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Title: THE OVERDISTENDED AND HYPOCONTRACTILE BLADDER DUE TO MASSIVE URINE OUTPUT

We reviewed two female cases with the overdistended, hypocontractile bladder secondary to massive urine output. Their bladders were underactive bladder and a large volume of residual urine was observed in both cases without any obstruction in the lower urinary tract.

Case 1:

A 32-year-old pregnant woman was referred us for her large underactive bladder with masive polyuria. Her bladder was aufully extended with a residual volume of 500-1000 ml. Her urinary output reached 15 to 20 litres a day. She noticed herself that urine volume bagan to increase in the early period of the pregnancy. Then, she consulted with a doctor and was diagnosed as neurogenic bladder dysfunction. As the treatment, clean intermittent catheterization (CIC) was suggested to do once a day.

Examinations with vasopressin and desmopressin revealed that this condition was induced by idiopathic diabetes inspidus. Treatment was started with daily administration of 10 μg of DDAVP(1-desamino-8-D-arginine vasopressin), and more frequent CIC, 5 times a day, was instructed. After three months of these treatment, she delivered a healthy baby. Her urine volume markedly decreased to 15,00 ml/day or less and residual urine volume also reduced to less than 100ml. On the pressure-flow study, she resumed normal bladder sensation and detrusor contraction. No obstructive pattern was recognised in the Shafer's nomogram. With continuous use of DDAVP, daily urine output has been well-controlled wihin 1,500 or 2,000 ml/day for these 9 months. Bladder compliance was improved from 16.7 to 37.3 ml/cmH2O, and detrusor pressure increased from 45 to 55 cmH2O on the pressure-flow study. However, ultrasonic estimation of bladder tissue weight was 65 and 86.9 g[1], suggesting that bladder hypertrophy was still continuing.

Case 2:

A 37-year-old woman presented with underactive bladder with residual urine volume of 500-800ml and massive urine output of 4,000-6,000 ml/Day. She had been diagnosed as neurogenic bladder associating with psychogenic polydipsia for 12 years in the previous hospital, where she was adviced to do CIC. She was sometimes annoyed with overflow incontinence, and an indwelling catheter was the solution for the trouble at night.

With water deprivation test, she was considered to have psychogenic polydipsia and started to ask a psychiatrist for counselling. The daily fluid intake was restricted to a maximum of 2,000 ml/day, which resulted in a decrease of urine volume around 2,500 to 3,000 ml/day. After 3 months, she resumed normal bladder sensation. Detrusor pressure increased from22 to 28 cmH2O on pressure-flow study. No obstruction was recognised in the Shafer's nomogram. Further followup observation was impossible because of her psychological reasons.

Discussion:

Overdistended, hypocontractile bladder is sometimes seen in patients with chronic urinary retension due to lower urinary tract obstruction.

Chronic and massive urinary overload tends to dilate the urinary tract partly or entirely. Hydronephrosis and/or urinary retention in the patients with congenital nephrogenic diabetes insupidus has been reported without apparent urinary tract obstruction[2]. On the other hand, the bladder dysfunction in patients with idiopathic diabetes inspidus is reported to be seen in the Wolfram syndrome. In this syndrome, the urinary tract involvement is common; upper urinary tract dilatation and bladder dilatation with atonic type of bladder dysfunction. A recent study, however, suggests that the presence and duration of diabetes inspidus does not correlate with the type of bladder dysfunction [3].

Patients with psychogenic polydipsia are involuntarily forced to take much water and a marked increase of urine output affects their urinary tract both mechanically and functionally.

In our cases, the detrusor decompression was undertaken by normalization of urine output and CIC. Results were a good resumption of normal bladder sensation and detrusor contrastility. The reason, however, why bladder hypertrophy still remained in the first case is not clarified.

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