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LOCKED-IN SYNDROME AND VOIDING

Aims of Study

The Locked-In Syndrome (LIS) is due to corticobulbar and corticospinal pathway interruption for cerebrovascular accident, trauma, severe motor neuropathy (e.g. Guillain Barrè syndrome) and pontine myelinolysis. These patients are mute and quadriplegic but fully conscious and the only way to communicate is with eyes and eyelids movements: blinking and gaze. Characteristic of these patients is that the level of the consciousness is maintained as well as the other cognitive functions.

Previously we reported 5 cases of LIS, now with the increasing consistency of our LIS population we can better comprehend and discuss the voiding behavior in LIS patients.

The aim of this study is to evaluate voiding dysfunction in LIS patients and to correlate urodynamic and clinical data with voiding rehabilitation after catheter removing.

Methods

We evaluated retrospectively the charts of 8 patients with locked-in syndrome who were referred to our urodynamic unit for urinary incontinence in the period between January 2000 and 2002. All the tetraplegic patients were vigil at the time of the studies and could communicate with eyelid movements. Urodynamic evaluation included filling cystometry and urethral sphincter electromyography with surface electrodes. Cystometry was performed transurethrally, filling with water at 20 ml per minute. The follow up consists in daily urine volume monitoring, clean intermittent catheterisation if necessary, pad test and post-voiding residue ultrasound evaluation.

Results

We have studied 8 patients (4 males and 4 females) 50 years old (range 16-77). The Locked-in syndrome was related in 7 cases to basilar artery thrombosis and in 1 case to cranial trauma. No other urological problems were present at the time of the study and the urodynamic examination has been performed 1-7 months after the vascular or traumatic accident.

During filling phase in 5 patients we found detrusor hyperreflexia. During voiding phase 2 patients were able to normal micturition and one patient showed detrusor areflexia. Among the 5 cases with detrusor hyperreflexia in 3 we founded an incomplete voiding. In all hyperreflexic patients we removed the catheter soon with good micturition control at follow-up and the tree patients with incomplete voiding received pharmacological therapy, 2 patients with high pressure hyperreflexia received anticholinergic and myolitic drugs. No alterations of the renal function have been observed until now.

In urology literature no specific data is available on LIS patients. Obviously the heavy comorbidity of these patients represents a key point in the therapeutic attitude and a multidisciplinary approach is required in these cases.

The incomplete voiding with detrusor hyperreflexia can be due to a pons lesion, in this area there are centers controlling the normal synergic voiding activity (detrusor sphincter dyssynergia) or to "pseudodyssynergia" in the sense that in presence of normal consciousness and intact afferent pathway, the patient voluntary contracts the sphincter during urgency to prevent or reduce urine leakage. In the situation of objective communication difficulties and reduction of ability of the patients to express his detrusor sensibility, is difficult to discriminate detrusor sphincter dyssynergia to "pseudodyssinergia" so we prefer to use the term of incomplete voinding.

Most of our patients showed detrusor hyperreflexia, this probably due to the interruption of inhibitor impulses from upper center, only one patient showed detrusor areflexia. We didn't found apparently explanation for these opposite detrusor behaviors, clinical information like sex, age, neurological lesion, time from accident didn't help in the discriminating, Probably the explanation is in the LIS complexity: a syndrome with coexisting different neurological lesions.

In 2 patients there was a normal voiding, the patients express the mcturition stimulus during filling and they were able to voluntary detrusorial contraction one of these with complete voiding.

Conclusions

LIS patients in urology represent a rare condition but the multidisciplinary approach and a particular attention to the communicational difficulties with the rehabilitation team members are important aspects in these patients.

In some LIS patients the filling and the voiding function can remain normally, but most of patients present urodynamic alterations. The key points of preserving the renal function and secondarily to achieve a satisfactory spontaneous voiding represent a rational therapeutic attitude. In our point of view, to help recovering a normal voiding activity, to remove as soon as possible the catheter, to begin a specific pharmacological therapy, to program a cleaned intermittent catheterisation if necessary and to monitor the voiding function, seems to be a correct therapeutic approach.