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Case Report

Diabetic cystopathy due to detrusor muscle dysfunction in a child with type I diabetes mellitus

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ABSTRACT

The word diabetic cystopathy (DC) refers to lower urinary symptoms due to diabetic neuromyopathy and is common in adults while very unusual in children with diabetes mellitus. This case report seeks to delineate a distinctive case concerning a 9-year-old boy who was diagnosed with DC due to detrusor muscle dysfunction confirmed by urodynamic studies, induced by type 1 diabetes mellitus of four years duration. DC should be considered in children with long standing urinary symptoms without urinary tract infection in type 1 diabetes mellitus even if it is rare condition.

Keywords: Children, Diabetic cystopathy, Diabetes mellitus, Urodynamic studies

INTRODUCTION

Diabetes mellitus affects various systems in the body as a complication including urological system. Bladder dysfunction in diabetes manifest with bladder symptoms that range from overactivity to underactivity and its prevalence is 25-87% in adults.[1] The overactive bladder symptoms are mainly frequency with or without incontinence, urgency and nocturia. It is caused by compensatory bladder hypertrophy and associated myogenic and neurogenic alterations due to hyperglycemia induced polyuria.[2] As the disease progresses, the underactive bladder symptoms get predominates due to decompensation of bladder muscle resulting into atonic bladder caused by hypo-contractile detrusor muscle. The underactive bladder symptoms are slow urinary stream, straining, hesitancy, along with a sensation of incomplete bladder emptying.[3]

The word diabetic cystopathy (DC) was first framed by Frimodt Moller in 1976 and it refers to lower urinary symptoms due to diabetic neuromyopathy.[4] It is characterized by decreased bladder sensation, increased bladder capacity and impaired bladder emptying with resultant increased post void residual volume due to damage of visceral afferent fibres in the bladder wall.[5] The urodynamic evaluation is the cornerstone for the diagnosis of DC.

DC is very common in adults while very unusual in children with diabetes mellitus. This case report seeks to delineate a distinctive case concerning a 9-year-old boy who was diagnosed with DC due to detrusor muscle dysfunction induced by type 1 diabetes mellitus of four years duration.

CASE REPORT

A 9-year-old boy born by a 2nd degree consanguineous marriage and a known case of type 1 diabetes mellitus since last 4 years of age came with complaint of dribbling of urine especially during night, urgency, and increased frequency of urine 15 to 16 times a day since last 1 year along with incomplete voiding, needing more time to void

and reduced bladder sensation since last two months. There was no history of dysuria. His height and weight were at 25th and 10th centile respectively. On examination bladder was distended and rest systemic examination including central nervous system was normal. Digital rectal exam showed normal anal tone and there was intact bulbocavernosus reflex. Ultrasonography of abdomen and pelvis showed trabeculated thickened bladder wall with significant vesical residue (pre void 458 cc while post void residue 246 cc) (Figure 1).

His glycated haemoglobin (HbA1c) was 9.92% and serum electrolytes, renal function tests and urine for micro albuminuria were normal. Urine culture showed no organisms. Cystourethrogram showed pear shaped, trabeculated bladder outline suggesting longstanding bladder outlet obstruction or neurogenic bladder. Uroflowmetry showed low flow pattern and protraction. (voided volume was 158 ml, maximum flow rate was 12.3 ml/sec, average flow rate was 5.6 ml/sec and voiding time was 28 sec). Urodynamic study showed During filling phase 261 ml of saline was infused via a filling line at slow fill of 2 to 10 ml/min. First desire, normal desire and strong desire were noted at 118 ml, 200 ml and 255 ml fill respectively. There were no abnormal detrusor contractions. The compliance was reduced. Functional capacity of the bladder was normal. Bladder sensation was also normal. During the voiding phase the patient failed to void in spite of profound abdominal straining and significant intermittency. The detrusor pressure did not rise above 20 cm of water. The study indicated detrusor underactivity most likely due to detrusor myopathy associated with juvenile diabetes (Figure 2). Magnetic resonance imaging (MRI) brain and spinal cord were normal. He was put on basal bolus insulin therapy along with oral bethanechol (0.2 mg/kg/day three times a day). Patient was trained to perform clean intermittent self-catheterization every 3-4 hours and pelvic floor exercises was advised.

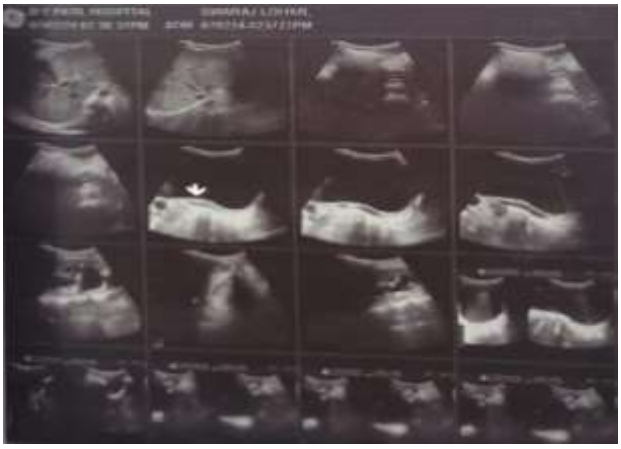

Figure 1: USG abdomen and pelvis scan showing trabeculated thickened bladder wall.

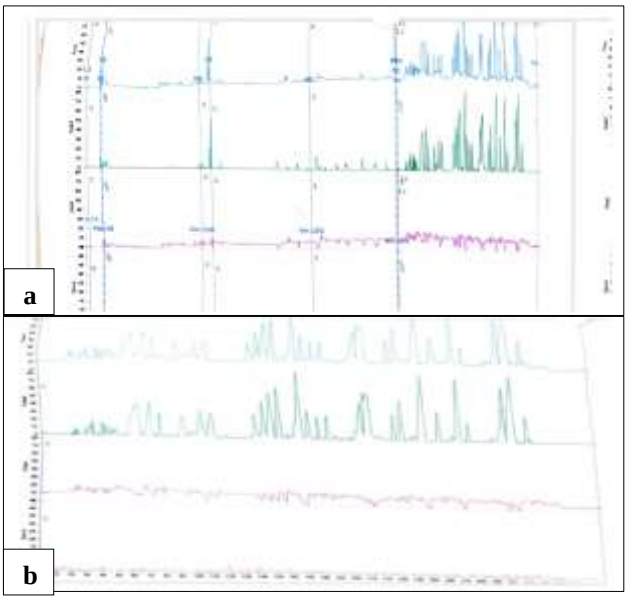

Figure 2 (a and b): Urodynamic studies graph showing detrusor muscle underactivity.

DISCUSSION

Diabetes mellitus can affect bladder function through a mix of myogenic and neurogenic mechanisms. Bladder dysfunction which commonly present as diabetic cystopathy, leads to a decrease in emptying efficiency over time as the disease progresses.[6] This phenomenon is commonly attributed to autonomic neuropathy via axonal degeneration and segmental demyelination.[7]

The pathophysiology of DC may stem from five potential sources: the dysfunction could be because of alterations in muscarinic receptor densities, especially with an increase in M2 receptors; direct neural damage from high blood sugar levels due to glycosylated end products can speed up microvascular disease, impair endothelial function, and negatively impact nitric oxide release; sorbitol production in a high blood sugar environment could lead to elevated reactive oxygen species, causing oxidative stress within muscle cells; elevated blood sugar levels can also lead to an increase in diacylglycerol production, activating protein kinase. This can impact the transcription of different connective tissue components such as type IV collagen, contractile proteins, and fibronectin, while also causing harm to nearby endothelial and neural cells. There might be a decrease in NGF, which was previously thought to protect bladder activity.[4]

Over 50% of men and women with diabetes have bladder dysfunction, its risk increases with the duration of diabetes and rarely observed in children. In 2001, Hoebeke and colleagues conducted a study involving 1000 participants, identified 4% of children with increased bladder capacities and hypo contractility.[8] Although our patient is school going with the duration of diabetes for last 4 years but still,

he developed bladder symptoms since last one year and diagnosed as DC due to detrusor muscle myopathy.

Assessing patients with DC involves standard urologic assessment, urinalysis, uroflowmetry, post void urine measurement, urine culture, and neurological examination, renal function. Urodynamic studies are cornerstone for diagnosis. The complications arising from bladder dysfunction include asymptomatic bacteriuria, symptomatic urinary tract infection, vesicoureteral reflux, hydroureteronephrosis, pyelonephritis, nephrolithiasis and urosepsis.[9]

Management goals focus on addressing symptoms, preventing, and treating urinary tract infections, ensuring proper bladder emptying and tight glycemic control. To achieve these goals, management strategies are categorised into three main classes namely behavioural, pharmacological, and surgical. Pelvic floor physiotherapy and biofeedback have been used to successfully treat children with dysfunctional voiding. Patients should be encouraged to void frequently every 2-4 hour and to use double voiding technique, Crede's manual compression of lower abdomen can also be useful in facilitating urine. Parasympathomimetic agents, including direct muscarinic receptor agonists or anticholinesterases, have been used in increasing bladder contractility. Bethanechol and carbachol are most used drugs. Bladder outlet surgeries have high risk of failure in men with detrusor underactivity not dependent on catheter.[3,10]

CONCLUSION

Although diabetic cystopathy due to detrusor muscle dysfunction is common in adults with diabetes but it should be considered in children with long standing urinary symptoms without urinary tract infection in type 1 diabetes mellitus even if it is rare condition.

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REFERENCES

1. Frimodt-Moller C. Diabetic cystopathy: epidemiology and related disorders. Ann Intern Med. 1980;92:318-21.
2. Daneshgari F, Liu G, Birder L, Hanna-Mitchell AT, Chacko S. Diabetic bladder dysfunction: current translational knowledge. J Urol. 2009;182:S18-26.
3. Golbidi S, Laher I. Bladder Dysfunction in Diabetes Mellitus. Front Pharmacol. 2010;1:136.
4. Hill SR, Fayyad AM, Jones GR. Diabetes mellitus and female lower urinary tract symptoms: A review. Neurol Urodynamics. 2007;27(5):362-7.
5. Kebapsi N, Yenilmez A, Efe B, Entok E, Demirustu C. Bladder dysfunction in type 2 diabetes patients. Neurourol Urodyn. 2007;26:814-9.
6. Brown JS, Wessels H, Chancellor MB, Howards SS, Stamm WE, et al. Urologic complications of diabetes. Diabetes Care. 2005;28:177-85.
7. Lifford KL, Curhan GC, Hu FB, Barbieri RL, Grodstein F. Type 2 Diabetes Mellitus and Risk of Developing Urinary Incontinence. J Am Geriatr Soc. 2023;53(11):1851-7.
8. Hoebeke P, Van Laecke E, Van Camp C, Raes A, Van De Walle J. One thousand video urodynamic studies in children with non-neurogenic bladder sphincter dysfunction. BJU Int. 2001;87:575-80.
9. Nanigian DK, Keegan KA, Stone AR. Diabetic cystopathy. Current Bladder Dysfunction Reports. Springer Science and Business Media LLC. 2007;2(4):197-202.
10. Sasaki K, Yoshimura N, Chancellor MB. Implications of diabetes mellitus in urology. Urol Clin North Am. 2003;30(1):1-12.

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