Unusual neurologic manifestations of Vogt-Koyanagi-Harada disease: a systematic literature review

Moussa Toudou-Daouda1* and Abdoul Kadir Ibrahim-Mamadou2

Abstract

Background and Purpose: The usual neurologic manifestations of Vogt-Koyanagi-Harada (VKH) disease include aseptic meningitis and headaches. We performed the present study to review all unusual neurologic manifestations reported in VKH disease to summarize them.

Methods: A literature search was performed in the English language on Scopus and Medline via PubMed from 1946 to July 31, 2021, by using the following terms: “Vogt Koyanagi Harada disease” OR “VKH disease” AND “Brain” OR “Spinal cord” OR “CNS” OR “Central nervous system” OR “Neurologic” OR “Peripheral nervous system” OR “Polyneuropathies. Our inclusion criteria were unusual neurologic manifestations of VKH disease.

Results: Our literature search yielded 417 total articles (PubMed = 334, Scopus = 83) from which 32 studies comprising 43 patients (22 men and 21 women, of which 62.8% were younger than 50 years) were included in this systematic literature review. Regarding the study design, all studies were case reports and published between 1981 and 2021. CNS involvement was the most reported (93%) in VKH disease. Peripheral nervous system involvement represents 7% of cases. The cerebral lesions were parenchymal inflammatory lesions in the white matter or posterior fossa with or no contrast enhancement (16.3%), leptomeningitis (9.3%), pachymeningitis (7%), meningoencephalitis (2.3%), ischemic stroke (4.6%), hemorrhagic stroke (2.3%), transient ischemic attack (2.3%), and hydrocephalus (2.3%). The optic nerve lesions were anterior ischemic optic neuropathy (20.9%) and optic neuritis (9.3%). Concerning spinal cord lesion, it was mainly myelitis (14%).

Conclusion: This systematic literature review provides a summary of the different unusual neurologic manifestations reported in VKH disease.

Keywords: Nervous system, Vogt-Koyanagi-Harada disease, unusual neurologic manifestations

Background

Vogt-Koyanagi-Harada (VKH) disease is characterized by bilateral ocular involvement associated with extraocular manifestations such as neurological (related to aseptic meningitis: headache, neck and back stiffness), auditory (tinnitus, hearing loss, and vertigo), and integumentary (alopecia, poliosis, and vitiligo) [1]. VKH disease is a rare multisystemic autoimmune disease, mediated by T cells directed against melanocytes strongly present in the eye (choroids), inner ear, meninges, and the integumentary system [2, 3]. This disease affects mainly patients aged between 20 and 50 years, females (with a female/male ratio of 2:1), Asians, Native Americans, and Hispanics [2]. The origin of this disease remains unknown. The role of genetic factors has been recognized in the pathogenic mechanisms of VKH disease due to its strong association

*Correspondence: moussatoudou@gmail.com
1 Department of Neurology, National Hospital of Niamey, PO Box 238, Niamey, Niger
Full list of author information is available at the end of the article
with certain HLA antigens [1, 4, 5]. According to the data from a systematic review and meta-analysis, HLA-DRB1*0404, HLA-DRB1*0405, and HLA-DRB1*0410 are risk sub-alleles for VKH disease [6]. VKH disease occurs in people with a genetic predisposition who are exposed to one or more environmental triggers. Infectious agents such as Epstein-Barr virus and cytomegalovirus are the mains environmental triggers reported [7, 8].

The usual neurologic manifestations of VKH disease include aseptic meningitis and headaches [9]. However, unusual neurologic manifestations had been reported in VKH disease. We performed the present systematic literature review (SLR) to summarize the different unusual neurologic manifestations reported in VKH disease.

**Methods**

**Study design**

The present study is a SLR focused on the unusual neurologic manifestations of VKH disease. The review protocol was not previously registered. We conducted this SLR according to the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement. All articles included in this SLR are referenced.

**Search strategy**

To carry out this SLR, a literature search was performed on Scopus and Medline via PubMed from 1946 to July 31, 2021. In both electronic databases, the literature search was performed by using the following terms: “Vogt Koyanagi Harada disease” OR “VKH disease” AND “Brain” OR “Spinal cord” OR “CNS” OR “Central nervous system” OR “Neurologic” OR “Peripheral nervous system” OR “Polyneuropathies. The search was conducted in the English language.

**Study selection**

All records identified during the literature search were independently screened by the two authors (MTD and AKIM). The first stage consisted of screening based on titles and abstracts of all identified records through the literature search to identify potentially eligible articles. The second stage consisted of screening based on the full text of all potentially eligible articles to identify articles meeting the inclusion criteria of our SLR. The sole inclusion criteria for our SLR was VKH disease associated with unusual neurological involvement. We made no restrictions on the language.

**Data extraction and analysis**

We manually extracted the following data from the included studies: study authors, year of publication, country, study design, age, gender, and main results. Data extraction was completed independently by the two authors (MTD and AKIM), and any discrepancies were resolved by discussion and consensus. We reported our findings using qualitative descriptive statistics. A meta-analysis was not performed because the included studies were all case reports.

**Ethics statement**

Ethics approval and written informed consent were not required for this SLR because all the data were extracted from public access databases and no primary data were collected or generated during the review process.

**Results**

The studies selection process was showed in Fig. 1. Our literature search yielded 417 total articles (PubMed = 334, Scopus = 83). After reviewing titles and abstracts, 370 studies were excluded because they were unrelated to the aim of our SLR. Among the remaining 47 potentially eligible studies, 11 were excluded for duplicity. After reviewing the full texts of the remaining 36 articles, 4 studies were excluded because they reported usual neurologic manifestations of VKH disease. Eventually, 32 studies [10–41] fulfilled our inclusion criteria and were retained in our SLR.

**Study characteristics**

Our SLR included a total of 32 publications, comprising 43 patients (22 men and 21 women, of which 62.8% were younger than 50 years). Regarding the study design, all studies were case reports and published between 1981 and 2021. There were 27 articles in English and 5 in Japanese. Table 1 summarizes the characteristics of the included studies.

**Unusual neurologic manifestations of VKH disease**

Table 1 summarizes the main unusual neurological manifestations in this SLR. CNS involvement was the most reported (93%) in VKH disease. Only reported by one study [33], peripheral nervous system involvement represents 7% of cases. Among the CNS involvement (40 cases), cerebral lesions represented 52.5% of cases (21/40), followed by the optic nerve lesions (13/40 = 32.5%) and the spinal cord (6/40 = 15%).

The cerebral lesions were parenchymal inflammatory lesions in the white matter or posterior fossa with or no contrast enhancement (16.3%) [19–24, 34], leptomenigitis (9.3%) [27–30], pachymeningitis (7%) [19, 31, 32], meningoencephalitis (2.3%) [35], ischemic stroke (4.6%) [36, 37], hemorrhagic stroke (2.3%) [38], transient ischemic attack (2.3%) [39], and hydrocephalus (2.3%) [40].
The optic nerve lesions were anterior ischemic optic neuropathy (20.9%) [10–13] and optic neuritis (9.3%) [25, 26]. Concerning spinal cord lesion, it was mainly myelitis (14%) [14–18].

**Discussion**

In the present SLR, we found that unusual neurologic manifestations of VKH disease are rare, and all reported studies are case reports. The evidence level of nervous system involvement or neurologic manifestations of VKH disease is moderate to high quality. In the majority of studies included in this SLR, the patients had benefited from an exhaustive exploration that had permitted ruling out other conditions such as Behçet’s disease, neuromyelitis optica spectrum disorder, tuberculosis or sarcoidosis. All included patients had an established diagnosis of VKH disease. The patients with ischemic stroke [36, 37] had undergone a work-up that had permitted ruling out a cardiac or atherosclerotic origin.

VKH disease is a systemic autoimmune disorder affecting melanocyte-rich tissues, such as the eyes, inner ear, meninges, and skin [2, 3]. The unusual neurologic manifestations of VKH disease are various and dominated by cerebral involvement, like inflammatory parenchymal lesions. The precise pathophysiological mechanism by which VKH disease leads to cerebral or spinal cord involvement is unclear. The brain, optic nerves (prolongation of the brain), and the spinal cord are surrounded by meninges. These meninges contain strongly melanocytes which are T cell targets in VKH disease [42]. That could explain the cerebral involvement, optic nerves (optic neuritis), and the spinal cord observed in VKH disease.
**Table 1** Characteristics of included studies

| Year   | First author | Study Design | Country      | Sex/Age (years) | Presenting Neurological Symptoms and Signs | Neurologic Manifestations Reported | Diagnostic criteria |
|--------|--------------|--------------|--------------|-----------------|------------------------------------------|-----------------------------------|---------------------|
| 2021   | Yu et al.    | CR           | South Korea  | M/43            | Paraplegia, sensory deficit in both lower extremities, positive Babinski’s sign, dysuria | Longitudinal myelitis               | Complete VKH disease according to Revised Diagnostic Criteria [9] |
| 2020   | Patyal et al.| CR           | India        | F/28            | –                                         | AION                              | Probable VKH disease [9] |
| 2020   | El Beltagi et al. | CR  | U.S.A       | M/38            | –                                         | Leptomeningitis of the cerebellar folia | Probable VKH disease [9] |
| 2019   | Le et al.    | CR           | Australia    | F/69            | Ataxia of the 4 limbs                      | Medial temporal lobes leptomeningitis | Incomplete VKH disease [9] |
| 2018   | Pellegrini et al. | CR  | Italy       | F/42            | –                                         | Unilateral neuroretinitis           | Complete VKH disease [9] |
| 2017   | Algahtani et al. | CR  | Saudi Arabia| F/39            | Dysarthria, confusion, and status epilepticus | Hyperintense periventricular lesions mimicking multiple sclerosis | Incomplete VKH disease [9] |
| 2016   | Valenzuela et al. | CR  | U.S.A       | M/32            | –                                         | Pachymeningitis along the clivus, Ischemic stroke | Incomplete VKH disease [9] |
| 2014   | Vergaro et al. | CR           | Italy        | M/12            | Choreic movements, unsteady gait          | Diffuse pachymeningitis with cerebellar pontine angle inflammatory lesion | Incomplete VKH disease [9] |
| 2014   | Sheriff et al. | CR           | U.S.A        | F/58            | Peripheral facial palsy, hypoglossal nerve dysfunction, facial hypoesthesia, hemiataxia | –                                 | Incomplete VKH disease [9] |
| 2013   | Gu et al.    | CR           | China        | F/50            | Tetraparesis, positive bilateral Babinski’s sign | Acute myelitis | Incomplete VKH disease [9] |
| 2013   | Kales et al. | CR           | Turkey       | M/27            | –                                         | Hyperintense lesion in the periventricular deep white matter | – |
| 2013   | Naeini et al. | CR           | Iran         | F/57            | Disturbances of consciousness             | Encephalopathy with hyperintensity in the right temporal, both frontal and right parietal lobes | Incomplete VKH disease [9] |
| 2012   | Loh Y        | CR           | U.S.A        | M/35            | Severe vertigo, bidirectional gaze-evoked nystagmus | Basilar leptomeningitis | – |
| 2011   | Lohman et al. | CR           | U.S.A        | M/28            | –                                         | Leptomeningitis of the cerebellar folia and the interpeduncular fossa | Probable VKH disease [9] |
| 2010   | Tang et al.  | CR           | China        | F/16            | Paraparesis, positive bilateral Babinski’s sign | Acute longitudinal myelitis | Incomplete VKH disease [9] |
| 2010   | Han et al.   | CR           | South Korea  | F/54            | –                                         | Anterior temporal lobes pachymeningitis | Incomplete VKH disease [9] |
| 2009   | Baheti et al.| CR           | India        | M/26            | Gaze-evoked nystagmus, bilateral upper and lower limb incoordination, gait ataxia | Cerebellar hemorrhagic stroke | Complete VKH disease [9] |
| Year | First author         | Study Design | Country | Sex/Age (years) | Presenting Neurological Symptoms and Signs | Neurologic Manifestations Reported | Diagnostic criteria                  |
|------|----------------------|--------------|---------|----------------|------------------------------------------|-----------------------------------|-------------------------------------|
| 2009 [10] | Nakao et al | CR | Japan | F/79 | – | AION | Incomplete VKH disease [9] |
|       |                     |              |         | M/65 | – | AION | Incomplete VKH disease [9] |
|       |                     |              |         | M/64 | – | AION | Incomplete VKH disease [9] |
|       |                     |              |         | M/63 | – | AION | Incomplete VKH disease [9] |
|       |                     |              |         | F/54 | – | AION | Incomplete VKH disease [9] |
|       |                     |              |         | M/70 | – | AION | Incomplete VKH disease [9] |
| 2009 [15] | Dahbour SS | CR | Jordan | F/37 | Paraparesis, positive Lhermitte's sign, positive Romberg's test, urinary urgency | Acute myelitis | Complete VKH disease [9] |
| 2009 [20] | Hashimoto et al | CR | Japan | M/28 | One-and-a-half syndrome, facial nerve palsy, stuporous, palate paresis | Brainstem encephalitis | Complete VKH disease [9] |
| 2007 [25] | Rajendram et al | CR | U.S.A | F/35 | – | Optic neuritis | Probable VKH disease [9] |
|       |                     |              |         | F/35 | – | Optic neuritis | Probable VKH disease [9] |
|       |                     |              |         | M/25 | – | Optic neuritis | Probable VKH disease [9] |
| 2007 [40] | Yamamoto et al | CR | Japan | F/43 | Urinary incontinence, disturbance of consciousness | Hydrocephalus | – |
| 2006 [13] | Abematsu et al | CR | Japan | F/51 | – | AION | – |
| 2001 [33] | Najman-Vainer et al | CR | U.S.A | M/43 | Weakness and decreased sensitivity of the lower limbs, abolition of Achilles tendon reflexes and diminution of the patellar tendon reflexes | Guillain-Barré syndrome | Incomplete VKH disease [9] |
|       |                     |              |         | F/63 | Weakness of the 4 extremities and facial muscles, areflexia | Guillain-Barré syndrome | Incomplete VKH disease [9] |
|       |                     |              |         | M/48 | Weakness of the 4 extremities, bilateral facial nerve palsy, areflexia | Guillain-Barré syndrome | Probable VKH disease [9] |
| 2000 [35] | Kamondi et al | CR | Hungary | F/36 | Somnolence, hemiparesis, supranuclear hypoglossal paresis | Meningoencephalitis | – |
| 1999 [12] | Yokoyama et al | CR | Japan | M/68 | – | AION | Contrast enhancement of both the uveas and the cerebellar vermis | – |
| 1995 [23] | Osaki et al | CR | Japan | M/57 | Truncal ataxia | – | – |
Concerning anterior ischemic optic neuropathy (AION), the pathophysiological mechanism of its occurrence is uncertain. The vascularization of the optic disc is organized as follows: 1) the lamina cribrosa region is supplied by centripetal branches directly from the short posterior ciliary arteries (PCAs) or from the circle of Haller and Zinn formed by the short PCAs (when that is present), and 2) the prelaminar region is supplied by the fine centripetal branches from the peripapillary choroidal vessels [43]. Severe uveitis with choroidal involvement causes inflammatory infiltration of the peripapillary choroidal vessels with a high risk of their obliteration that could explain the occurrence of the AION in VKH disease.

Magnetic resonance imaging (MRI) is the preferred imaging technique for detecting brain or spinal cord lesions in patients with VKH disease and helps in the differential diagnosis of VKH disease with multiple sclerosis. MRI can detect the meningeal inflammatory process in patients with VKH disease by showing pachymeningeal or leptomeningeal enhancement.

Peripheral nervous system involvement found in this SLR was Guillain-Barré syndrome [33]. The pathophysiological mechanism of Guillain-Barré syndrome in VKH disease is not well known. Since melanocytes and Schwann cells (myelin-producing cells) had the neural crest as a common embryologic origin [44], it is easy to suppose that a disease involving melanocytes (such as VKH disease) can cause peripheral nervous system involvement.

Limitations
The main limitation of this SLR is that it is mainly based on case reports. However, VKH disease is a rare condition and its unusual neurologic manifestations are even rarer, which would explain the small number of reported cases in the literature.
Conclusions

This SLR summarizes the findings of existing studies on unusual neurologic manifestations of VKH disease and provided data on the pathophysiological mechanisms of the occurrence of these neurologic manifestations during this disease. Nervous system involvement or neurologic manifestations of VKH disease have been well documented in patients included in this SLR. To our knowledge, our study is the sole systematic review performed on the unusual neurologic manifestations of VKH disease.

Abbreviations

AION: Indicates anterior ischemic optic neuropathy; CNS: Central nervous system; MRI: Magnetic resonance imaging; SLR: Systematic literature review; VKH: Vogt-Koyanagi-Harada.

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Authors' contributions

MTD (neurologist, MD) is the first. AKIM (internist, MD) is co-author.

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Ethics approval and consent to participate

NA

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NA

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Author details

1 Department of Neurology, National Hospital of Niamey, PO Box 238, Niamey, Niger. 2 Department of Medicine and Medical Specialties, Regional Hospital Center of Dosso, Dosso, Niger.

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