Cerebral parenchymal and cervical spinal lesions of Vogt-Koyanagi-Harada disease: a case report
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Introduction

- Vogt-Koyanagi-Harada disease (VKH) is an autoimmune systemic disorder characterized by inflammation of the uvea, meninges, auditory and integumentary system.
- Neurological symptoms of VKH include meningitis, consciousness disturbance, cranial nerve palsy, hemiplegia, and ataxia.1-3
- In VKH, there are several reports of cases with involvement of brain parenchyma including spinal cord.

Case Presentation

A Japanese 64-year-old woman

- [CC] reduction in eyesight, truncal ataxia
- [PMH] hypertension, VKH (developed at 46-year-old)
- [HI]
  - X-17 years: She was diagnosed as VKH at the other hospital. She received oral corticosteroid treatment, with improvement of her visual impairment.
  - X-1 week: She developed leaming to the left on gait with impairment of bilateral visual fields.
  - X-1 day:
    - Ophthalmic evaluations showed upper left homonymous quadrantanopia.
  - X:
    - She was referred to our department.

- [Physical examination]
  - HT: 158 cm
  - BW: 48.7 kg
  - BT: 36.6 °C
  - PR: 67 / min
  - Other vital signs were normal.
  - Meningeal sign (-)
  - Skin lesion (-)
  - Sunset glow fundus (+)

- [Neurological positive findings]
  - # Upper left homonymous quadrantanopia
  - # Disturbance of disturbed fine movement of left hand
  - # Mann’s test (+)
  - # Posture deviated to the left on gait

- [Laboratory findings]
  - Routine laboratory tests were normal including CBC and CRP.

  - [Serum chemistry]
    - Anti-AQP4 Ab
    - ANA
    - Anti-TPO Ab
    - Anti-CL Ab
    - Anti-SS-A Ab
    - Anti-SS-B Ab
    - PR3-ANCA
    - MPO-ANCA
    - Vit B1
    - Vit B12
    - ACE
    - ADA

  - [HLA allele typing] DR4, DRB1*04

Discussion

- **VKH patients with brain or spinal cord lesions**

<table>
<thead>
<tr>
<th>Author</th>
<th>Abnormal MRI lesions</th>
<th>Treatment / Clinical course</th>
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<tbody>
<tr>
<td>Nitta4</td>
<td>cerebellar white matter, basal ganglia, left cerebral peduncle</td>
<td>oral PSL 40 mg; oral steroid taper; no relapse</td>
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<tr>
<td>Ikedaa</td>
<td>left anterior limb of internal capsule, left parietal lobe</td>
<td>oral PSL; no treatment; no relapse</td>
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<tr>
<td>Iwasata</td>
<td>left frontal lobe, periventricular white matter</td>
<td>mPSL d. i.v. 1 g × 3 days i. v.; oral steroid taper; improved; no relapse</td>
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<tr>
<td>Hashimoto6</td>
<td>left middle cerebellar peduncle</td>
<td>mPSL d. i.v. 1 g × 5 days i. v.; oral steroid taper; improved; no relapse</td>
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<tr>
<td>Tang6</td>
<td>cervical and thoracic cord (C6-T9)</td>
<td>oral steroid taper; immunosuppressive therapy; no relapse</td>
</tr>
<tr>
<td>Shauquan9</td>
<td>cervical cord (C3-L)</td>
<td>mPSL d. i.v. 1 g × 3 days i. v.; mPSL d. i.v. 0.5 g × 30 days i. v.; oral steroid taper; no relapse</td>
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<tr>
<td>Dabhouli12</td>
<td>bilateral subcortical white matter, cerebral peduncle</td>
<td>mPSL d. i.v. 0.5 g × 5 days i. v.; oral steroid taper; no relapse; no relapse; no relapse</td>
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- Present case

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<th>63/F Japan</th>
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<td>Disturbance, right cerebral peduncle, optic tract, cervical cord (C3/C4)</td>
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<tr>
<td>mPSL d. i.v. 1 g × 3 days i. v.; oral steroid taper; no relapse; no relapse</td>
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- Our case is characterized by the concurrent appearance of brain and spinal cord lesions.
- There is only one case10, accompanied by both lesions besides this case.
- There was no VKH case reported with recurrence during treatment, except for this case.

Conclusion

- We presented a patient with VKH who showed abnormal MRI lesions of brain and spinal cord.
- We regarded these MRI lesions as those caused by VKH.
- Early diagnosis and systemic administration of corticosteroids will be very important to suppress the acute inflammatory process in central nervous system, and prevent the recurrences and development of the disease.

References