complaints of a lesion discharging urine in the lower abdomen since birth. As reported by the family members, he had a reddish mass protruding from the low abdomen. The shaft of his penis was split in the middle. Urine used to trickle from the mass. There was no skin cover in that region. Due to social stigma they did not report it to doctors and the kid was kept outcast (fig. 1, 2).

Figure 1

Figure 1: the lesion



{image:2}

Clinical examination: On examination, there was an ulceroproliferative lesion in the supra-pubic region. There was a granulated lesion with areas of necrosis. The posterior wall of the urinary bladder could be visualized. The shaft of his penis was split from the midline dorsally. Both testes were in the scrotum and normal. A bilateral inguinal hernia was present. Urine was seen tickling from two ureteral orifices. On palpation, the symphysis pubis was absent.

Investigations: Apart from routine blood tests, biopsy of the lesion was performed and yielded squamous cell carcinoma of the bladder. CECT of the abdomen was done to know

about the infiltration of the tumor.

Steps of the operation: The abdomen was opened through a midline incision with peritumoral incision keeping a 1cm skin margin (fig. 3). The ureters were separately identified. Radical cystectomy was done. The ureters were separately anastomosed to an ileal conduit by Leadbetter's technique2. Ileostomy was created at a premarked site in the right iliac fossa. The defect in the abdominal wall was covered by partial closure with prolene sutures and the rest of the defect was covered by a right tensor fasciae latae (TFL) flap as the patient had a stable pelvis (fig. 4).

{image:3}

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Postoperatively the patient was allowed oral nutrition after 48 hours. TFL flap release was done after 3 weeks (fig. 5). The patient was advised to attend postoperative radiotherapy. Histopathological reports revealed moderately differentiated squamous cell carcinoma with muscle invasion (extending half thickness of muscle).

{image:5}

DISCUSSION

PATHOGENESIS OF EXTROPHY

Several theories have been propounded but none universally accepted. It is accepted that an error in embryogenesis is the cause of extrophy. It is the appearance, timing, and function of cloacal membrane that are believed to play a role in the development of extrophy. [3]

In 1962, Marshall[1] proposed the persistence of the cloacal membrane during fetal development as the cause of extrophy.[7] Johnston proposed premature disappearance of the cloacal membrane as the cause of extrophy.

COMPLICATIONS

Possible complications include cystitis cystica and/or glandularis, squamous metaplasia, adenocarcinoma and squamous cell carcinoma (rare)[2]. Males have a broad and shortened penis which is defect dorsally and may have a dorsal chordee. In females the mons pubis is absent.

TENSOR FASCIAE LATAE FLAP ANATOMY

The tensor fasciae latae flap is a myocutaneous flap based on the tensor fasciae latae.[4,5,6]. Origin: The TFL is a small thin flat muscle that takes its origin from the anterior 5cm of the outer edge of the iliac crest, the anterior superior iliac spine and the iliac tubercle.

Insertion: Its fibers run downwards and backwards and are inserted in the iliotibial tract over the lateral aspect of the knee. Its size is 5 x 15 inches.

Vascular supply: It has a single dominant vascular pedicle (Type I). The main vascular supply is from the ascending branch of the lateral circumflex artery, which is a branch of the profunda femoris artery.

Nerve supply: is by the superior gluteal nerve.

The flap may be raised as standard or extended flap (standard flap dimensions: 10 x 20cm; extended flap dimensions: 15 x 40cm.

FLAP DISSECTION

The anterior border of the flap is marked by drawing a line from the anterior superior iliac spine to the lateral condyle of the tibia. The greater trochanter marks the posterior boundary. Superiorly this flap can be taken from the iliac crest; inferiorly it should stop within 5 to 8 cm from the joint

The junction of the proximal and middle third is often the site of a perforator that pierces the tensor fascia lata. We mark this and incorporate this point in the flap. The TFL perforator can be a lifeboat in the rare circumstance when the distal perforators are of poor quality or injured during dissection. The junction of the middle and distal third is marked and is also incorporated into the flap. This area defined by the middle third of the axis line generally encompasses all perforating vessels.

For elevation of the flap, the skin incision is started at the lower border and continued at the medial and lateral borders. The wound is then deepened and the deep fascia is sutured to the skin to prevent shearing movements. The flap is then elevated off the vastus lateralis in a relatively avascular plane, upwards and subfascially. The vascular pedicle is identified approximately 8-10cm below the anterior superior iliac spine, as it enters from the medial aspect. The flap is rotated to cover the defect either under a tunnel or directly in severe cases. The deep fascia is fixed by Vicryl 3/0 interrupted sutures and the skin is closed by nylon sutures. The donor site is closed either primarily or with skin grafting.

OUR CASE

Our case was different in the sense that, apart from the late presentation of extrophy, our patient had squamous cell

carcinoma which is rare. Moreover, his pelvis had adapted so that he could walk only with a waddling gate. As the pelvis had stabilized itself in a compromised way, the accepted method of correction of extrophy ("Mitchell's technique of single staged operation" or "complete primary extrophy repair") was not done [1]. Instead, after constructing the bladder reservoir, the anterior abdominal wall defect was closed by tensor fasciae latae flap which was adequate.

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