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Case report

HA280 immunoadsorption, an alternative treatment for neuromyelitis optica spectrum disorders?

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ABSTRACT

We report a case of neuromyelitis optica spectrum disorders (NMOSD) with complete loss of vision in the left eye in a patient who was not satisfied with the effect of methylprednisolone therapy, which was improved by HA280 immunoadsorption (IA) therapy. HA280 is a relatively cheaper IA column (made in China) often used in the treatment of rheumatoid immune-related diseases. HA280 can effectively remove inflammatory markers in serum. However, whether the HA280 IA column is suitable for NMOSD is unknown. This case suggests that the HA280 IA column has a potential therapeutic effect on NMOSD and may be an alternative treatment for steroid-resistant NMOSD. There may be therapeutic targets other than anti-AQP4 antibody. Identifying the inflammatory substances that could be removed to contribute to NMOSD recovery is worthy of further study, and the results could provide new ideas for acute NMOSD treatment. Moreover, the HA280 IA column is relatively cheap and allows lower-income families to use it.

1. Introduction

Immunoadsorption (IA) therapy is a new method for the treatment of neuroimmune diseases(Oji and Nomura, 2017). IA is regarded as a possible alternative to plasmapheresis. The TR-350 IA column has been shown to be effective in neuromyelitis optica spectrum disorders (NMOSD)(Kleiter et al., 2018). However, the TR-350 IA column is expensive and has not been widely used in China. The adsorbent HA280, which is relatively cheaper, also absorbs and removes the inflammatory mediators and cytokines in blood and this absorbent has often been used in the treatment of rheumatic immunity diseases in China (Xu et al., 2016). However, whether the HA280 IA column is suitable for NMOSD is unknown.

In this case, we successfully treated an NMOSD patient with complete vision loss using a HA280 IA column (Jafron Biomaterial Co., Ltd., Zhuhai, China).

2. Case

A 36-year-old female presented to the Department of Ophthalmology in our hospital and complained of sudden loss of vision accompanied by postocular pain in the left eye for 5 days. On admission, she had no light perception in her left eye, and the visual acuity (VA) of the right eye was only 20/33.3 due to an attack four months ago. Optic nerve magnetic resonance imaging (MRI) showed a swollen and tortuous left optic nerve (Fig. 1). The ophthalmologist considered that the patient had developed optic neuritis and administered 500 mg/day methylprednisolone (MP) therapy for 3 days, followed by oral prednisone at 60 mg for maintenance. However, there was no improvement in the VA of the patient. Consultation and examination by a neurologist reveals that the serum anti-AQP4 antibody was positive, and the titer was 1:320 (using the cell-based assay). No obvious abnormalities were found in the cerebrospinal fluid or other routine blood items. Finally, the patient was diagnosed with NMOSD. The patient received IA therapy by HA280 IA column once every other day for a total of 5 times(Xu et al., 2016). Light sensation in the left eye was recovered immediately after the first IA treatment, and VA reached 150 cm "counting fingers" on the left eye after completion of the 5th IA treatment. The patient was discharged at 1 week after the 5th IA treatment, and the left eye VA returned to 20/66.7 (Fig. 2). The patient continued treatment with azathioprine (100 mg/day) and prednisone (60 mg/day, gradually reducing the dose) in the outpatient clinic. Two weeks after discharge, the left eye VA of the patient returned to 20/40.

3. Discussion

In our case, because the initial treatment had no significant effect, we recommended that the patient start a second round of treatment

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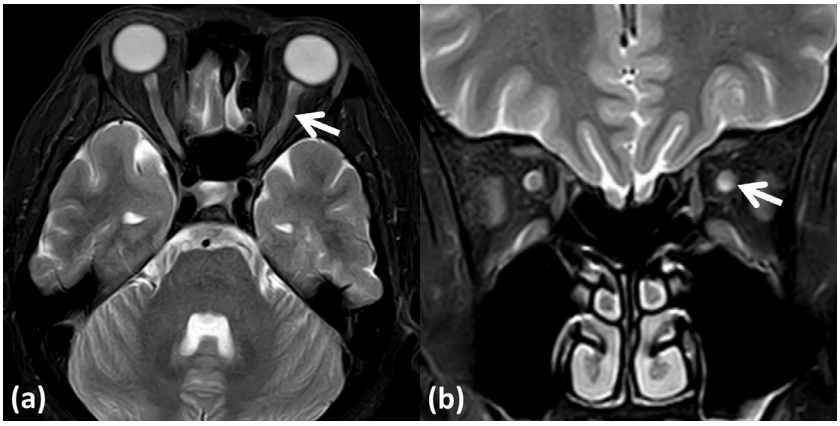

Fig. 1. MRI images of the optic nerve on admission. (a) Transverse position. The left optic nerve is swollen and tortuous, and the path is fuzzy (arrow). (b) Coronary position. The fibers and sheath of the left optic nerve are swollen and blurred (arrow).

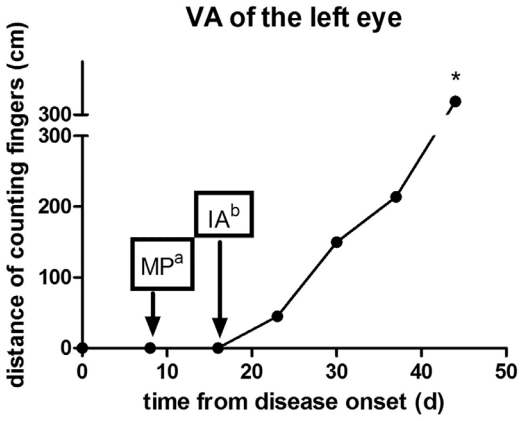

Fig. 2. The VA in the left eye of the patient changed since onset. (a) Methylprednisolone (500 mg for 3 days, followed by oral prednisone at 60 mg for maintenance) was given on the 8th day of onset. The patient was unable to see any light in the left eye. (b) HA280 IA treatment (once every other day, five times in total) was initiated at day 16 of onset, then the VA was assessed weekly. The VA in left eye of the patient was recorded by the distance of "counting fingers". The VA in the left eye of the patient gradually improved. *At discharge, the left eye VA of the patient was improved to 20/66.7.

immediately. Considering the side effects of another higher dose of methylprednisolone shock therapy, there is uncertainty about the effectiveness of this treatment. In addition, the patient's family could not afford the high medical costs of plasmapheresis or human immunoglobulin therapy. Eventually, the patient's husband asked to try a relatively cheaper treatment with HA280 IA therapy (it is estimated that the treatment cost of HA280 IA is approximately 1/2–2/3 that of immunoglobulin). Surprisingly, after the patient completed the last IA treatment, the serum anti-AQP4 antibody was tested again, and the antibody titer did not decrease.

The symptoms of the patient improved, but the anti-AQP4-antibody titer did not decrease. We speculate that the possible cause for this effect is that NMOSD has many non-AQP4-antibody therapeutic targets. The targets that have been shown to be effected are B cells, plasma cells, T helper cells, C5, IL-6, and IL-17(Bienia and Balabanov, 2013). Previous studies have reported that the HA280 IA column can effectively remove C3, C4, IgA, IgG, IgM and other inflammatory mediators or harmful substances in the blood(Xu et al., 2016), which may have led to an improvement in NMOSD activity. Further research is needed to identify the inflammatory mediators that could be removed to promote the recovery of the patient and to find new therapeutic targets for NMOSD.

Of course, we cannot completely rule out the possibility of spontaneous remission or delayed effects of methylprednisolone therapy in this patient. However, anti-AQP4 antibody-positive NMOSD vision damage is often more severe than simple optic neuritis or multiple sclerosis, and blindness can result if left untreated(Wu et al., 2019). Our observations are based on expert consensus, and the consensus suggests initiating continued treatment when glucocorticoid therapy is not effective(Trebst et al., 2014). The patient was unable to see any light in the left eye until HA280 IA therapy was initiated. Therefore, it is hard to deny that HA280 plays a role in the treatment. The efficacy of HA280 IA remains to be further studied.

In conclusion, HA280 IA may be a potential alternative treatment for steroid-resistant NMOSD, and the therapeutic effect is worth further study. Further research may help us to identify other possible therapeutic targets in NMOSD. Moreover, the HA280 IA column is relatively cheap and allows use by lower-income families.

Declaration of Competing Interest

All authors declare that they do not have any direct or indirect commercial financial incentives associated with the publication of this work.

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