ORIGINAL RESEARCH PAPER

Urology

STUDY ON "MANAGEMENT OF VESICOURETERAL RELFUX IN NEUROGENIC BLADDER

KEY WORDS:

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ABSTRACT

Vesico-ureteric reflux (VUR) is a major cause of morbidity in patients with neurogenic bladder dysfunction. If it is not therapeutically intervened, it leads to major complications like hydronephrosis and ascending urinary tract infections (UTI), which further lands up in progressive renal deterioration and renal failure. The foundation of managing reflux in these neurogenic bladders is to maintain low bladder pressures. Previous studies have focused mainly on using intermittent catheterization and medications to lower the bladder pressures. Alternatively, the patients that are in need of bladder augmentation can have spontaneous resolution of their reflux with the resulting increase in capacity. Surgical intervention is called for when bladder capacity is adequate and the reflux persists or if it is part of a larger operation to increase capacity and to manage outlet resistance. In some instances, reimplantation is necessary because the ureters interfere with the bladder neck procedure. Aside from open and robotic surgical intervention the use of endoscopic injectable agents is beginning to become more popular.

INTRODUCTION:

Neurogenic bladder (NGB) affects over 90% with spinal cord injury (SCI), 50-80% with multiple sclerosis (MS) and about 95% patients with spina bifida. Neurologic conditions in children leading to neurogenic bladder include congenital neural tube defects (myelomeningocele, lipomeningocele, sacral agenesis, and occult lesions causing tethered cord). Acquired causes such as spinal cord tumours or trauma or sequelae of transverse myelitis are less frequent.1 Up to a third of children with neurogenic bladder have VUR. Those affected may have detrusor external sphincter dyssynergia and/or poor bladder compliance, leading to high bladder storage and voiding pressures. Vesicoureteral reflux (VUR) is a significant risk factor for pyelonephritis and renal scarring. VUR can occur through a defective ureterovesical junction (UVJ) or an overwhelmed normal UVJ mechanism such as in bladder dysfunction of congenital, acquired, or behavioural aetiology. A commonly accepted view is that VUR in neurogenic bladder represents a secondary type of reflux whereby the primary cause is elevated bladder pressures rather than a defective UVJ.[2-4]

Other theories regarding etiopathogenesis of this conditions is pointing towards chronic infections, which may have tremendous influence in weakening valve mechanism, and anatomic disruptions such as bladder trabeculations and diverticula occurring near the ureteral orifice. The latter is postulated to prevent the passive compression of the submucosal ureteral tunnel, which plays a major role in reflux prevention. Dysfunctional voiding can also perpetuate reflux, making secondary VUR of all grades less likely to resolve with age compared to primary VUR.[5-7]

Vesico-ureteric reflux (VUR) is a major cause of morbidity in patients with neurogenic bladder dysfunction. If it is not therapeutically intervened, it leads to major complications like hydronephrosis and ascending urinary tract infections (UTI), which further lands up in progressive renal deterioration and renal failure.8,9Over the past two decades, there have been major advances in the understanding of the anatomy, physiology and pathophysiology of the bladder and neurologically impaired bladder. These advances stem from a dedication to basic science and clinical research investigation of these realms of science.

What has been learnt from this vast amount of study is the application of new surgical techniques and medical interventions to assist both adults and children in maintaining normal bladder and urinary sphincter function and urinary continence. Some of these surgical techniques are minimally invasive, while others require more invasive modalities.10 The loss of supra-spinal control leads to neurogenic detrusor over-activity (NDO), thus causing urinary incontinence, and detrusor sphincter dyssynergia (DSD), which results in elevated bladder pressure during the storage and voiding phases. NDO, DSD, and high pressure often lead to structural bladder damage, vesicoureteral reflux (VUR), upper urinary tract dilation (UUTD), and renal insufficiency. Therefore, the management and treatment for NB should protect upper urinary tract (UUT) function, achieve urinary continence, improve the quality of life, and restore LUT function.[11]

MATERIALS AND METHODS: We included about 40 confirmed subjects aged between 12-74 years who were having VUR due to neurogenic bladder. All these subjects were evaluated for the detailed history, physical examination and series of investigations which included urodynamic investigations, upper urinary tract imaging & X-ray digital fluoroscopy. The initial evaluation is essential to determine the therapeutic scheme for long-term treatment and follow-up. A urodynamic investigation is the only method that can objectively assess the function and dysfunction of the LUT. It is essential to describe the LUT status in patients with NLUTD. The quality of the urodynamic recording and its interpretation must be ensured. The urodynamic tests are performed and all urodynamic findings must be reported in detail as per international continence society (ICS) technical recommendations and standards. Upper Urinary Tract Imaging: In patients with NLUTD, elevated intravesical pressures can be transmitted to the UUT causing hydronephrosis (HN) and ureteral dilation (UD), which are referred to as UUTD. Ureteral obstruction at the bladder wall is another cause for UUTD, but is less of a concern. UUTD or deterioration can lead to chronic renal failure. Therefore, evaluation and protection of UUT function is extremely important in the management of NB.

Currently, the most common method used to detect HN and UD is ultrasonography (US). X-ray digital fluoroscopy was

used for identifying VUR and was graded from I–V based on the international reflux grading system (IRGS). Here in our study we have mainly focused on subjects with VUR. VUR are graded from I-V based on IRGS, hydronephrosis and ureteral dilatation was graded into 1-4 and if ureteral obstruction was present (left/right) is noted. Renal function was assessed based on eGFR (estimated glomerular filtration rate using Cockcroft-Gault formula) was considered normal if GFR was >70mL/min in single or both kidneys, similarly renal insufficiency (RI) was diagnosed as compensatory RI if total GFR is >50mL with Serum Creatinine <178 mmol/L and Decompensation RI, If GRF is <50 mL with serum creatinine >178 mmol/L.[12-14]

MANAGEMENT MODALITIES:

Combined Oral Medications with Clean Intermittent Catheterization: Oral antimuscarinic (anticholinergic) medications for NGB have been a mainstay of medical therapy for decades in both adult and pediatric patients with SCI, MS or spina bifida. They are the most widely cited treatment for NGB among international guidelines. Antimuscarinics and clean intermittent catheterization (CIC) were the best option for bladder management in spinal cord injury with detrusor over activity. Muscarinic receptor antagonists have traditionally been viewed to act by binding to receptors on the detrusor muscle preventing acetylcholine release from parasympathetic nerves. These receptors are now known to be located both on the detrusor and the mucosa and the newer pharmacotherapeutic agents have been shown to bind to both receptor sites. CIC initiation is clinician-dependent and may start in the new-born period. In fact, CIC and medical therapy alone is associated with up to a 30%–50% resolution of VUR within 2–3 years.[15-16]

Ureteral reimplantation: This procedure alone is an acceptable intervention for nonaugmented patients when there is adequate bladder capacity and compliance. Surgery in these bladders can be technically difficult and bloody, with an increased risk of ureteral obstruction especially with intravesical techniques. The reduction of bladder pressures in patients who undergo reimplantation is felt to be essential for favourable outcomes, and for this reason CIC and anticholinergics are commonly implemented following surgery.

More recently the use of alpha blockers have shown some promise in reducing bladder pressures via 2 mechanisms. As expected a reduction in outlet resistance lowers intravesical pressures but there has been evidence that vesical volumes can be increased by nonselective alpha blockers. With regard to ureteral reimplantation technique, several approaches exist. The Politano-Ledbetter technique was the first described for management of primary VUR and was the predominant technique performed prior to the description of the cross-trigonal technique by Cohen in 1977.[17,18]

Endoscopic anti-reflux procedures: Despite the very high success rates of open ureteral reimplantation in primary VUR, endoscopic antireflux surgery such as with subureteral injection of Deflux has been popularized due to relative ease of use and substantially reduced morbidity. Its role in secondary VUR is evolving, though it has drawn appeal for similar reasons. Success rates for these procedures in neurogenic bladder patients has been reported at anywhere from 53% to 86%, which are lower than success rates in primary VUR.[19-20]

Augmentation cystoplasty: In patients who have (Augmented Bladders) high-grade reflux in the setting of a dysfunctional neurogenic bladder, augmentation cystoplasty is typically performed with ureteral reimplantation. It has been postulated that persistence of reflux post operatively in these patients may simply be a measure of the quality of the augmentation surgery, similar to the persistence of incontinence following these surgeries. The downside of an augmentation-only method of management is that it does not account for other mechanisms that may factor into VUR in these patients such as UVJ incompetence from surrounding distortions (trabeculations or diverticula) or from chronic infection. After enterocystoplasty, bacteriuria is common and risk of infection remains significant. It is unclear whether these factors play a role when VUR persists after augmentation surgery. Long-term complications of augmentation cystoplasty are also not insignificant, and include reduced bone mineral density and osteoporosis along with bladder calculi. Bladder rupture is another risk that can potentially lead to death in these patients. Decision to proceed with this major surgery should be weighed heavily against the risks and benefits.[6]

CONCLUSIONS: Compared to primary VUR, secondary VUR in neurogenic bladder patients is less likely to spontaneously resolve, less likely to be cured with antireflux surgery independent of technique or surgical approach. The common denominator that likely separates this patient population from achieving the success rates of the primary VUR population is suboptimal bladder dynamics. Whether reflux in neurogenic bladder is addressed by CIC/anticholinergics, selective and nonselective alpha blockers, ureteral reimplantation, endoscopic surgery, or bladder augmentation alone, the key to improved outcomes appears to be optimization or preservation of adequate bladder capacity and compliance. The risks and benefits of management options must be weighed with consideration of each individual case.

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