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CASE BASED REVIEW

Rheumatoid meningitis without a history of rheumatoid arthritis: a case report and literature review

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Abstract

Rheumatoid meningitis (RM) is a rare extra-articular manifestation of rheumatoid arthritis, usually with non-specific symptoms. In most cases, head magnetic resonance imaging (MRI) shows lamellar enhancements in leptomeninges and pachymeninges, but definitive diagnosis relies on meningeal biopsies. Here, we reported a 43-year-old RM patient without a previous history of rheumatoid arthritis. He came to seek medical assistance because of fever and headache. The head MRI showed bilateral enhancements in leptomeninges and pachymeninges, and blood tests showed that serum IgM rheumatoid factor (RF) (1010.0 IU/ml) and anti-cyclic citrullinated peptide (CCP) antibody (654.24 RU/ml) became positive with a further increase with the progression of the disease. After treatment with steroids, clinical symptoms were relieved. We also reviewed previous history, symptoms, and serum, cerebrospinal fluid and imaging findings in 15 RM cases without a history of rheumatoid arthritis published since 2010. Consistent with previous reported cases, the current case suggests importance of meningeal biopsies and increases in serum RF and anti-CCP antibody in diagnosis of RM. In addition, previous joint symptoms and chronic headaches, and leptomeningeal and pachymeningeal lesions on head MRI are also of great significance for the diagnosis.

Keywords Rheumatoid meningitis · Rheumatoid arthritis · Meninges

Introduction

Rheumatoid meningitis (RM) is a rare extra-articular manifestation of rheumatoid arthritis (RA), mainly involving leptomeninges and pachymeninges [1]. It can occur at various stages of the disease usually with non-specific clinical manifestations and can imitate various neurological diseases such as malignant tumors or infections in the central nervous system [1, 2]. At present, most cases of RM reported in the literature had a history of RA, and RM without a history of RA was relatively rare. The terms "rheumatoid meningitis" and/or "rheumatoid leptomeningitis" were used in PubMed identifying 15 cases RM without a history of RA in the English literature, published from January 2010 to June 2022 [3, 4, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 19, 20]. It is currently believed that the diagnosis of the disease depends on meningeal biopsies [1], which pose a great challenge to clinical diagnosis, especially when a meningeal biopsy is not available or the patient refuses to do the biopsy.

Recently, we encountered a patient without RA-related history and clinical manifestations, who was initially misdiagnosed as an intracranial infection, and finally diagnosed as RM with a meningeal biopsy. We also reviewed 15 cases of RM without a history of RA published up to June 2022, focusing on medical history, symptoms, findings of serum and cerebrospinal fluid tests, and magnetic resonance imaging (MRI)/computed tomography (CT) scans (see Table 1).

Case presentation

The patient was a 43-year-old man who was admitted to the hospital in January 9, 2019 due to repeated headaches. The headache started after a cold two months before admission, which manifested as forehead pain and lasted for several hours for each episode, and occurred frequently in the afternoon and night. The patient also showed a gradual change in

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Table 1 Summary of Reports of RM cases without a history of RA since 2010

	Patient (Age, sex)	Medical History	Symptoms	CSF				Serum		MRI/CT
				Intracranial pressure (cmH_2O)	Cells (per μL)	Protein (mg/L)	Glucose (mg/L)	RF (IU/mL)	Anti-CCP (IU/mL)	
Lee et al. [3]	72, F		Stroke symptoms, Mental disorder, epilepsy	–	12 Cells (L: 94%, N: 1% M: 5%)	25	58	Normal	Normal	Enhancement in leptomeninges and pachymeninges over cerebellum on the MRI scans
Magaki et al. [4]	37, M	Chronic headache relieved for half a year by the use of small dose of glucocorticoid which was intended to treat his facial paralysis	Headache	–	16 Cells (L: 96%)	35	89	83	> 250	Abnormal leptomeningeal enhancement in the left frontal and parietal sulci, with associated fluid-attenuated inversion recovery (FLAIR) hyperintensity within the sulci, without edema or mass effect
Magaki et al. [4]	62, F	Mild arthralgia for 12 months	Stroke like Symptoms, confusion and seizures	–	–	–	–	–	–	Diffuse leptomeningeal enhancement and white matter low density areas compatible with a meningitic process on the CT scans
McKenna et al. [5]	59, M	Osteoarthritis for 10 years	Stroke like Symptoms with fever and chills	–	*L: 96%	672	46.8	88.2	> 340	Increased signal with obliteration of sulci due to leptomeningeal thickening over the right frontoparietal lobe on the MRI scans

Table 1 (continued)

	Patient (Age, sex)	Medical History	Symptoms	CSF				Serum		MRI/CT
				Intracranial pressure (cmH_2O)	Cells (per μL)	Protein (mg/L)	Glucose (mg/L)	RF (IU/mL)	Anti-CCP (IU/mL)	
Kim et al. [6]	66, M	Intermittent multiple joint pain	Epilepsy, psychotic and stroke-like symptoms	Normal	11 white blood Cells	Normal	Normal	Increased	1448	Leptomeningeal enhancement of the CSF spaces with subdural empyema along the falx and right convexity in the MRI scans and FLAIR images revealed hyperintense lesions in the bilateral frontal cortical areas
Schuster et al. [7]	48, M	Mild persistent headache and night sweating for 3 months	Stroke symptoms	–	300 mono-nuclear cells	137	–	298	> 340	Leptomeningeal enhancement of the CSF spaces, with subdural empyema along the falx and right high convexity and Flair images revealed hyperintense lesions in the bilateral frontal cortical areas

Table 1 (continued)

	Patient (Age, sex)	Medical History	Symptoms	CSF				Serum		MRI/CT
				Intracranial pressure (cmH_2O)	Cells (per μL)	Protein (mg/L)	Glucose (mg/L)	RF (IU/mL)	Anti-CCP (IU/mL)	
Finkelshtein et al. [8]	66, M	Right ring finger Pain and limited flexion for 18 months	Epileptic seizure	–	Normal	Normal	Normal	25	266	Effacement of the sulci in the right upper parietal area with Regional dural and leptomeningeal enhancement, high-signal intensity in the sulci on FLAIR sequence, and two small high-signal subcortical foci in this area on T2/FLAIR sequences. No restriction on diffusion-weighted imaging sequence was found
Inan et al. [9]	70, M	Arthralgia for ten years	Fever, headache, nausea, vomiting	–	140 Cells (N: 62%, L: 28%)	113	34	108	–	–
Kolenc et al. [10]	77, F	–	Epilepsy	–	Normal	Normal	–	171.7	405.3	The diffusion-weighted imaging (DWI) and FLAIR images showed hyperintensity of the subarachnoid space in the left frontal and parietal lobes. Gadolinium enhanced T1 weighted image (T1WI) showed the pia mater enhancement
Jessee et al. [5]	68, F	Osteoarthritis	Epileptic seizures, fatigue, stroke-like symptoms	–	8 Cells (N: 5%, L: 61%, M: 4%)	64	56	208	95.8	Hypoattenuation of white matter in the left frontal lobe with leptomeningeal enhancement on the head CT

Table 1 (continued)

	Patient (Age, sex)	Medical History	Symptoms	CSF				Serum		MRI/CT
				Intracranial pressure (cmH_2O)	Cells (per μL)	Protein (mg/L)	Glucose (mg/L)	RF (IU/mL)	Anti-CCP (IU/mL)	
Lubomski et al. [13]	41, M	Polyarthritis of large and small joints for 18 months	Psychosis irritated, delusion of being persecuted	–	1 Cell (M: 100%)	39	61.2	8 (Negative)	> 600	Leptomeningeal enhancement over both hemispheres especially the right frontal lobe on MRI scans
Shibahara et al. [14]	63, M	–	Fever, headache and vomiting	–	37 Cells (L: 100%)	98	Normal	140	472	Leptomeningeal enhancement over both hemispheres especially the right frontal lobe on MRI Scans
Kira et al. [15]	93, M	No	Unconsciousness, epileptic seizure	150	3 Cells (N:66.7%;)	68	64	223	306	Hyperintensity of the subarachnoid space in the left frontal and parietal lobes in the diffusion-weighted imaging (DWI) and FLAIR images. Gadolinium enhanced T1 weighted image (T1WI) showed the pia mater enhancement
Rodriguez Alvarez et al. [19]	60, M	*Naegleria fowleri* infection in the central nervous system	Leg weakness and fever	–	56 (97% L)	27.7	70	579	> 125	Leptomeningeal enhancement at bilateral frontal lobe and parietal lobe, interhemispheric fissure

Table 1 (continued)

	Patient (Age, sex)	Medical History	Symptoms	CSF			Serum			MRI/CT
				Intracranial pressure (cmH_2O)	Cells (per μL)	Protein (mg/L)	Glucose (mg/L)	RF (IU/mL)	Anti-CCP (IU/mL)	
Chouk et al. [20]	62, F	Medical history of acute rheumatic fever in childhood	Sudden numbness of the proximal upper right limb	–	20	51	–	90	340	Supra-tentorial thickening of leptomeninges in hypersignal on T2 FLAIR and diffusion sequences and post-contrast enhancement in leptomeninges

RA Rheumatoid arthritis; *RF* rheumatic factor; *RM* rheumatoid meningitis; *RNP* Ribonucleoprotein; *N* neutrophils; *M* monocytes; *L* lymphocytes

– Did not mention in the references

*The total number of cells in the CSF was not mentioned in the reference

Table 2 Results of CSF and serum tests before and after treatment

Test	Days after admission				Normal range
	2 d	7 d	14 d	30 d	
CSF					
Pressure (cmH_2O)	320	250	220	180	80–180
Leukocytes (per μL)	90	180	150	30	0–10
Neutrophils (%)	20%	15%	18%	0	0–6%
Lymphocytes (%)	73%	67%	75%	99%	40–80%
Monocytes (%)	7%	18%	7%	1%	15–45%
Protein (mg/L)	908	1112	1205	556	200–400
Glucose (mmol/L)	3.0	2.2	2.5	3.3	2.8–4.4
Chloride (mmol/L)	117.0	126.0	119.0	123.0	120.0–130.0
Serum					
RF (IU/ml)	–	–	1010.0	1100.0	0–20
Anti-CCP (RU/ml)	–	–	654.24	> 1678.75	0–25
CRP (mg/L)	17.60	–	29.20	2.63	0–8.0

– not performed

personality and manifested as easily irritated and angered. Physical examination showed inattention, emotional instability, positive meningeal irritation signs (neck stiffness), normal cranial and spinal nerve examinations, and negative pathological neurological signs.

After admission, the patient was found to have a fever and his body temperature fluctuated between 37.8 and 38.9 °C. The headache was relieved and the body temperature returned to normal with the use of ibuprofen. On the second day after admission, a lumbar puncture was performed, and the results of the cerebrospinal fluid (CSF) tests were shown in Table 2. Briefly, the CSF pressure was 320 cmH_2O (normal range: 80–180 cmH_2O), protein 908 mg/L (200–400 mg/L), glucose 3.0 mmol/L (2.8–4.4 mmol/L), chloride 117.0 mmol/L (120.0–130.0 mmol/L), white blood cell counts 90/μL (0–10/μL), neutrophils 20.0%, lymphocytes 73.0%, and monocytes 7.0%.

Autoimmune encephalitis-associated IgG antibodies in blood and CSF, such as antibodies against N-methyl-D-aspartate receptor subunit NR1 (NMDAR-NR1), α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor subunits 1 and 2 (AMPAR1/2), gamma-aminobutyric acid B receptor subunits B1 and B2 (GABABR1/2), glutamate decarboxylase 65 (GAD65), glycine receptor (GlyR), contactin-associated protein-like 2 (CASPR2), leucine-rich glioma-inactivated protein 1 (LGIP1), metabotropic glutamate receptor 5 (mGluR5) and dipeptidyl-peptidase-like protein 6 (DPPX) and Neurexin3α, examined with the cell

transfection method were all negative. In addition, CSF tests showed capsular polysaccharide antigen negative, Indian ink staining of cryptococci negative, modified acid-fast staining negative as well as DNA virus PCR negative. Moreover, blood tests showed TS-POT.TB negative, C-reactive protein 17.60 mg/L (0–8.0 mg/L), erythrocyte sedimentation rate 39 mm/h (0–15 mm/h), and normal blood routine.

Electroencephalographic (EEG) examination showed diffuse slow waves, and head magnetic resonance examination (see Fig. 1) demonstrated increases linear signals in bilateral frontal and parietal sulcus with the diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) sequences, and enhanced signals in pachymeninges and some leptomeninges observed in the MRI contrast-enhanced scans.

Considering the possibility of viral meningitis in the initial diagnosis, antiviral therapy with ganciclovir 0.4 g/d for 6 days was initiated. However, the patient's headache and fever did not improve. On the 7th day after admission, a lumbar puncture was performed (see Table 2) and the CSF was examined. The CSF tests showed that the pressure was 250 cmH_2O, protein 1112 mg/L, glucose 2.2 mmol/L, chloride 126.0 mmol/L, and white blood cell count 180/μL (neutrophils 15.0%, lymphocytes 67.0%, and monocytes 18.0%). In addition, cytological examinations of the CSF showed no malignant cells. The CSF sample was also processed for examination of bacterial genes on the Berry Genomics NextSeq CN500 platform in the Laboratory of the Wuhan Kindstar Diagnostics Co., Ltd. (Wuhan, China), with the use of a protocol for the validated metagenomic Next-Generation Sequencing assay, as described previously [21, 22] and showed no pathogenic microorganisms.

On the 14th day after admission, a lumbar puncture was performed again (see Table 2) and the CSF tests showed that the pressure was 220 cmH_2O, protein 1205 mg/L, glucose 2.5 mmol/L, chloride 119.0 mmol/L, white blood cell count 150/μL (neutrophils 18.0%, lymphocytes were 75.0%, monocytes 7.0%), and IgG4 negative. Blood tests showed erythrocyte sedimentation rate was 44 mm/h, C-reactive protein 29.20 mg/L (0–8.0 mg/L), rheumatoid factor (RF) 1010.0 IU/ml (0–20 IU/ml), and anti-CCP IgG antibody 654.24 RU/ml (0–25 RU/ml) (see Table 2). However, anti-double-stranded DNA, anti-nuclear (ANA, IgG), anti-smith, anti-neutrophil cytoplasmic (ANCA, IgG), anti-Ro (SSA)/La (SSB), anti-cardiolipin, anti-phospholipid, and other lupus antibody tests were all negative.

The patient's medical history was retaken and it was found that the patient often experienced joint pain in mainly

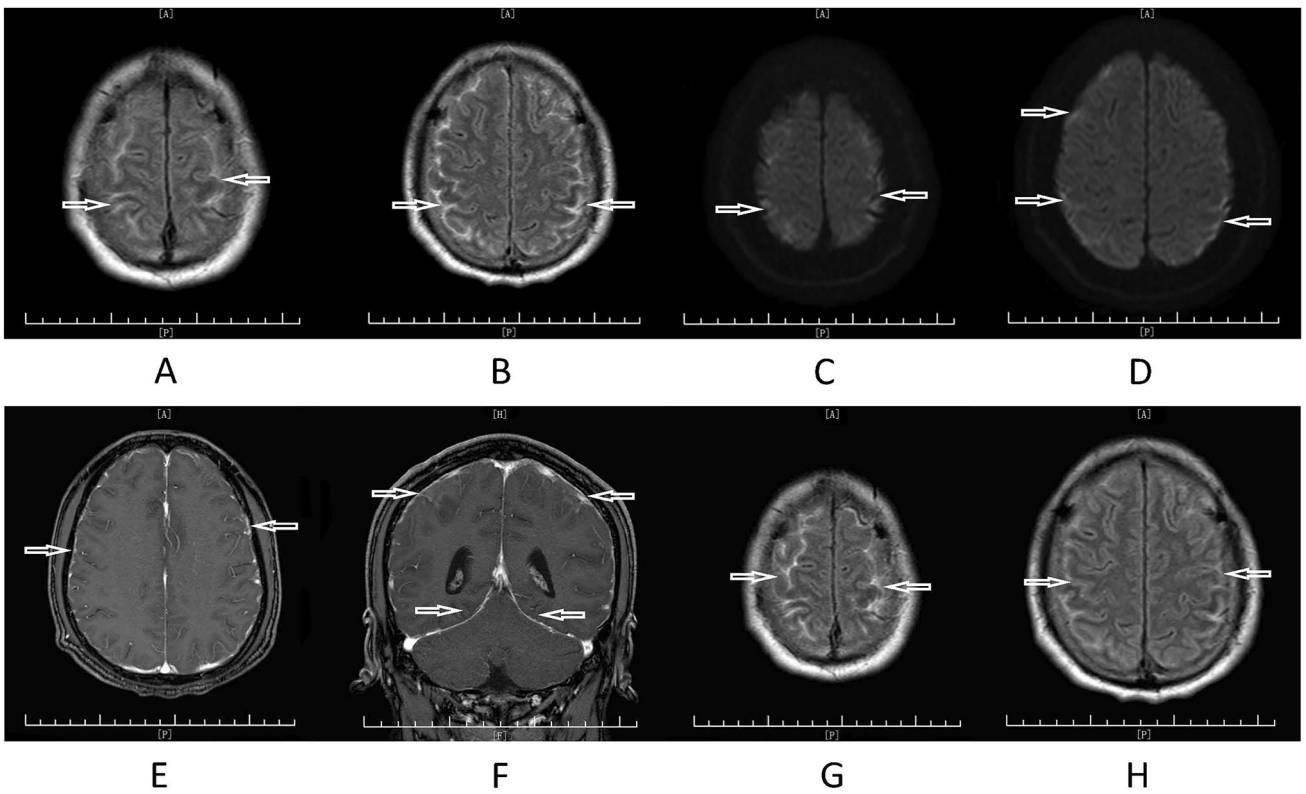

Fig. 1 Head magnetic resonance imaging with FLAIR (**A**, **B**), DWI (**C**, **D**), and enhanced T1WI (**E**, **F**) and FLAIR sequence (**G**, **H**). The leptomeningeal signals on bilateral frontal and parietal lobes were increased in the DWI and FLIAIR images, and more significantly elevated in enhanced T1WI and FLAIR sequence scans

in the metacarpophalangeal, wrist and ankle joints in the past two years. There was no fever, joint swelling and morning stiffness during the attack. After taking ibuprofen, the pain was relieved. The patient did not pay any attention or go to hospital. X-rays of hand and shoulder joints did not show any abnormality. The meningeal biopsy showed a mixture of many lymphocytes and plasma cells, as well as a small number of neutrophils.

After treatment with oral prednisone 60 mg/d for 7 days, the patient's headache and fever were relieved. On the 30th day after admission, a lumbar puncture (see Table 2) was performed again. Examination of CSF showed the pressure was 180 cmH_2O, protein 556 mg/L, glucose 3.0 mmol/L, chloride 123.0 mmol/L, and white blood cells 30/μL (lymphocytes 89.0%, and monocytes 11.0%). The serum level of rheumatoid factor was 1100.0 IU/ml, and anti-CCP antibody was higher than 1678.75 RU/ml (1 RU/mL roughly equates to 2 pg/mL according to the manufacturer). On the 36th day after admission, the patient was discharged from the hospital with normal body temperature and personality, and no headache. He continued to take oral prednisone 30 mg/d. Six months after discharge, the patient was followed up by telephone. The patient's previous symptoms did not return and no symptom of arthritis was reported. The dose of prednisone was then reduced to 10 mg/d for maintenance. The patient revisited 1 year after the discharge and did not report any discomfort and the head MRI was also normal. Since examination of serum RF and anti-CCP antibody showed they were in the normal range (RF: 5 IU/ml, and anti-CCP antibody: 16 RU/ml), the tests needed to be repeated but the patient was unavailable until 2 months later. The repeated tests showed RF and anti-CCP antibody were positive (RF: 90 IU/ ml, anti-CCP antibody: 200 RU/ml).

Discussion

RM can occur in patients with RA, but it almost always occurs in patients with a long course of seropositive RA, even if RA has been alleviated [3, 16, 17]. However, there are a small percentage of patients with central nervous system involvement earlier than the onset of arthritis [3, 7]. These patients are often misdiagnosed in the early stage of the disease even for many times, which often poses great challenges to clinicians in the diagnosis of the disease and delays the treatment of the patients. Our patient had no history of RA, which led to misdiagnosis at the initial stage and difficult diagnosis process. This situation was very common in the cases reported in the literature. Almost all cases were not diagnosed right away in the early stage of the disease. The diagnosis of RM for the case that we reported was considered only when we found increased titers of RF and anti-CCP antibody. Then, we took the patient's medical history again and found that the patient had pain in the limb joints in the past two years and ibuprofen could relieve it. Later, MRI scans and biopsy of meninges were performed to confirm the diagnosis.

RM could be diagnosed in patients without a diagnosis of RA although 60.0% (9/15) of patients had rheumatoid disease-related clinical symptoms in the literature that we reviewed. The symptoms included joint pain, headache and relatively mild fever. Among them, joint symptoms most frequently occurred. The symptoms in most patients could be relieved with nonsteroidal anti-inflammatory drugs and were often ignored. Therefore, RM patients without a diagnosis of RA often have symptoms related to rheumatoid diseases (especially joint symptoms), which might provide a very meaningful hint for the diagnosis of RM.

Fever, headache, and mental abnormalities are common clinical manifestations in the course of RM. Because RM can imitate various neurological diseases, the clinical manifestations are diverse, and may include headache, seizures, behavior changes, dementia, and unconsciousness, and have hydrocephalus, venous sinus thrombosis, Parkinson's disease, and meningitis, stroke and cranial neuropathy-like symptoms [3]. Nissen et al. [1] reviewed 47 RM patients and found that 70% of them had stroke-like symptoms, and 36% had epilepsy, respectively. We reviewed 15 RM cases without a history of RA here and found that 60.0% (9/15) had epilepsy, 53.3% (8/15) had stroke -like symptoms, 33.3% (5/15) had neuro-psychiatric manifestations, and 26.6% (4/15) had fever and headache.

RM can affect leptomeninges, pachymeninges, or both at the same time, most of which manifests as asymmetrically localized involvement of meninges, although diffused involvement of meninges can occur in a small portion of RM patients [7]. MRI has high sensitivity in detecting meningeal inflammation [4] and is a very effective auxiliary method in the diagnosis of RM, usually showing asymmetric leptomeninges and pachymeninges enhancement [1]. Nissen et al. [1] analyzed the MRI/CT imaging of 29 RM cases with a history of RA, and found symmetric meningeal involvement was clearly present in 62% of patients. Here, we reviewed the MRI/CT imaging studies of 15 patients with no history of RA (Head MRI: 13/15; Head CT 2/15), found that 53.3% (8/15) patients showed asymmetrical frontal and/or parietal meningeal signals abnormally increased, 40.0% (6/15) patients showed unilateral meningeal signals abnormally increased, and 6.7% (1/15) patients had normal head magnetic resonance imaging. The patient in this case report showed bilateral symmetrical and diffuse abnormal increase in meningeal signals in the MR imaging, which is relatively rare in RM, even in RM patients with a history of RA. It has never been reported such MR imaging finding in RM patients without a history of RA.

The CSF results in RM patients were variable. All 15 patients underwent lumbar punctures and CSF examinations, 2 patients were reported to have normal CSF pressure, and 13 patients were not reported in the literature. The increased CSF pressure was found in 3 occasions in the patient reported here before treatment. The reason for increases in CSF pressure in the patient might be due to bilateral symmetric diffuse leptomeningeal and pachymeningeal inflammation, resulting in disrupting CSF circulation. In addition, the results of routine biochemical tests of CSF in the RM patients without a history of RA were also variable. They usually manifested as elevated CSF protein (6/10), normal glucose (9/11), and mild to moderate white blood cell increase (9/12). In addition, increased lymphocytes (9/12) and a small number of neutrophils (2/10) were also found. The patient reported here underwent lumbar punctures three times before treatment, and moderately increased white blood cells (mainly lymphocytes, 15–20% were neutrophils), elevated CSF protein, and slightly lower glucose were found, which were consistent with reports in the literatures [1, 16, 18].

Serum RF and especially anti-CCP antibody are very important indicators for the diagnosis of RA. It has been suggested in literature that anti-CCP antibody are highly specific for RA (> 90% specificity) [14]. However, serum RF and anti-CCP antibody might not be specific biomarkers for evaluation of RM treatment [1]. Nevertheless, the sensitivity of serum RF and anti-CCP antibody in diagnosis of RM is quite high. We reviewed and analyzed serum RF and anti-CCP antibody in 15 RM patients with no history of RA and found increased RF occurred in 12/15 (80.0%) patients and increased anti-CCP antibody occurred in 12/15 (80.0%) patients. This suggests that serum RF and anti-CCP antibody are positive in most RM patients with no history of RA. The patient reported here also showed increased serum RF and anti-CCP antibody, which was consistent with the reports in the literature [1, 2, 7, 18]. Furthermore, after treatment, the CSF markers and symptoms in the patient were significantly improved, but serum RF and anti-CCP antibody continued to increase. The decrease of serum RF and anti-CCP antibody was much slower than improvements of symptoms and falling of CSF markers, even if it could happen. This indicates that serum RF and anti-CCP antibody may not be suitable for treatment evaluation. Nissen et al. [1] have proposed that RF, anti-CCP antibody and CXCL-13 in CSF can be used as potential biomarkers, not only for diagnosis of RM, but also for the evaluation of treatment response. However, further studies are needed to clarify the issue.

The consensus on the management of rheumatoid meningitis is lacking. It was reported that the induction therapy for RM included oral/intravenous steroids as initial therapy in 97% of the cohort [19]. In the current case, steroid pulse was used as initial treatment, followed by a prednisone taper, which was consistent with common practice reported in the previous study [19]. The patient's symptoms improved rapidly and the patient was followed up for half a year without recurrence. Due to the satisfactory effects of steroid, biological agents and/or immunosuppressive agents were not added. However, there is some evidence suggesting that steroid therapy alone may not be sufficient, and other drugs, such as immunosuppressants, should be added [9]. It was reported that the most common combination therapy for RM induction was steroids, plus biologics, and/or immunosuppressants (53.3%), followed by steroids/disease-modifying-antirheumatic drugs (DMARDs) in 16.6% [19]. Further studies are needed to examine whether there are any differences in outcome between those treatments.

In short, RM without a history of RA is very easy to miss the diagnosis and misdiagnosis especially when meningeal biopsy is not available or not permitted. Increased serum RF, anti-CCP antibody and previous rheumatoid disease-related symptoms including often overlooked mild symptoms are very suggestive for diagnosis, and head MR imaging and meningeal biopsy are needed to confirm the diagnosis.

Author contributions GH: conceptualization; data curation; investigation; writing—original draft, review and editing; LW: data curation; investigation; writing—review and editing; ZM: data curation; investigation; writing—review and editing; DY: conceptualization; resources; funding acquisition; project administration; supervision; writing—original draft, review and editing.

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Availability of data and materials The dataset supporting the conclusions of this paper is shown within the article and no additional data could not be shared publicly due to patients' privacy.

Declarations

Conflict of interest The authors report no conflicts of interest in relation to this case report.

Informed consent Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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