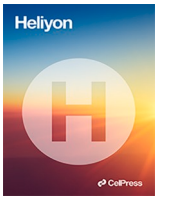
Heliyon 10 (2024) e32374

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon



Case report

Case report: Sacral neuromodulation for neurogenic lower urinary tract dysfunction in patient with neuronal intranuclear inclusion disease

Xiaosong Jin, Haibin Tang, Heng Yuan, Gang Chen *

Department of Urology, The First Affiliated Hospital of Chongqing Medical University, Chongqing, China

ARTICLE INFO

Keywords:
Neuronal intranuclear inclusion disease
Sacral neuromodulation
Neurogenic lower urinary tract dysfunction
Lower urinary tract symptoms
Case report

ABSTRACT

Neuronal intranuclear inclusion disease is an uncommon neurodegenerative disorder. The diagnosis of this condition has become more definitive based on current research. However, treatment options remain limited. Neurogenic lower urinary tract dysfunction is one of the prevalent and significant complications, which is the result of its multi-system effects. Here, we present the case of a 48-year-old man diagnosed with neuronal intranuclear inclusion disease based on existing evidence and was complicated by neurogenic lower urinary tract dysfunction. Despite undergoing some medical treatments, his lower urinary tract symptoms, including urinary incontinence, incomplete or intermittent voiding, etc, continued to worsen while upper urinary tract injury developed. Based on careful consideration of the patient's condition and the available findings, we performed an unprecedented sacral neuromodulation on him. Implementation of sacral neuromodulation led to significant improvement in his urination function and alleviate kidney function damage. Our case suggests a potential therapeutic role for sacral neuromodulation in the treatment of neurogenic lower urinary tract dysfunction associated with neuronal intranuclear inclusion disease. Additional research is required to determine the effectiveness of sacral neuromodulation in managing neurogenic lower urinary tract dysfunction caused by various etiologies.

1. Introduction

Neuronal intranuclear inclusion disease (NIID) is a rare multisystem progressive neurodegenerative disease that affects multiple systems and can be inherited either sporadically or through autosomal dominant inheritance. It is characterized by the presence of eosinophilic, anti-ubiquitin and anti-p62 antibody-positive intranuclear inclusions in cells of central and peripheral nervous system, skin, and internal organs [1]. Currently, it is believed that the genetic factor responsible for this condition involves an expansion of GGC repeats in the 5′ untranslated region of NOTCH2NLC [2]. NIID affects multiple bodily systems, including the central nervous system, and can present with a range of multisystem. These may encompass cognitive decline, Parkinsonian features, episodic encephalopathy, alterations in affect and mood as well as other central nervous system manifestations [3]. Additionally, it may manifest with limb muscle weakness, limb paresthesia along with other peripheral nervous. Involvement of the autonomic nervous system can result in pupillary constriction and bladder dysfunction. In addition to the central nervous system symptoms, urinary dysfunction is a

* Corresponding author.
E-mail address: chengang2308@163.com (G. Chen).

https://doi.org/10.1016/j.heliyon.2024.e32374
Received 31 December 2023; Received in revised form 1 June 2024; Accepted 3 June 2024
Available online 4 June 2024

highly prevalent and prominent manifestation in patients with NIID [4,5]. A number of studies have shown that more than half of patients with NIID have bladder dysfunction, and the detection of anti-p62 and anti-ubiquitin antibody-positive cells in kidney and bladder tissues proves that the urinary system is involved [6,7].

Sacral neuromodulation (SNM) involves the implantation of electrodes to deliver electrical pulses to the sacral nerves, thereby influencing spinal reflexes related to bladder, intestinal tract, sphincter and pelvic floor function in order to improve and restore urinary and excretory functions [8]. Although the mechanism of SNM is complex and still under investigation, this technology has become increasingly mature and widely used in clinical practice. The use of SNM has been approved by the American Urological Association, the European Association of Urology, and the US Food and Drug Administration for the treatment of severe refractory overactive bladder, urge frequent urination syndrome, urge incontinence, chronic pelvic pain, and non-obstructive urinary retention [9]. SNM is recognized as a possible therapy for neurogenic lower urinary tract dysfunction (NLUTD) in the current guideline. However, there are no specific guidelines or recommendations available [10,11]. In this case report, we present a patient with NLUTD due to NIID who underwent sacral nerve modulation and demonstrated significant improvement in postoperative micturition function as well as upper urinary tract function.

2. Case presentation

A 48-year-old male with height of 175cm and weight of 75kg, was admitted to our hospital due to a history of nocturnal enuresis for the past 2 years and worsening symptoms of incomplete emptying for the last 5 months. In November 2020, the patient gradually developed discomfort including headache, dizziness, slowed reaction time, memory deterioration, and unexplained vision loss. Additionally, he experienced occasional nocturnal enuresis. Subsequently, an MRI examination was conducted (Fig. 1). Considering the possibility of autoimmune encephalitis, patient received treatment involving hormone, anti-epileptic and anti-anxiety drugs intake, nerve nutrition supplementation as well as plasma exchange. However, there was no significant improvement observed in his condition. With the progress of the disease, the patient also had seizures, emotional anxiety and other symptoms, and nocturnal enuresis was more frequent. In August 2022, his nocturnal enuresis escalated in frequency. To avoid it, he urinated at 2 and 4 o 'clock at the reminder of family. Additionally, he experienced accompanying issues such as prolonged micturition time, hesitancy before urination, incomplete or intermittent voiding, reliance on abdominal pressure for urination, significantly diminished bladder sensation during filling, absence of relief during micturition, and difficulty distinguishing between defecation and urination. He had a skin biopsy. Inclusion bodies-like structures were observed in the ductal epithelial cells of eccrine sweat glands (located in the skin and subcutaneous tissue of the lateral left calf), which exhibited positive staining for ubiquitin and P62. Additionally, genetic analysis confirmed a total of 143 GGC repeats in the NOTCH2NLC gene, thus confirming a diagnosis of NIID (Fig. 2). In September 2022, the color Doppler ultrasound (CDU) of his urinary system revealed no bilateral ureteral dilatation (Fig. 3A). In February 2023, a repeat CDU and residual urine measurement were conducted, revealing bilateral hydronephrosis, bilateral ureteral dilatation, and an increased amount of residual urine (Fig. 3B and C). He has been diagnosed with diabetes mellitus for a duration of 2 years, and the blood sugar is controlled very well. He was married and worked as a dentist. The investigation into his family history revealed that no NIID patients with similar symptoms have been identified among his close relatives thus far, suggesting that he may be a sporadic case. After his admission to the hospital, a physical examination was conducted, revealing dullness on percussion of the bladder. He maintained a voiding diary for several days and underwent evaluation using the Neurogenic Bladder Symptom Score (NBSS), yielding a score of 38 (supplementary material 1 and 2). His serum creatinine level was measured at 132umol/L (reference range: 62-106μmol/L), and the estimated glomerular filtration rate (eGFR) was determined to be 57.7ml/(min*1.73m^2) (reference range: >90 ml/(min*1.73m^2)). All other laboratory parameters including blood routine, urine routine, liver function, electrolytes, coagulation function were within normal limits. A plain CT scan of the pelvis revealed a distended bladder with irregular thickening of the bladder wall (Fig. 4). Urodynamic studies demonstrated no spontaneous urine flow prior to insertion of the bladder piezometric tube, followed by extraction of approximately 180ml of urine upon insertion. Simultaneous measurement of pressure-flow rate showed increased bladder capacity, decreased sensation, and normal compliance. No detrusor overactivity was observed during retention of urine, and no detrusor contraction was observed during voiding (supplementary material 3). We considered that the patient had NLUTD caused by NIID, and we tried to perform surgery to help him improve urination and protect renal function. The initial procedure took place February 22, 2023. Under local anesthesia, a temporary sacral nerve stimulator was surgically implanted, with electrodes inserted

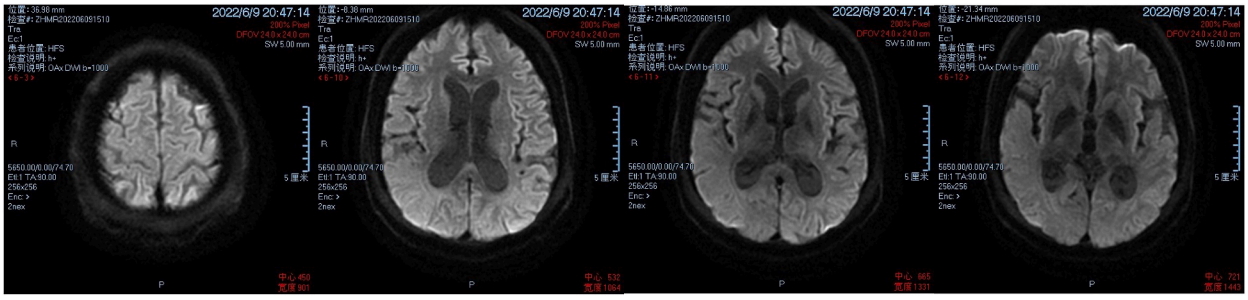


Fig. 1. MRI showed abnormal signals in multiple regions of the brain.

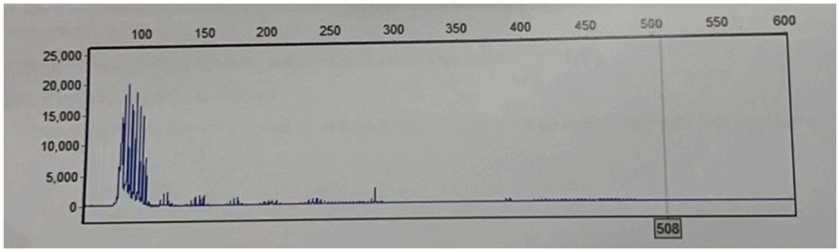


Fig. 2. The horizontal axis represents the fragment length (nt), and the vertical axis represents the fluorescence signal intensity.

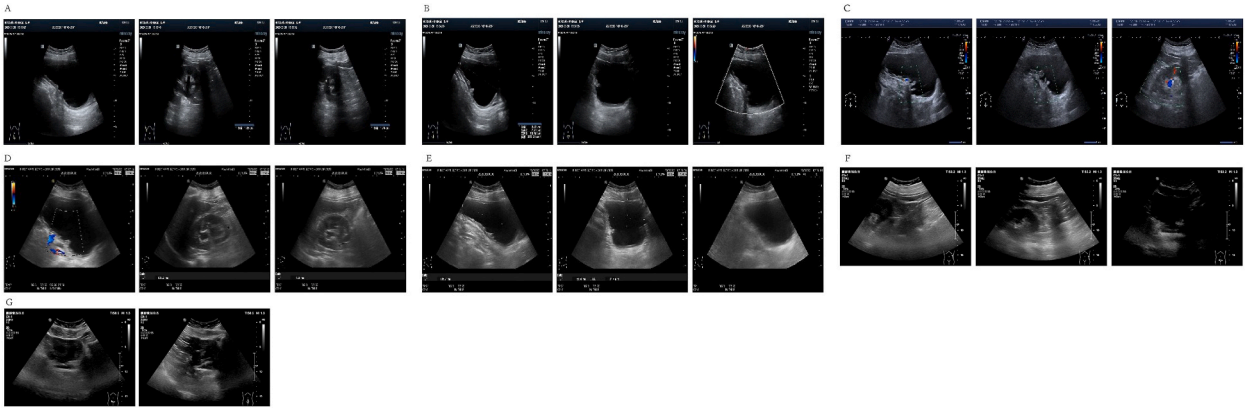


Fig. 3. (A) Mild prostate enlargement and no bilateral ureteral dilatation (September 2022) | (B) Bilateral hydronephrosis, bilateral upper ureteral dilatation, and bladder wall thickening (February 2023) | (C) Post-voided residual urine was approximately 500ml (February 2023) | (D) Bilateral hydronephrosis, bilateral upper ureteral dilatation (March 2023) | (E) Post-voided residual urine volume in the bladder was approximately 375ml (March 2023) | (F) Bilateral ureters were not dilated, the bladder wall was significantly thickened, and the prostate was regularly shaped (November 2023) | (G) The residual urine in the bladder after voiding was about 10ml (November 2023).

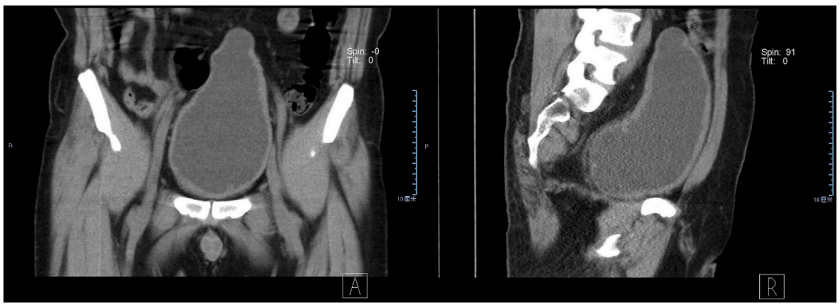


Fig. 4. Distended bladder with irregular thickening of the bladder wall.

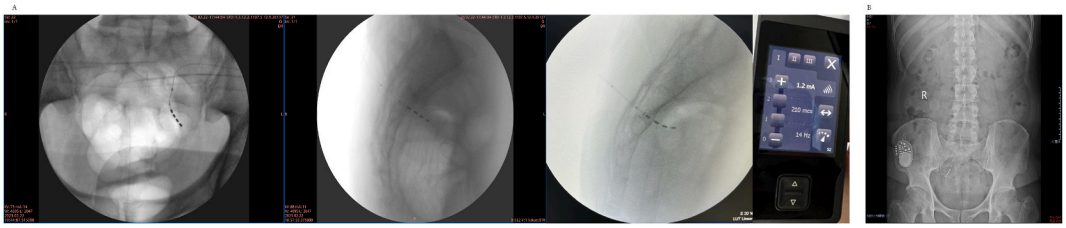


Fig. 5. (A) A temporary sacral nerve stimulator was surgically implanted | (B) Convert the temporary sacral electrical stimulator into a permanent one.

through S3 sacral foramen. The current was set to 1.2mA, pulse width to 210mcs, frequency to 14Hz, positive and negative electrodes to 0–3+, and multiple contacts were tested to confirm response(Fig. 5 A). On the night after surgery, the patient could get up and go to the bathroom without bed-wetting. The patient was observed in the hospital for 5 days after the operation. Since the operation, he no longer wet the bed after falling asleep at night, and he did not need an alarm clock or other people to remind him to get up. He kept a voiding diary for the days after surgery, with reduced voiding times at night. Following the patient's return home, there was a significant improvement in his urinary symptoms, prompting him to opt for hospital readmission for surgical intervention permanent sacral nerve stimulation implantation. On March 8, 2023, we conducted procedure to convert the temporary sacral electrical stimulator into a permanent one(Fig. 5 B). Post-procedure observation within the hospital setting revealed an absence of nocturnal enuresis. On March 20, 2023, CDU of his urinary system was performed in the outpatient clinic, which revealed little change and a decrease in residual urine volume(Fig. 3 D and E). Renal function testing, performed on March 21, 2023, revealed a serum creatinine level of 83umol/L (reference range: 62-106μmol/L) and an estimated glomerular filtration rate of 101.1 ml/(min*1.73m^2) (reference range: >90 ml/(min*1.73m^2)). The patient was followed up at our hospital's outpatient department on November 20, 2023. He reported the absence of nocturnal enuresis or decreased nocturnal urination post-surgery, significant reduction in urination time, and improvement in NBSS score (supplementary material 4). Renal function test revealed a serum creatinine level of 87umol/L (reference range: 62-106μmol/L). CDU showed no bilateral hydronephrosis and bilateral ureters were not dilated. Residual urine was about 10ml (Fig. 3F and G). Moreover, we found from our communication with the patient that the frequency of headache and dizziness was reduced, the mental state was better, and the reaction time was faster than before. Urodynamic assessment with pressure flow rate synchronous measurement indicated normal bladder capacity and weakened sensory function, while compliance remained normal without detrusor overactivity during urine storage and normal detrusor muscle contraction during voiding (supplementary material 3). Notably, there was significant improvement observed in the patient's clinical symptoms as well as renal function, hydronephrosis of the kidney and residual urine volume in the bladder. It is recommended that the patient continues to undergo regular follow-up examinations at the outpatient clinic to verify the long-term effects.

3. Discussion

NIID poses challenges in diagnosis due to its highly variable clinical manifestations. Over the past few decades, there has been a dearth of minimally invasive examination methods that could effectively aid in diagnosing NIID. However, depending on the identification of eosinophilic inclusions in skin biopsy specimens, the detection of high signal along the cortico-medullary junction through diffusion-weighted imaging of the brain, and GGC repeat amplification analysis of the NOTCH2NLC gene, it is now possible to achieve clearer diagnoses for this rare disease even when presented with multi-system and complex clinical manifestations [12,13]. Some studies revealed that bladder dysfunction is a prevalent clinical manifestation among NIID. Yun et al. discovered in their study involving 247 NIID patients that approximately 64 % exhibited autonomic dysfunction, with 119 of these individuals experiencing unexplained bladder dysfunction, which stands as the most common symptom associated with autonomic dysfunction [14]. Hongfei Tai conducted a comprehensive investigation on 223 NIID patients, revealing that 55.6 % of them suffered from rectal and bladder dysfunction [15]. Additionally, Nakamura M et al. found that voiding dysfunction was sometimes an initial symptom preceding other neurological manifestations in certain NIID patients [16]. Currently, there are no effective treatments available to cure or slow down the progression of NIID; therefore, symptomatic treatment is primarily employed in clinical practice alongside nutritional support and psychotherapy to alleviate symptoms and enhance quality of life for affected individuals [17].

Urinary retention in male patients can be classified as obstructive or non-obstructive according to the causes. The obstruction may be caused by internal causes such as prostatic hyperplasia, lower urinary tract malignancy, urinary tract infection, blood clots, urethral strictures, and bladder stones, or by compression from the lesion structure outside the urinary tract. Repeated urinary tract infections can cause structural changes in the urethra, resulting in urethral strictures and obstructions, such as urethral diverticula [18]. Non-obstructive urinary retention is mostly caused by abnormal neurological function of the lower urinary tract, including the bladder. Acute causes of NLUTD include compression of the spinal cord or cauda equina nerve root and central nervous system emergencies. Diabetic neuropathy can cause chronic impairment of urination function. Taking anticholinergic drugs may also have adverse effects on urinary tract neurological function [19–21].

Pharmacotherapy of NLUTD is currently only performed in the setting of detrusor overactivity. Although the use of drugs to improve detrusor contraction has been studied, their use in clinical practice is controversial and there is insufficient evidence to recommend its routine use to improve bladder emptying. To date, no drug treatment for neurogenic detrusor underactivity has been recommended [22]. In patients with chronic urinary retention, bladder urine drainage is required. Clean intermittent catheterization is a safe method, but it requires repeated catheterization operations. The most common complications include urinary tract infection, bleeding, and urethral lesions. The discomfort during catheterization can reduce the compliance of patients. Some factors may limit the implementation of clean intermittent catheterization, such as urethral lesions, motor sequelae, tremor or difficulty in operation, cognitive impairment, neuropathic pain, etc., resulting in the need for long-term indwelling catheters in patients. Long-term indwelling catheter can lead to complications such as urinary tract infection and bladder stones. Chronic inflammation caused by urinary tract infection may lead to an increased risk of bladder cancer [23]. Cystostomy is a method of bladder urine drainage without repeated catheterization operation. However, cystostomy cannot be easily accepted by patients because of its invasiveness and nursing difficulties. Some patients with long-term indwelling catheter or bladder stoma may have recurrent urinary tract infection, which is closely related to the onset of encephalopathy [7,24]. Sphincterotomy has the potential to limit male reproduction, and is relatively invasive. The procedure can also lead to a series of complications, such as hematuria, bacteremia, recurrence of urinary tract infection, urethral stricture, and autonomic nerve reflex disorders [25,26].

In this case report, the patient was diagnosed with NIID based on his multisystemic symptoms, diagnosis and treatment across multiple medical centers, genetic analysis, skin biopsy, and other ancillary examinations. Despite undergoing various medical treatments, there was partial improvement in neuropsychological function and reduction in cranial lesions; however, lower urinary tract symptoms continued to progress. It is worth noting that the patient has diabetes, which may have contributed to the dysuria. However, he has a history of good glycemic control and no history of peripheral neuropathy, such as distal extremity neuropathy, or renal disease. His dysuria symptoms developed and progressed after the central nervous symptoms of NIID. We concluded that the patient had NLUTD due to NIID. NLUTD refers to impaired bladder and urinary sphincter function resulting from specific alterations in the central nervous system and/or peripheral nervous system caused by various neurological disorders [22,27]. The patient had nocturnal urinary incontinence, decreased perception of urination, weakness of urination, increased residual urine, weakened bladder sensation during retention of urine, and weakened detrusor contraction during contraction, which reflected the overfilling of the bladder and caused filling urinary incontinence. The feeling of urination and motor function of patients are affected by the disease, and they cannot normally perceive the desire to urinate and actively urinate. We noted that the upper urinary tract structure was affected, possibly related to the obstruction caused by lower urinary tract symptoms, and that the patient's renal function was also impaired to some extent. NLUTD is associated with various complications, including deterioration of the upper urinary tract and an increased risk of bladder malignancy, leading to significant morbidity and mortality. The management of NLUTD aims to control urinary incontinence, enhance quality of life, and prevent or minimize complications such as urinary tract infections and impaired upper urinary tract function. Timely intervention can help prevent further decline in renal function [25,28]. SNM demonstrates efficacy not only in addressing lower urinary tract dysfunction but also intestinal dysfunction [29]. In the case of non-obstructive urinary retention, SNM has been proven to restore coordinated urination and is an effective treatment approach [8,30]. Based on these studies and the patient's preferences, we have decided to perform SNM.

We conducted a comprehensive literature search using databases such as PubMed, MEDLINE, Embase, and Google Scholar with specific keywords including "neuronal intranuclear inclusion disease," "neurogenic lower urinary tract dysfunction," and "sacral neuromodulation." To the best of our knowledge, there is currently no documented evidence of SNM being utilized for the treatment of NIID complicated with NLUTD.

Our patient demonstrated significant improvement in lower urinary tract symptoms following sacral nerve stimulator implantation. The patient achieved nocturnal continence and experienced reduced frequency of nighttime urination, shorter voiding time, and absence of the need for abdominal pressure to initiate urination. Additionally, there was no longer any occurrence of urinary incontinence during bladder filling. We observed a recovery in the patient's urinary perception and function post-surgery, as well improved clarity in their sensation of both urination and defecation. The patient's renal function, hydronephrosis of kidney, urodynamics and bladder residual urine volume showed improvement during the follow-up period. As can be seen from the voiding diary we recorded after the first surgery, the patient's urinary incontinence improved significantly after the first temporary SNM. Recent studies have shown that there is a significant correlation between subjective voiding diary parameters and urodynamic examination results before and after SNM, and urodynamic examination can be used to replace voiding diary recording, so as to shorten the cycle of testing treatment effect [31]. The urodynamics was evaluated again at the postoperative follow-up, and the voiding activity was significantly normalized compared with that before surgery. These findings collectively indicate that SNM was beneficial for managing NLUTD in a patient with NIID. SNM is a minimally invasive and reversible treatment. The precise mechanism of its action remains unclear and may be attributed to the stimulation of afferent and efferent nerve fibers that connect the bladder and pelvic floor, spinal interneurons, as well as the central nervous system [32]. This stimulation likely affects not only motor responses of the detrusor or urethral sphincter but also exerts significant effects on bowel function, sexual function, and pelvic pain management. Consequently, SNM can be considered a systemic treatment approach rather than one solely targeting bladder-related symptoms [33,34]. In many cases, SNM involves stimulating the S3 nerve root using an electrode implanted through puncture techniques. This preference could be attributed to its inclusion of sensory fibers from the pelvic floor along with parasympathetic fibrous muscle activity while establishing a reflex pathway connecting the spinal cord to higher centers within responsible for regulating micturition [35].

The efficacy of SNM in the treatment of NLUTD caused by neurological disorders remains unclear [36]. It has now been suggested that SNM could be equally effective in treating such patients. Some evidences suggest that the success rate of SNM treatment for individuals with neurological diseases or injuries may be comparable to the established indications of SNM for non-neurogenic Lower urinary tract dysfunction [37].SNM has a significant impact on bladder dysfunction in patients with multiple sclerosis (MS), which is also a central nervous system disease often accompanied by urinary dysfunction [38,39]. Daniele Minardi et al. performed SNM on 25 MS patients with refractory lower urinary tract symptoms, of whom 15 patients (60 %) showed significant improvement in voiding symptoms [40]. Liechti, Martina D et al. demonstrated that sacral nerve electrical stimulation exhibited for NLUTD. However, there remains a dearth of long-term and large-scale clinical studies to substantiate its long-term effectiveness and safety [41]. Long-term treatment necessitates consideration of the progressive deterioration of neurological status over time and disease progression, while future research should longitudinally investigate the correlation between bladder symptoms, changes in neurological status, and SNM outcomes [42]. Despite the potential impact of numerous neurological diseases and injuries on lower urinary tract function, symptoms exhibit considerable consistency [36]. There is currently no standardized approach for sacral nerve stimulation in terms of parameters, frequency, and electrode positioning across different neurological diseases [29]. Furthermore, it remains unclear which patients with neurological conditions are most suitable for this intervention. Necessitating further comprehensive research to validate its efficacy [26]. Further research is required to investigate the specific mechanism of SNM action, including elucidating how SNM induces alterations in afferent signals and brain activity [43,44]. Advancements in device technology, such as miniaturized devices equipped with enhanced batteries or charging systems, as well as devices compatible with magnetic resonance imaging, will enhance the user-friendliness of SNM [45]. Therefore, it is crucial to foster multidisciplinary collaboration for effective management of lower

urinary tract dysfunction [46,47].

4. Conclusion

With ongoing research and of diagnostic methods and criteria, an increasing number of cases of NIID can now be definitively diagnosed. Currently, treatment for NIID primarily focuses on managing symptoms to minimize complications, but there is no effective means to alleviate or reverse the underlying disease itself. Lower urinary tract dysfunction is a prevalent manifestation among NIID patients with complex clinical presentations. It necessitates careful attention and effective intervention to mitigate physical and psychological impacts on patients while enhancing their prognosis. SNM represents a potential, minimally invasive and reversible treatment technique that has demonstrated promising short-term efficacy. however, further studies are required for validation purposes. Future clinical interventions and research should prioritize exploring this potential therapeutic approach.

Funding statement

The authors declare that this study received funding from the Senior Medical Talents Program of Chongqing for Young and Middle-aged Science supported by Chongqing Municipal Health Commission (number 2022GDRC014) and Smart Medicine Project of Chongqing Medical University in 2021 (project No. ZHYX202124). The funder was not involved in the study design, collection, analysis, interpretation of data, the writing of this article or the decision to submit it for publication.

5. Data availability statement

The authors confirm that the data supporting the findings of this study are available within the article [and/or its supplementary materials]. Further inquiries can be directed to the corresponding author.

Ethics statement

The patients/participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article. No animal studies are presented in this manuscript. No potentially identifiable human images or data is presented in this study.

CRediT authorship contribution statement

Xiaosong Jin: Writing – original draft, Investigation. **Haibin Tang:** Data curation. **Heng Yuan:** Investigation. **Gang Chen:** Writing – review & editing, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e32374.

References

[1] Y. Yan, et al., The clinical characteristics of neuronal intranuclear inclusion disease and its relation with inflammation, Neurol. Sci. 44 (9) (2023) 3189–3197.
[2] Y. Tian, et al., Expansion of human-specific GGC repeat in neuronal intranuclear inclusion disease-related disorders, Am. J. Hum. Genet. 105 (1) (2019) 166–176.
[3] Y. Liu, et al., Clinical and mechanism advances of neuronal intranuclear inclusion disease, Front. Aging Neurosci. 14 (2022).
[4] H. Liang, et al., Clinical and pathological features in adult-onset NIID patients with cortical enhancement, J. Neurol. 267 (11) (2020) 3187–3198.
[5] J. Sone, et al., Clinicopathological features of adult-onset neuronal intranuclear inclusion disease, Brain 139 (12) (2016) 3170–3186.
[6] Y.H. Liu, et al., Neuronal intranuclear inclusion disease in patients with adult-onset non-vascular leukoencephalopathy, Brain 145 (9) (2022) 3010–3021.
[7] X. Lu, D. Hong, Neuronal intranuclear inclusion disease: recognition and update, J. Neural. Transm. 128 (3) (2021) 295–303.
[8] D. Liberman, M.J. Ehlert, S.W. Siegel, Sacral neuromodulation in urological practice, Urology 99 (2017) 14–22.
[9] M. Mass-Lindenbaum, et al., Sacral neuromodulation - when and for who, Int. Braz J. Urol. 47 (3) (2021) 647–656.
[10] M. Stöhrer, et al., EAU guidelines on neurogenic lower urinary tract dysfunction, Eur. Urol. 56 (1) (2009) 81–88.
[11] D.A. Ginsberg, et al., The AUA/SUFU guideline on adult neurogenic lower urinary tract dysfunction: treatment and follow-up, J. Urol. 206 (5) (2021) 1106–1113.
[12] X. Ren, et al., Skin biopsy and neuronal intranuclear inclusion disease, J. Dermatol. 50 (11) (2023) 1367–1372.
[13] L. Bao, et al., Current advances in neuronal intranuclear inclusion disease, Neurol. Sci. 44 (6) (2023) 1881–1889.
[14] Y. Tian, et al., Clinical features of NOTCH2NLC-related neuronal intranuclear inclusion disease, J. Neurol. Neurosurg. Psychiatr. 93 (12) (2022) 1289–1298.

[15] H. Tai, et al., Clinical features and classification of neuronal intranuclear inclusion disease, Neurology. Genetics 9 (2) (2023) e200057.
[16] M. Nakamura, et al., Two cases of sporadic adult-onset neuronal intranuclear inclusion disease preceded by urinary disturbance for many years, J. Neurol. Sci. 392 (2018) 89–93.
[17] H.K. Raza, et al., Recent progress in neuronal intranuclear inclusion disease: a review of the literature, Neurol. Sci. 41 (5) (2020) 1019–1025.
[18] K. Gkeka, et al., Acute urinary retention in a woman due to large urethral diverticulum: a case report, Continence Reports 6 (2023) 100030.
[19] M. Billet, T.A. Windsor, Urinary retention, Emerg. Med. Clin. 37 (4) (2019) 649–660.
[20] K.M. Verhamme, et al., Drug-induced urinary retention: incidence, management and prevention, Drug Saf. 31 (5) (2008) 373–388.
[21] D.C. Serlin, J.J. Heidelbaugh, J.T. Stoffel, Urinary retention in adults: evaluation and initial management, Am. Fam. Physician 98 (8) (2018) 496–503.
[22] J.C. Truzzi, et al., Neurogenic bladder – concepts and treatment recommendations, Int. Braz J. Urol. 48 (2) (2022) 220–243.
[23] M. Kennelly, et al., Adult neurogenic lower urinary tract dysfunction and intermittent catheterisation in a community setting: risk factors model for urinary tract infections, Advances in Urology 2019 (2019) 1–13.
[24] M. Kumagai, et al., Vesicostomy: an alternative approach for complicated adult patients with urinary retention, Continence Reports 8 (2023) 100040.
[25] R.A. Vince, A.P. Klausner, Surveillance strategies for neurogenic lower urinary tract dysfunction, Urol. Clin. 44 (3) (2017) 367–375.
[26] J.N. Panicker, Neurogenic bladder: epidemiology, diagnosis, and management, Semin. Neurol. 40 (5) (2020) 569–579.
[27] J.B. Gajewski, M.J. Drake, Neurological lower urinary tract dysfunction essential terminology, Neurourol. Urodyn. 37 (S6) (2018).
[28] S. Agrawal, R.R. Agrawal, H.M. Wood, Establishing a multidisciplinary approach to the management of neurologic disease affecting the urinary tract, Urol. Clin. 44 (3) (2017) 377–389.
[29] J.N. Panicker, C.J. Fowler, T.M. Kessler, Lower urinary tract dysfunction in the neurological patient: clinical assessment and management, Lancet Neurol. 14 (7) (2015) 720–732.
[30] P.E.V. Van Kerrebroeck, T.A.T. Marcelissen, Sacral neuromodulation for lower urinary tract dysfunction, World J. Urol. 30 (4) (2012) 445–450.
[31] J. Drossaerts, et al., The value of ambulatory urodynamics in the evaluation of treatment effect of sacral neuromodulation, Urol. Int. 102 (3) (2019) 299–305.
[32] S. De Wachter, D. Vaganee, T.M. Kessler, Sacral neuromodulation: mechanism of action, European Urology Focus 6 (5) (2020) 823–825.
[33] M.A. Averbeck, J. Moreno-Palacios, A. Aparicio, Is there a role for sacral neuromodulation in patients with neurogenic lower urinary tract dysfunction? Int. Braz J. Urol. 46 (6) (2020) 891–901.
[34] T. Marcelissen, et al., Sacral neuromodulation as a treatment for chronic pelvic pain, J. Urol. 186 (2) (2011) 387–393.
[35] A. Abello, A.K. Das, Electrical neuromodulation in the management of lower urinary tract dysfunction: evidence, experience and future prospects, Therapeutic Advances in Urology 10 (5) (2018) 165–173.
[36] A. van Ophoven, et al., Systematic literature review and meta-analysis of sacral neuromodulation (SNM) in patients with neurogenic lower urinary tract dysfunction (nLUTD): over 20 Years' experience and future directions, Adv. Ther. 38 (4) (2021) 1987–2006.
[37] T.M. Kessler, et al., Sacral neuromodulation for neurogenic lower urinary tract dysfunction: systematic review and meta-analysis, Eur. Urol. 58 (6) (2010) 865–874.
[38] H. Abboud, et al., Neuromodulation in multiple sclerosis, Multiple Sclerosis Journal 23 (13) (2017) 1663–1676.
[39] I. Rahnama, S. M, Neuromodulation for functional bladder disorders in patients with multiple sclerosis, Multiple Sclerosis Journal 26 (11) (2020) 1274–1280.
[40] D. Minardi, G. Muzzonigro, Sacral neuromodulation in patients with multiple sclerosis, World J. Urol. 30 (1) (2012) 123–128.
[41] M.D. Liechti, et al., Sacral neuromodulation for neurogenic lower urinary tract dysfunction, NEJM Evidence 1 (11) (2022).
[42] K.M. Peters, et al., Clinical outcomes of sacral neuromodulation in patients with neurologic conditions, Urology 81 (4) (2013) 738–744.
[43] I. Masood, et al., Sacral neuromodulation in patients with neurogenic lower urinary tract dysfunction: a multicenter retrospective study from China, Neuromodulation: Technology at the Neural Interface 24 (7) (2021) 1278–1283.
[44] C.K. Moore, J.J. Rueb, S. Derisavifard, What is new in neuromodulation? Curr. Urol. Rep. 20 (9) (2019) 55.
[45] S. De Wachter, et al., New technologies and applications in sacral neuromodulation: an update, Adv. Ther. 37 (2) (2020) 637–643.
[46] M.J. Drake, et al., Neurogenic lower urinary tract dysfunction: clinical management recommendations of the Neurologic Incontinence committee of the fifth International Consultation on Incontinence 2013, Neurourol. Urodyn. 35 (6) (2016) 657–665.
[47] J.M. Marra, et al., Neurogenic disorders and the lower urinary tract dysfunction: proposed approach for the gynecologist, Reprod. Sci. 30 (7) (2023) 2087–2091.