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CASE REPORT

Reversible posterior leukoencephalopathy syndrome in a patient with Takayasu arteritis

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Abstract Reversible posterior leukoencephalopathy syndrome (RPLS) has been identified in several connective tissue diseases. However, there are no reports of RPLS associated with Takayasu arteritis (TA). We report the first case of TA associated with RPLS. A 23-year-old woman presented with sudden headache and vomiting, followed by generalized tonic–clonic seizures and mental changes two weeks after administration of oral prednisolone. MRI showed hyperintense signals on T2 and FLAIR images in the bilateral temporal–parietal–occipital lobes, left frontal lobe, and left cerebellar hemisphere. Three weeks after starting control of convulsions and blood pressure with plasmapheresis, high-dose methylprednisolone, and cyclophosphamide, the clinical manifestations and abnormal signals on MRI completely resolved. These reversible clinical and radiological changes are consistent with vasogenic edema in the central nervous system, indicating RPLS. Although high-dose methylprednisolone and cyclophosphamide are thought to cause RPLS, we think that it is justified to use these agents, at least in difficult cases, for making a clear-cut differentiation from CNS vasculitis, as long as blood pressure and fluid volume are well controlled. Moreover, we suggest that RPLS should be included in differential diagnosis of acute neurological changes in connective tissue diseases, including TA.

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Introduction

Takayasu arteritis (TA) is a systemic inflammatory disease which affects the aorta and its branches. The inflammatory processes cause narrowing, occlusion, and/or dilation of the arteries resulting in a variety of symptoms. Involvement of the carotid and vertebral arteries induces cerebrovascular manifestations such as vertigo, syncope, headache, and convulsions mainly due to cerebral ischemia. However, there are no reports regarding reversible posterior leukoencephalopathy syndrome (RPLS) associated with TA [1–5].

RPLS is a new clinico-radiological entity, manifested radiologically by reversible edema in the cortical and subcortical white matter of the parietal-occipital lobes. Headache, seizures, and mental changes are the prime clinical manifestations [6]. RPLS can be associated with hypertensive encephalopathy, renal failure, blood transfusion, and eclampsia [6], and may also be caused by drugs such as immunosuppressive therapy, interferon alpha, cytotoxic chemotherapy, and corticosteroids [6–9, 13–16, 18–31]. Moreover, RPLS has been identified in several connective tissue diseases such as systemic lupus erythematosus (SLE), Wegener's granulomatosis (WG), Churg–Strauss syndrome (CSS), microscopic polyangitis (mPA), and Henoch–Schonlein purpura (HSP) [6–31].

We describe herein a patient with TA who developed acute neurological changes that were initially attributed to vasculitis. These changes were subsequently found to conform with RPLS. Our report is the first to describe a case of RPLS associated with TA.

Case report

A 23-year-old woman with TA was admitted because of low-grade fever, fatigue, and lymphadenopathy. The left radial arterial pulsations were diminished, and the blood pressure in the left arm was reduced (right: 134/67 mmHg, left: 105/46 mmHg). The laboratory findings were as follows: microcytic anemia (hemoglobin: 6.6 g/dl), leukocytosis (9500/μl), thrombocytosis (93.1×10^4/μl) and increased CRP (20.2 mg/dl). The anti-nuclear antibody was negative. The serum complements level was increased due to inflammation (C3:106 mg/dl, C4:25 mg/dl, CH50: 53 U/ml). The anti-cardiolipin IgG antibody (10 U/ml: normal <10) and anti-cardiolipin(β2GP1 antibody (4.9 U/ml: normal <3.5) were slightly positive. Renal function was normal (BUN: 10.0 mg/dl, Cr: 0.48 mg/dl), but urinalysis showed microscopic hematuria (RBC: 10/HPF) and proteinuria (0.1 g/day). Positron emission tomography (PET) scanning showed increased uptake of fluorodeoxyglucose (FDG) in the subclavian, thoracic, axillary, and carotid arteries (shown in Fig. 1a). Magnetic resonance imaging (MRI) revealed stenosis of the left subclavian artery and wall thickening of the subclavian, thoracic, and carotid arteries (shown in Fig. 1b). Brain MRI/MRA revealed no major abnormalities in the cerebrovascular system except decreased flow signal of the left vertebral artery, suggesting involvement of the disease in this artery. Based on these findings, the patient was diagnosed as having TA, and prednisolone (PSL; total: 30 mg/day, 0.8 mg/kg) was administered on hospital day 4. The fever and fatigue improved, and CRP decreased from 20.2 to 3.2 mg/dl. However, CRP did not become negative. On day 14, therefore, the dose of PSL was increased to 45 mg/day (1 mg/kg). Thereafter, CRP decreased to 0.37 mg/dl, suggesting that the active inflammation of TA was seemingly controlled.

However, on day 18, the patient complained of sudden headache and vomiting. Her blood pressure reached 182/72 mmHg, and serum creatinine became 0.78 mg/dl. Brain CT scanning showed no abnormal findings such as subarachnoid hemorrhage (SAH) and cerebral hemorrhage. Headache and vomiting did not improve for two days. On day 20, MRI showed mild hyperintense signals on T2, high

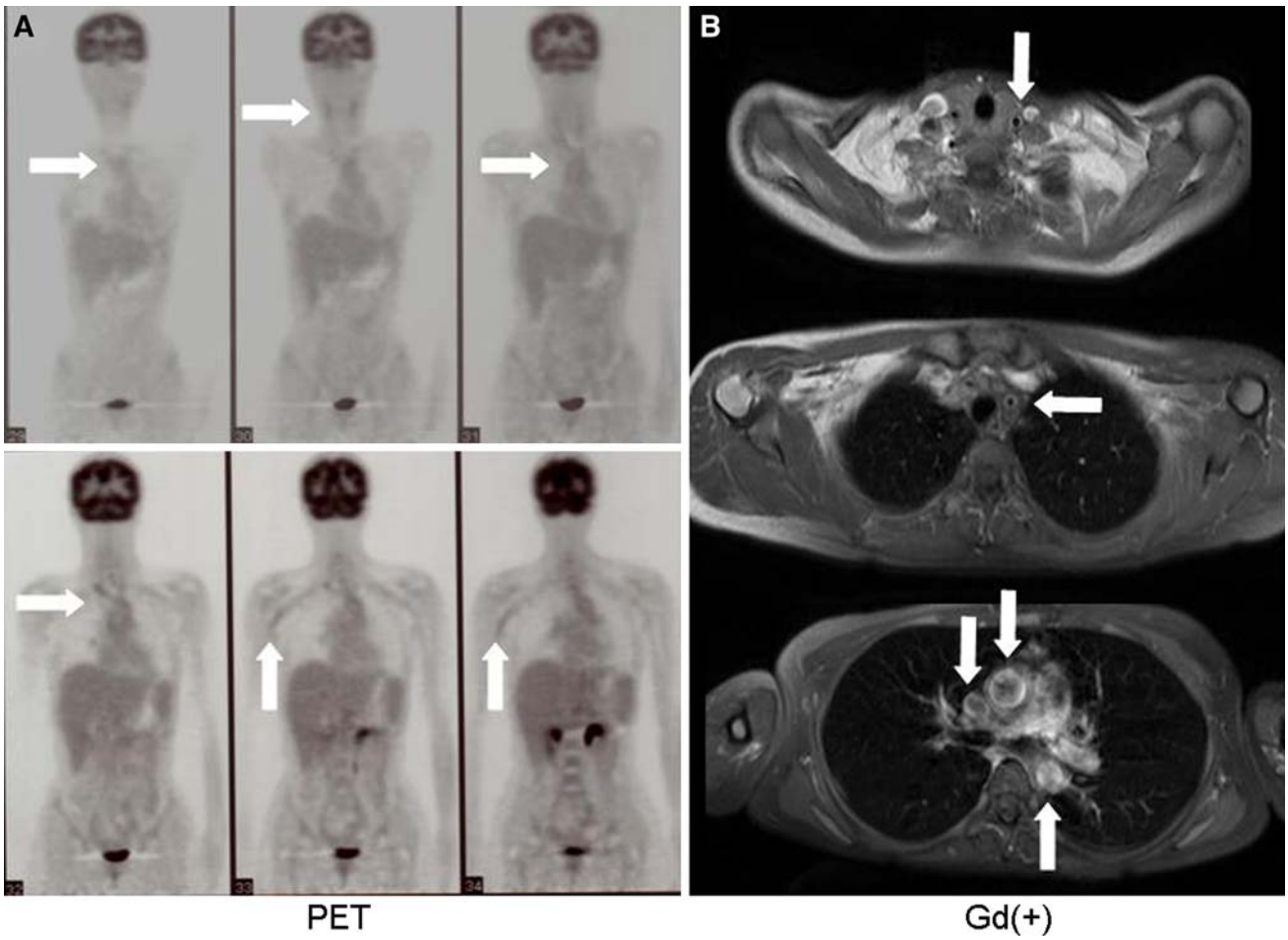

Fig. 1 **a** PET scan showing increased uptake of FDG in the thoracic, axillary, and carotid arteries. **b** MRI revealing wall thickening of the pulmonary, thoracic, and carotid arteries

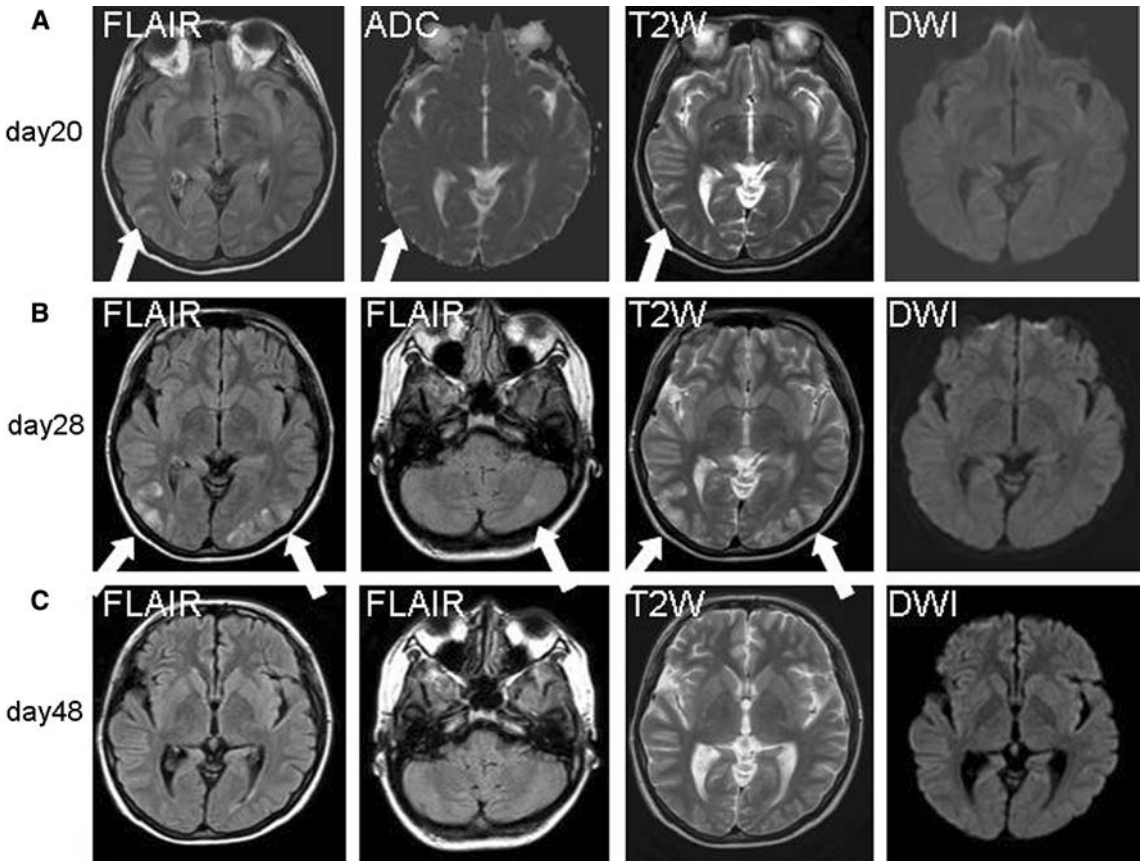

Fig. 2 **a** MRI showed mild hyperintense signals on T2 and FLAIR images and apparent diffusion coefficient (ADC) map of the cortical white matter in the parietal lobes on day 20. There were no significant signal alterations in DWI. **b** MRI showed more hyperintense signals on T2 and FLAIR images of the cortical and subcortical white matter, involving the bilateral temporal–parietal–occipital lobes and the left cerebellar hemisphere on day 28. There were no significant signal alterations on DWI. **c** MRI showed no abnormal signals on T2 and FLAIR on day 48. The previous changes completely resolved. The cerebral ventricles were oppressed in the images on days 20 and 28. The *white arrows* indicate hyperintense signals on T2, FLAIR, and ADC

fluid-attenuated inversion recovery (FLAIR) images, and apparent diffusion coefficient (ADC) map in the bilateral parietal lobes (Fig. 2a). However, there were no significant signal alterations in diffusion-weighted imaging (DWI) (Fig. 2a). The MR images were initially interpreted as showing SAH. Cerebral angiography showed no intracranial aneurysm. Just after angiography, generalized tonic–clonic seizures and mental changes developed, and her blood pressure reached 180/120 mmHg. Diazepam, phenytoin, and nifedipine were administered to control convulsions and hypertension. On the next day (day 21) her blood pressure decreased to 120/70 mmHg, and seizures and mental changes disappeared. Headache was still not improved. Phenytoin and nifedipine were continued for strict control of convulsions and hypertension. On day 23 the mental changes developed again. Contrast MRI showed hyperintense signals on T2 and FLAIR images in the bilateral temporal–parietal–occipital lobes, left frontal lobe, and left cerebellar hemisphere (data not shown). These findings were not a typical pattern of central nervous system (CNS) vasculitis. Directly after contrast MRI, seizures developed again. Her blood pressure reached 216/113 mmHg. Diazepam and glycerol were added to phenytoin and nifedipine to control convulsions and the associated brain edema. The patient also received pulse-dose methylprednisolone (1 g × 3 days), because the possibility of CNS vasculitis could not be completely eliminated. On examination, cerebrospinal fluid was normal (data not shown) on the next day (day 24).

On day 28, generalized tonic–clonic seizures and mental changes developed again. Her blood pressure reached 173/119 mmHg. MRI showed more hyperintense signals on T2 and FLAIR images in the bilateral temporal–parietal–occipital lobes and left cerebellar hemisphere (Fig. 2b). Cyclophosphamide (1 g × 1 day, on day 29) and plasmapheresis (FFP × 4 days, on days 30–33) were added to the anti-hypertensive therapy and anti-convulsive therapy as pulse-dose methylprednisolone could not control seizures and mental changes and did not improve abnormal MRI findings. Also, thiopental was administered as

anti-convulsive agent via intubation as phenytoin could not control the convulsions. On day 34, PSL was switched to betamethasone (6 mg intravenous drip/day) for avoidance of mineral corticoid effects. Seizures disappeared after intubation. Thiopental was discontinued four days after intubation (on day 32). Extubation was performed 11 days after intubation (on day 39). On day 46, physical examination revealed no neurological deficits and CRP became negative. Therefore, betamethasone was decreased to 4 mg per oral/day. On repeating MRI on day 48, the previous changes were completely resolved (Fig. 2c). On day 60, betamethasone was decreased to 3.5 mg per oral/day and the patient was discharged.

Discussion

RPLS has been identified in several connective tissue diseases, for example SLE, WG, CSS, and mPA [6, 13, 24–26, 29, 31, 32] (Table 1). However, there are no reports of RPLS associated with TA. In this report we present the first case of TA associated with RPLS.

An essential factor in the development of RPLS is vasogenic edema in the brain caused by disruption of the blood–brain barrier because of endothelial dysfunction [6, 13, 24–26, 29, 31, 32]. RPLS associated with connective tissue diseases is mostly reported in SLE and mPA affecting small vessels including arterioles, venules, and/or capillaries (shown in Table 1). In those cases, endothelial injury may be caused by the underlying vasculitis itself and/or its associated complications such as vessel spasm and angiopathy. Anti-phospholipid antibodies and anti-endothelial antibodies may also play a role in the endothelial injury [33, 34].

However, many SLE and mPA cases do not develop RPLS. We therefore assume that underlying endothelial injury alone may be insufficient for development of RPLS and that other causative factors may be necessary to trigger disruption of the blood–brain barrier for development of RPLS. First, hypertension is thought to be one aggravation factor. A sudden increase of blood pressure disrupts the auto-regulatory capacity of the cerebral vascular system and triggers disruption of the blood–brain barrier in cases with underlying endothelial injury. As shown in Table 1, most patients with RPLS have severe hypertension (BP: $>$ 170/110 mmHg) [6–10, 12–22, 24–31]. Second, patients with RPLS were previously treated with several agents, for example cyclosporine, cyclophosphamide, and corticosteroids [7, 9, 13–16, 18–27, 29– 31] (Table 1). These agents could be associated with RPLS. Corticosteroids with mineral corticoid effects, in particular, may cause hypertension and induce RPLS. On the other hand, some RPLS patients were treated with high-dose methylprednisolone, cyclophosphamide, and/or plasmapheresis as final resolution [9,14, 16,18, 20, 25–28, 32], as shown in Table 1. There is controversy regarding the relationship of these agents to RPLS. Third, contrast agents are believed to cause osmotic disruption of the blood–brain barrier [35]. These contrast agents may trigger seizures, and seizures may subsequently aggravate the vasogenic edema in brains in which autoregulation has been disrupted.

Taken together, RPLS in connective tissue diseases is closely associated with endothelial injury of small vessels caused by underlying vasculitis, anti-endothelial antibodies, and/or anti-phospholipid antibodies. This disruption of the auto-regulatory capacity of the cerebral vascular system is an important aggravating factor triggering RPLS, and these conditions are induced by several factors such as rapid increase of blood pressure (caused by corticosteroids, renal failure, and volume overload), cytotoxic agents, and/or contrast agents.

TA is categorized as large-vessel vasculitis (Chapel Hill Conference). Therefore, the reasons why RPLS occurs in TA have to be fully discussed. First, we consider that small vessels might have been also injured by poorly controlled vasculitis. Second, anti-phospholipid antibodies were slightly positive in this case and may play a role in the endothelial injury. Third, anti-endothelial antibodies may play a role in the endothelial injury. Anti-endothelial antibodies are found in most patients with TA [34], although we did not investigate anti-endothelial antibodies in our case.

In addition to endothelial injury associated with TA, we consider that hypertension played the most important role in triggering RPLS in this case, as discussed in other collagen diseases. A rapid increase of blood pressure was observed on each occasion of neurological symptoms suspected of RPLS. Our patient developed seizures and mental changes immediately after receiving contrast agents used in angiography and MRI. However, contrast agent was not used at first onset on day 18. Therefore, contrast agents may not be a direct cause, although contrast agents may aggravate the existing vasogenic edema in our case.

Our patient was a young woman and it was necessary to avoid irreversible brain changes that could cause long-term sequelae. Although our patient had clinico-radiological features of RPLS at the onset, the possibility of CNS vasculitis could not be completely eliminated. Therefore, she was treated with plasmapheresis, high-dose methylprednisolone, and cyclophosphamide in addition to antihypertensive and anti-convulsive therapy. Subsequently, these agents did not aggravate her condition, and resolution of the imaging abnormalities on follow-up MRI and regression of the clinical manifestations was achieved one month after the first onset. Therefore, it is not clear whether

Table 1 Clinical characteristics of 43 RPLS patients with connective tissue diseases

Authors	Diagnosis	Drugs	Age/ Sex	BP (mmHg)	Treatment	Neurological findings	Radiological findings
Hinchey	SLE	NS	30F	200/110	AH	Resolution in 2 weeks	Resolution
Hinchey	SLE	NS	39F	200/130	AH	Resolution in 2 weeks	Resolution
Pavlakis	SLE	PSL, IVCY	14M	220/130	AH, AC	Resolution in 12 days	Resolution in 16 days
Delanty	SLE	NS	28F	220/130	AH, AC	NS	NS
Arai	ANCA	PSL (60 mg/day), mPSL pulse, CY	57M	200/140	AH, increase of PSL(180 mg/day)	Partial resolution in 5 days	Partial resolution in 10 days
Schwartz	SLE	NS	38NA	150/90	NS	NS	NS
Casey	SLE	NS	13F	NS	NS	NS	Resolution in 20 weeks
Mukherjee	SLE	NS	22F	206/144	NS	Persistent deficits	Persistent changes
Mukherjee	SLE	NS	23F	174/103	NS	Resolution in 2 weeks	Resolution in 2 weeks
Primavera	SLE	PSL, IVCY	22F	200/130	AH, AC	Resolution in 4 days	Resolution
Primavera	SLE	CS, mPSLpulse, AZA, IVCY,	22F	170/110	AH, AC HD	Resolution in 10 days	Resolution
Primavera	SLE	PSL, CY	30F	210/125	AH, AC HD	Resolution in 2 weeks	Resolution
Primavera	WG	mPSLpulse, IVCY	23M	220/150	AH, AC	Resolution in 3 days	Resolution in 2 weeks
Kawano	ANCA	mPSLpulse	73F	200/116	AH, mPSLpulse	Resolution in 2 weeks	Resolution in 7 weeks
Kawano	ANCA	mPSLpulse	77F	186/123	AH, mPSLpulse	No resolution	Resolution in 4 weeks
Dy	SLE	PSL (40 mg/day), CY	31F	156/103	AH	NS	NS
Prasad	SLE	NS	NS	160/110	NS	Resolution in 4 weeks	Persistent changes
Patrick	SLE/SSc	CS, IVCY	39F	170/100	AH, switch to MMF	Rapid resolution	Partial resolution in 2 weeks
Mavragani	SLE	CS, IVCY, RTX	38F	210/120	AH, AC, mPSLpulse, IVCY, PP	Resolution in 1 day	Resolution in 6 weeks
Ohta	WG	PSL, mPSLpulse, CY	14F	180/92	AH, AC, HD	Gradual resolution	Resolution in 3 weeks
Shin	SLE	PSL(60 mg/day), mPSLpulse Vin, CyA	28F	130/80	AC, cessation of CyA	Resolution	Resolution in 4 weeks
Thaipisuttikul	SLE	PSL	20F	220/150	AH, AC, mPSLpulse	Resolution	Resolution in 2 weeks
Pasupuleti	SLE	PSL, CY, HQ	19F	180/110	AH, HD	Resolution in 2 days	Resolution in 2 days
Metzler	CSS	CS, IFNα	48F	NS	Switch to MTX	None	Persistent changes
Metzler	CSS	PSL, IFNα	59F	NS	Withdrawal of INFα, reduction of PSL	Resolution	Persistent changes
Magnano	SLE	PSL, mPSLpulse CY, MMF	24F	210/100	AH, AC	Resolution in 1 day	NS
Magnano	SLE	PSL, mPSLpulse, CY	32F	156/94	AH	Resolution	NS
Magnano	SLE	PSL, mPSLpulse, CY	37M	NS	AH, AC	Resolution in 1 week	Partial resolution in 2 weeks
Magnano	SLE	PSL, mPSLpulse, CY, IVCY, HQ	30F	158/110	AH, HD	Resolution in 2 days	Resolution in 5 weeks

Table 1 continued

Authors	Diagnosis	Drugs	Age/Sex	BP (mmHg)	Treatment	Neurological findings	Radiological findings
Magnano	SLE	NS	40F	180/100	AH, AC	NS	Resolution in 4 weeks
Min	SLE	mPSLpulse, IVCY	22F	200/110	AC, mPSLpulse, switched to MMF, PP	Resolution in 4 days	Resolution in 5 weeks
Kur	SLE	CS, CyA	29F	206/135	AH, AC, cessation of CyA	Resolution in 2 weeks	Resolution in 3 weeks
Kur	SLE	PSL	23F	140/90	AC, mPSLpulse, IVCY	Gradual resolution	Resolution in 4 weeks
Kur	SLE	CS, AZA, MMF	23F	194/126	AH, AC mPSLpulse, IVCY, PP	NS	Resolution in 4 weeks
Tajima	ANCA	PSL	76F	136/86	mPSLpulse	Resolution	Resolution in 3 weeks
Sasayama	HSP	None	16F	180/120	AH, mPSLpulse, HD, PP	Rapid resolution	Resolution in 2 weeks
Ishimori	SLE	Dexamethasone(120 mg×3/day), IVCY	47F	200/110	NS	Resolution in 1 day	Resolution in 1 week
Ishimori	SLE	mPSLpulse	20F	132/104	NS	Gradual resolution	Resolution in 1 week
Ishimori	SLE	PSL, CY, MMF	25M	176/105	AH	Resolution in 4 days	Resolution in 12 weeks
Ishimori	SLE	mPSL(60 mg × 4/day)	24F	169/108	AH	Resolution in 1 week	Resolution in 10 days
Zar	SLE	Hydrocortisone(100 mg×4/day), MMF	20F	190/110	AH, cessation of MMF	Resolution in 2 days	Resolution in 8 weeks
Nishio	WG	PSL, mPSLpulse, IVCY	15F	126/60	AH, AC, mPSLpulse, PP	Gradual resolution	Resolution in 2 weeks
Our case	TA	PSL (45 mg/day)	23F	182/72	AH, AC, mPSLpulse, IVCY, PP	Resolution in 3 weeks	Resolution in 4 weeks

NS not stated, *CS* corticosteroid, *CY* cyclophosphamide, *RTX* rituximab, *Vin* vincristine, *CyA* cyclosporine, *HQ* hydroxychloquine, *MMF* mycophenolate mofetil, *AZA* azathioprine, *AH* antihypertensive, *AC* anticonvulsant, *HD* hemodialysis, *PP* plasmapheresis

methylprednisolone, cyclophosphamide, and/or plasmapheresis themselves may directly cause RPLS, although it is believed they can. We would rather consider that these agents and treatments were necessary to control underlying vasculitis thought to be causes of endothelial injury with consequent vasogenic edema. However, the blood pressure and fluid volume should be strictly controlled in cases when these agents and treatments are used.

Regarding to the prognosis of RPLS, neurological findings and radiological findings are reversible and improve within two and four weeks, respectively, in most autoimmune cases, as shown in Table 1. However, hemorrhagic and/or ischemic changes remain as long-term sequelae in some cases. Different prognosis was not observed for RPLS associated with autoimmune diseases and RPLS with different causes, for example eclampsia, hypertensive encephalopathy, and immunosuppressive therapy [6, 11, 17].

In conclusion, we report, for the first time, a case of TA associated with RPLS. We think that, in our case, development of RPLS and disruption of the blood–brain barrier may be triggered by an increase of blood pressure, probably because of PSL, in addition to endothelial injury because of underlying vasculitis and/or anti-phospholipid antibodies. Finally, it is essential to achieve appropriate and prompt recognition of RPLS, because any delay in diagnosis and treatment may cause irreversible changes in the brain. We suggest that RPLS should be included in the differential diagnosis of acute neurological changes in connective tissue diseases, including TA.

Conflict of interest The authors have declared no conflicts of interest.

References

1. Hall S, Barr W, Lie JT, Stanson AW, Kazmier FJ, Hunder GG. A study of 32 North American patients. Med (Baltimore) 1985; 64:89–99.
2. Kerr GS, Hallahan CW, Giordano J, Leavitt RY, Fauci AS, Rottem M et al. Takayasu arteritis. Ann Intern Med. 1994; 120:919–29.

3. Johnston SL, Lock RJ, Gompels MM. Takayasu arteritis: a review. J Clin Pathol. 2002;55:481–6.
4. Mwipatayi BP, Jeffery PC, Beningfield SJ, Matley PJ, Naidoo NG, Kalla AA et al. Takayasu arteritis: clinical features and management: report of 272 cases. ANZ J Surg. 2005;75:110–7.
5. Ringleb PA, Strittmatter EI, Loewer M, Hartmann M, Fiebach JB, Lichy C et al. Cerebrovascular manifestations of Takayasu arteritis in Europe. Rheumatology. 2005;44:1012–5.
6. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494–500.
7. Pavlakis SG, Frank Y, Kalina P, Chandra M, Lu D. Occipital-parietal encephalopathy: a new name for an old syndrome. Pediatr Neurol. 1997;16:145–8.
8. Delanty N, Vaughan C, Frucht S, Stubgen P. Erythropoietin-associated hypertensive posterior leukoencephalopathy. Neurology. 1997;49:686–9.
9. Arai M, Shigeno K, Wada M. A reversible posterior leukoencephalopathy syndrome in a patient with classical polyarteritis nodosa. Clin Neurol. 1997;37:64–6.
10. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F. Diffusion-weighted MR imaging in hypertensive encephalopathy: Clues to pathogenesis. AJNR Am J Neuroradiol. 1998;19:859–62.
11. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: Utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. AJNR Am J Neuroradiol. 2000;21:1199–206.
12. Mukherjee P, McKinstry RC. Reversible posterior leukoencephalopathy syndrome: evaluation with diffusion-tensor MR imaging. Radiology. 2001;219:756–65.
13. Primavera A, Audenino D, Mavilio N, Cocito L. Reversible posterior leukoencephalopathy syndrome in systemic lupus and vasculitis. Ann Rheum Dis. 2001;60:534–7.
14. Kawano H, Kimura E, Ishizaki M, Nishida Y, Matsumoto N, Yamashita T et al. Reversible posterior leukoencephalopathy syndrome in two patients with microscopic polyarteritis nodosa. Clin Neurol. 2002;42:949–53.
15. Dy GK, Swaroop VS. 31-year-old woman with confusion and labile behavior. Mayo Clin Proc. 2002;77:557–60.
16. Yong PFK, Hamour SMA, Burns A. Reversible posterior leukoencephalopathy in a patient with systemic sclerosis/systemic lupus erythematosus overlap syndrome. Nephrol Dial Transplant. 2003;18:2660–2.
17. Prasad N, Gulati S, Gupta RK, Kumar R, Sharma K, Sharma RK. Is reversible posterior leukoencephalopathy with severe hypertension completely reversible in all patients? Pediatr Nephrol. 2003;18:1161–6.
18. Mavragani CP, Vlachoyiannopoulos PG, Kosmas N, Boletis T, Tzioufas AG, Voulgarelis M. A case of reversible posterior leukoencephalopathy syndrome after rituximab infusion. Rheumatology. 2004;43:1450–1.
19. Ohta T, Sakano T, Shiotsu M, Furue T, Ohtani H, Kinoshita Y et al. Reversible posterior leukoencephalopathy in a patient with Wegener granulomatosis. Pediatr Nephrol. 2004;19:442–4.
20. Shin KC, Choi HJ, Bae YD, Lee JC, Lee EB, Song YW. Reversible posterior leukoencephalopathy syndrome in systemic lupus erythematosus with thrombocytopenia treated with cyclosporine. J Clin Rheumatol. 2005;11:164–6.
21. Thaipisuttikul I, Phanthumchinda K. Recurrent reversible posterior leukoencephalopathy in a patient with systemic lupus erythematosus. J Neurol. 2005;252:230–1.
22. Pasupuleti DV, Miranda M, Vattipally V. Posterior reversible encephalopathy syndrome. Am Fam Phys. 2005;72:2430–2.
23. Metzler C, Lamprecht P, Hellmich B, Reuter M, Arlt AC, Gross WL. Leukoencepalopathy after treatment of Churg–Strauss syndrome with interferon α. Ann Rheum Dis. 2005;64:1242–3.
24. Magnano MD, Bush TM, Herrera I, Altman RD. Reversible posterior leukoencephalopathy in patients with systemic lupus erythematosus. Semin Arthritis Rheum. 2006;35:396–402.
25. Min L, Zwerling J, Ocava LC, Chen IA, Putterman C. Reversible posterior leukoencephalopathy syndrome in connective tissue diseases. Semin Arthritis Rheum. 2006;35:388–95.
26. Kur JK, Esdaile JM. Posterior reversible encephalopathy syndrome—an underrecognized manifestation of systemic lupus erythematosus. J Rheumatol 2006;33:2178–83.
27. Tajima Y, Matsumoto A. Reversible posterior leukoencephalopathy syndrome in p-ANCA-associated vasculitis. Intern Med 2006;45:1169–71.
28. Sasayama D, Shimojima Y, Gono T, Kaneko K, Matsuda M, Ikeda S. Henoch–Schonlein purpura nephritis complicated by reversible posterior leukoencephalopathy syndrome. Clin Rheumatol 2007;26:1761–3.
29. Ishimori ML, Pressman BD, Wallace DJ, Weisman MH. Posterior reversible encephalopathy syndrome: another manifestation of CNS SLE ? Lupus 2007;16:436–43.
30. Zar T, Samson W, Palmisano J. Reversible posterior leukoencephalopathy syndrome (RPLS) in a patient with systemic lupus erythemtosus (SLE) and lupus nephritis. Nephrol Dial Transplant. 2007;22:2400–1.
31. Mak A, Chan BP, Yeh IB, Ho RC, Boey ML, Feng PH et al. Neuropsychiatric lupus and Reversible posterior leukoencephalopathy syndrome: a challenging clinical dilemma. Rheumatology. 2008;47:256–62.
32. Nishio M, Yoshioka K, Yamagami K, Morikawa T, Konishi Y, Hayashi N et al. Reversible posterior leukoencephalopathy syndrome: a possible manifestation of Wegener's granulomatosis-mediated endothelial injury. Mod Rheumatol. 2008;18:309–14.
33. Belizna C, Lartigue A, Favre J, Gilbert D, Tron F, Lévesque H et al. Antiphospholipid antibodies induce vascular functional changes in mice: a mechanism of vascular lesions in antiphospholipid syndrome? Lupus. 2008;17:185–94.
34. Eichhorn J, Sima D, Thile B, Lindschau C, Turowski A, Schmidt H et al. Anti-endothelial cell antibodies in Takayasu arteritis. Circulation. 1996;94:2396–401.
35. Frontera JA, Spellman JP, Mohr JP. Contrast-induced neurotoxicity and selective cortical injury. Cerebrovasc Dis. 2007;24:148–51.