


# Primary cerebral immunoglobulin light chain amyloidoma in a patient with multiple sclerosis

Marissa J M Traets <sup>1</sup>, Krisna Chuwonpad,<sup>1</sup> Roos J Leguit,<sup>2</sup> Stephan T F M Frequin,<sup>3</sup> Monique C Minnema<sup>4</sup>

<sup>1</sup>Department of Internal Medicine, Sint Antonius Hospital, Nieuwegein, Netherlands

<sup>2</sup>Department of Pathology, University Medical Centre Utrecht, Utrecht, The Netherlands

<sup>3</sup>Department of Neurology, Sint Antonius Hospital, Nieuwegein, Netherlands

<sup>4</sup>Department of Hematology, University Medical Centre, Utrecht, Netherlands

## Correspondence to

Marissa J M Traets;  
M.J.M.Traets@umcutrecht.nl

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## SUMMARY

A man in his 60s, known with multiple sclerosis, presented with seizures and paresis of the left arm and leg. Brain imaging showed a white matter lesion, right parietal, which was progressive over the last 6 years and not typical for multiple sclerosis. Brain biopsy showed a B-cell infiltrate with IgA lambda monotypic plasma cell differentiation and amyloid deposits, typed as lambda immunoglobulin light chain (AL). Bone marrow biopsy and PET/CT ruled out a systemic lymphoma. Extended history taking, blood and urine testing (including cardiac biomarkers) identified no evidence of systemic amyloidosis-induced organ dysfunction. Primary cerebral AL amyloidoma is a very rare entity where optimal treatment is difficult to assess. The patient was treated with locally applied volumetric modulated arc radiotherapy, 24 Gy, divided in 12 fractions. Afterwards, the paresis of the left arm partially resolved, and the function of the left leg improved. Seizures did not occur anymore.

## BACKGROUND

Amyloidosis is a rare condition characterised by deposition of misfolded proteins in various tissues and organs in the body. Amyloid fibrils contain multiple proteins with a beta-pleated sheet configuration. The clinical presentation of amyloidosis is dependent of the type of the precursor protein from which the fibrils are constructed.<sup>1</sup> There are around 27 types of amyloidosis, which can be genetic or acquired.<sup>2</sup> Amyloidosis can be organ specific, such as Alzheimer's disease or systemic. The most common systemic types of amyloidosis are immunoglobulin light chain (AL), amyloid A and transthyretin amyloidosis. In AL amyloidosis, an acquired type, clonal plasma cells produce abnormal free light chain immunoglobulins causing amyloid deposits in organs. AL amyloidosis is in most cases a systemic disease with multiple organ involvement (eg, heart, kidneys, gastrointestinal and peripheral nervous system). AL amyloidosis is mostly caused by an underlying plasma cell proliferative disorder, and rarely by an underlying lymphoplasmacytic lymphoma or other non-Hodgkin's lymphoma, such as a marginal zone lymphoma or chronic lymphocytic leukaemia.<sup>1 3 4</sup>

Primary cerebral AL amyloidoma is a localisation of AL amyloidosis within the brain without signs of systemic disease. Optimal treatment is difficult to assess, as this is a very rare entity. Surgical resection and radiotherapy are described in the literature.<sup>5-7</sup> As in other forms of localised AL amyloidosis, the

role of systemic treatment can be questioned when there are no signs of a systemic clonal plasma cell proliferative disorder or non-Hodgkin's lymphoma.

## CASE PRESENTATION

A man in his 60s presented at the neurologist in 2012 with stumbling of the left leg since 2009. His medical history consisted of a thoracic transverse myelitis in 1980, with complete recovery. He was diagnosed with multiple sclerosis based on a transverse myelitis, multiple white matter lesions on MRI scan ([figure 1](#)), including cervical lesions, and oligoclonal bands in the cerebrospinal fluid (CSF). Initially from 2014, he was treated with glatiramer acetate. Two years later, there was a switch to dimethyl fumarate and fampridine. The neurological symptoms of the patient were progressive over the years with development of paresis of the left arm and leg and impaired coordination of the left hand. Annual follow-up MRI scans of the brain were performed and showed an increase of an irregularly shaped white matter lesion in the right parietal lobe ([figure 2](#)). Initially, the lesion was thought to be part of the multiple sclerosis. However, the lesion was not typical for multiple sclerosis as the lesion was increasing and there was persistent MRI enhancement despite treatment with immunotherapy. Also the localisation and size of the lesion were atypical as in multiple sclerosis multiple smaller lesions were expected, mostly located in the periventricular or juxtacortical white matter. Further, these lesions are often found in the corpus callosum, infratentorial areas and the spinal cord (especially the cervical segment).<sup>8</sup> In our patient, the lesion was located in the right parietal lobe. The patient had progressive complaints: paresis of the left leg was progressive, and the patient was partially wheelchair dependent since 2018. In 2020 and 2021, he developed focal and generalised seizures with Todd's paresis of the left hand. CSF showed no malignant cells and routine laboratory testing was unremarkable. [Figure 3](#) shows the timeline of above case presentation.

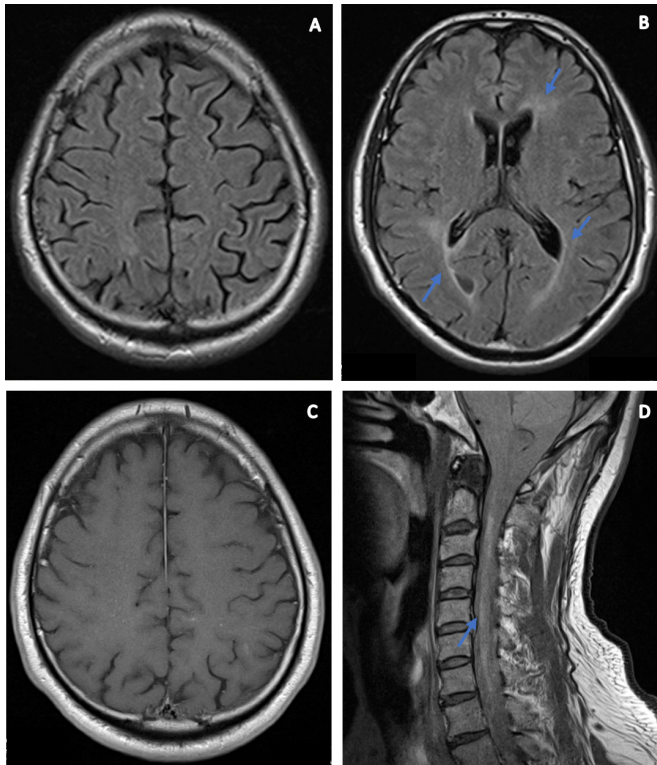
## DIFFERENTIAL DIAGNOSIS AND INVESTIGATIONS

Progressive cerebral lesions can be due to tumours in the central nervous system (CNS) such as amyloidoma, glioma, CNS lymphoma or cerebral metastases. Non-malignant diagnoses as infectious (syphilis, cryptococcosis, progressive multifocal leucoencephalopathy (caused by human polyoma-virus 2 [JC virus]), tuberculosis) or autoimmune



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**Figure 1** MRI at diagnosis of multiple sclerosis (2013) with periventricular and cervical white matter lesions and no enhancement after contrast (A, B) TIRM images. Arrows indicate the white matter lesions which are interpreted as multiple sclerosis lesions. (C) T1 with contrast. (D) TIRM cervical spine. Arrow indicate cervical lesions. Abbreviation: TIRM, turbo inversion recovery magnitude.

(sarcoidosis, systemic lupus erythematosus) causes should be investigated as well. Diagnostic tests for infectious and autoimmune diseases were all negative. In 2021, a brain biopsy was performed, which showed a perivascular infiltrate consisting of small B-cells and plasma cells, and the presence of amyloid localised in vessel walls and as depositions in the brain tissue (figure 4A,B). Immunohistochemistry demonstrated B cell (CD20 and PAX5 positive) and plasma cells (CD138 and MUM1

positive), which were monotypic for IgA and lambda light chains (figure 4C-F). CD3 showed sparse T-cells. B-cells were CD23, CD5 and cyclin D1 negative. Plasma cells were CD56 and cyclin D1 negative. Based on the IgA positivity, a plasma cell neoplasm was at first considered, but as CD20 and CD138 showed distinct cell populations (figure 4C,D) instead of plasma cells with aberrant CD20 expression, it proved to be a low-grade B-cell lymphoma with plasmacytic differentiation. The lymphoma was classified a marginal zone lymphoma as droplet digital PCR did not detect a MYD88 mutation, thereby making a lymphoplasmacytic lymphoma unlikely. Diagnosis of amyloidosis is based on histologically proven amyloid. Congo red stain is the golden standard to detect amyloid in tissues.<sup>9</sup> Subsequently, the amyloid turns red and shows a characteristic apple green birefringence when using polarised light. As typing of the amyloid is essential in the diagnostic trajectory, immunoelectron microscopy (figure 5) was performed, showing positivity for lambda light chains, consistent with AL amyloidosis.

Further investigations were performed to detect a systemic B-cell lymphoma and/or systemic AL amyloidosis. Systemic AL amyloidosis should be ruled out as this is important for both prognosis and treatment choice. Bone marrow biopsy did not show amyloid depositions and there were both morphological as immunophenotypic, no signs of a B-cell lymphoma or plasma cell dyscrasia. There was no presence of M-protein in the blood or urine, the free light chains (FLC) and FLC ratio were normal and the haemoglobin level, calcium level, kidney function and beta-2-microglobulin were all within the normal range. Also repeated investigation of the CSF with immunophenotyping did not demonstrate presence of clonal B-cells. Our patient had no signs of cardiac involvement with normal levels of cardiac biomarkers (troponin and N-terminal pro-B-type natriuretic peptide [NT-proBNP]) and a normal electrocardiogram. Urine analysis did not reveal proteinuria and liver enzymes were normal. The nuclear positron emission tomography and computed tomography (PET/CT) scan with fluorodeoxyglucose (FDG) showed no signs of lytic bone lesions or a systemic disease. In conclusion, bone marrow biopsy and the absence of lesions on PET/CT ruled out a systemic clonal B-cell disease or plasma cell dyscrasia. Extended history taking, blood and urine testing identified no evidence of systemic amyloidosis-induced organ dysfunction. The patient was diagnosed with primary cerebral AL amyloidoma.

## TREATMENT

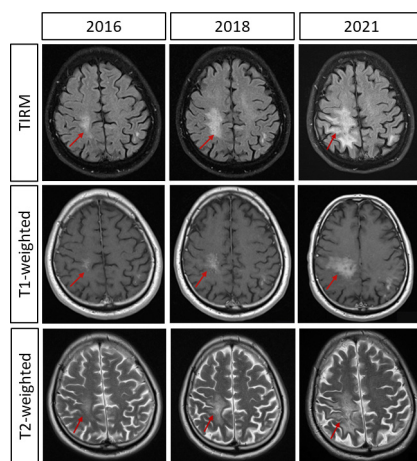
The patient was treated with locally applied volumetric modulated arc radiotherapy, 24Gy, divided in 12 fractions. Dexamethasone was prescribed during radiotherapy and tapered after completion of the radiation therapy.

## OUTCOME AND FOLLOW-UP

The patient had a good recovery with partial functionality of the left arm and improved function of the left leg. The seizures did no longer occur. MRI of the brain was performed 4 months after the last radiotherapy treatment and showed a slightly increased T2 effect in the right parietal lobe, which may be due to oedema because of the radiation. No new lesions were found.

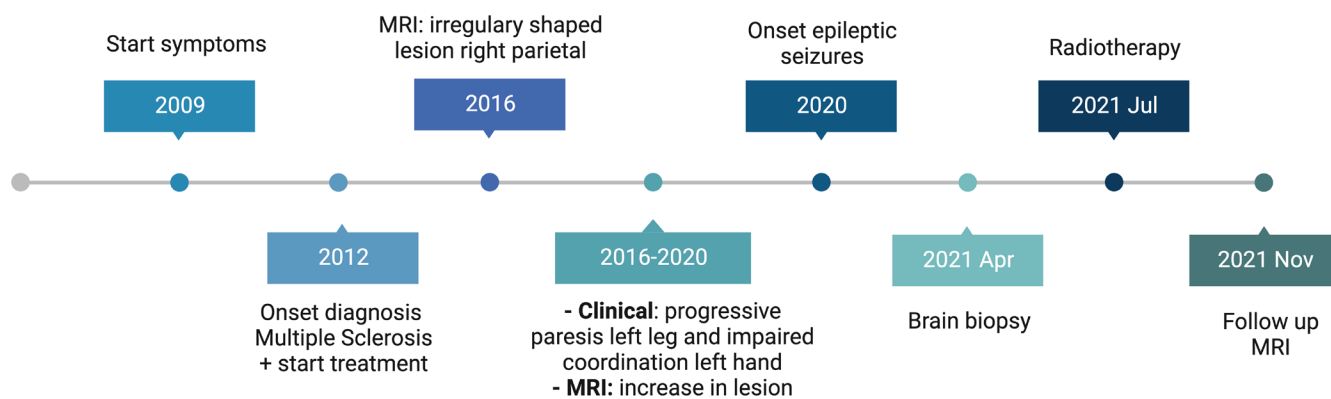
## DISCUSSION

In this case report a patient with a slowly progressive and symptomatic cerebral AL amyloidoma in the right hemisphere based on a local cerebral marginal zone lymphoma was described. To our knowledge, this is the first case described in the literature.



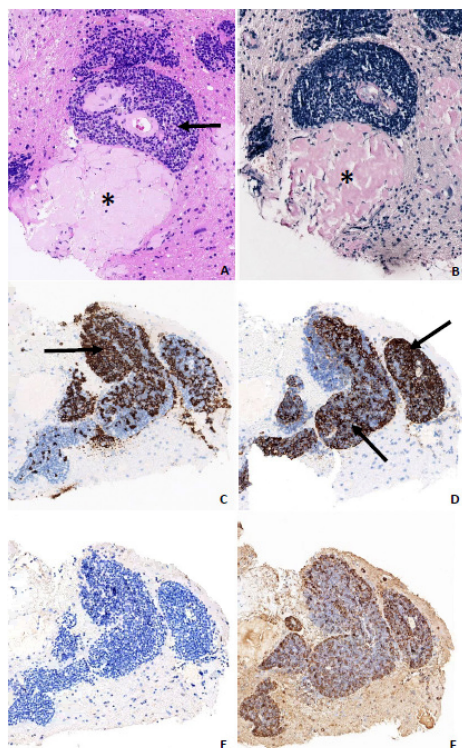
**Figure 2** MRI (TIRM, T1 and T2 with contrast) with progression of the lesion. Arrows indicate the white matter lesion in the right parietal lobe which is progressive over time. Abbreviation: TIRM, turbo inversion recovery magnitude.





**Figure 3** Timeline case presentation. Created with BioRender.com.

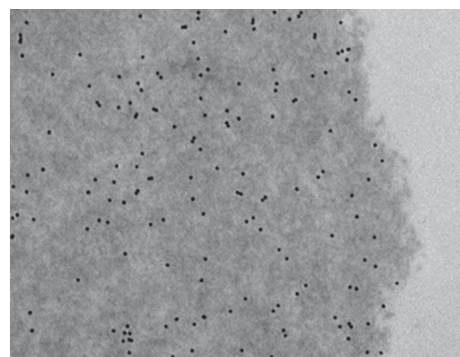
Diagnosis of AL amyloidoma and an IgA lambda positive marginal zone lymphoma was quite challenging and extraordinary. The majority of AL amyloidosis with IgA positivity is caused by a plasma cell proliferative disorder. However, as CD20 and



**Figure 4** Brain biopsy (right parietal) with AL amyloidosis and an IgA lambda positive marginal zone lymphoma. (A) H&E stain ( $\times 25$  magnification) showing amyloid (\*) and dens perivascular lymphoplasmacytic infiltrates (arrow). (B) The Congo red stain ( $\times 24$  magnification) shows positive red staining (\*) in the amyloid, which has green birefringence on polarisation (not shown). (C) The CD20 stain ( $\times 20$  magnification) stains the B-cells in the infiltrate (arrow). (D) The CD138 stain ( $\times 20$  magnification) shows plasmacytic differentiation in other parts of the perivascular infiltrates (arrows). (E) The kappa stain ( $\times 20$  magnification) is completely negative, whereas in (F) The lambda stain ( $\times 20$  magnification) shows positivity, consistent with a B-cell lymphoma with plasmacytic differentiation with lambda light chain restriction. As MYD88 mutation analysis was negative, this lymphoma is best classified as marginal zone lymphoma.

CD138 showed distinct cell populations instead of plasma cells with aberrant CD20 expression, it proved to be a low-grade B-cell lymphoma with plasmacytic differentiation. B-cell non-Hodgkin lymphomas with plasmacytic differentiation can also generate AL amyloid deposits. One case of a patient with a follicular lymphoma with plasmacytic differentiation was associated with AL amyloidosis located in the ileum.<sup>10</sup> In some articles, a lymphoid neoplasm was mentioned as underlying cause of the cerebral amyloidoma.<sup>6,11-16</sup> However, to diagnose, an underlying lymphoid neoplasm the infiltrate must have been typed. Only a few articles performed immunohistochemistry on the infiltrate with demonstration of B-cell clonality.<sup>14-16</sup> The article of Heß *et al* describes seven cases with cerebral amyloidoma and B-cell clonality. Follicular lymphoma, mantle cell lymphoma or chronic lymphocytic leukaemia were unlikely based on the immunohistochemistry results (negative for CD10, cyclin D1 and CD5). However, they have not performed MYD88, so the infiltrate could be either a marginal zone lymphoma or lymphoplasmacytic lymphoma.<sup>15</sup>

Surgical resection and radiotherapy are described in the literature as treatment options for cerebral AL amyloidoma.<sup>5-7</sup> One study collected data of 27 patients with cerebral amyloidoma of whom 5 patients were diagnosed by autopsy material.<sup>6</sup> Fourteen patients had the same subtype classification as the patient in this case: AL amyloidoma (lambda). Two patients were both lambda and kappa positive. Besides, the patients diagnosed by autopsy, all patients were treated with (stereotactic) biopsy or surgery for resection of the lesion. The authors recommend complete tumour



**Figure 5** Immunoelectron microscopy for lambda light chains. Immunoelectron microscopy shows amyloid fibrils reactive with the gold labelled antibody against lambda light chains.

resection for patients with localised cerebral AL amyloidoma as recurrence after complete resection is unlikely.<sup>6</sup> Surgery of the brain is difficult if the lesion involves important brain functions like the speech area, the brain stem or the visual cortex. One case report describes a patient with AL amyloidoma where the lesion was not feasible to resection.<sup>7</sup> A recently published case report describes two patients with primary CNS marginal zone lymphoma successfully treated with radiotherapy.<sup>17</sup> Another case of a patient with dural marginal zone lymphoma and amyloid depositions responded well to radiotherapy too.<sup>18</sup> Our patient was also successfully treated with fractionated radiation therapy and dexamethasone without progression during follow-up at 18 months after therapy. The aim of radiotherapy is elimination of the underlying neoplastic B-cell or plasma cell neoplasm which is responsible for the amyloid deposition. Without treatment, cerebral AL amyloidoma has the potency of further progression and worsening neurological symptoms. In this case, primary cerebral AL amyloidoma was successfully treated with locally applied radiotherapy as the patient already experienced improvement of the left arm after four fractions of radiotherapy.

Primary cerebral AL amyloidoma is a rare entity with only a few reported cases. Also involvement of the CNS by marginal zone lymphoma is extremely rare. Primary cerebral marginal zone lymphoma has most commonly been described in association with the dura mater.<sup>17 19–22</sup> The neurological symptoms of our patient (paresis left leg, impaired coordination left hand and seizures) were related to his white matter lesion in the right parietal lobe. Both symptoms and the size of the lesion were progressive over the years. The symptoms of the patient in this case were initially attributed to his multiple sclerosis. Multiple sclerosis is a chronic inflammatory demyelinating disease of the CNS where multiple lesions are located in the brain and spinal cord.<sup>23</sup> Case series showed involvement of the white matter of the brain in cerebral amyloidoma, and multifocal locations of cerebral AL amyloidoma have been described.<sup>6 24</sup> The time between progression of neurological symptoms and diagnosis of cerebral AL amyloidoma was approximately 3–5 years in this case. Because the symptoms were initially attributed to the multiple sclerosis there was a delay in the diagnosis. After multiple discussions between neurologists and radiologists, there was decided to perform the brain biopsy which is an invasive procedure with risks involved. In this case, our patient had two diagnoses, first multiple sclerosis and later primary cerebral AL amyloidoma. Factors contributing to the development of primary cerebral AL amyloidoma and marginal zone lymphoma remain to be elucidated. Therapy-induced amyloidoma was ruled out, because the onset of the lesion was before start of immunomodulatory therapy (glatiramer acetate and dimethyl fumarate). Patients with a specific chronic infectious or autoimmune disease are more prone to develop marginal zone lymphoma. For instance, *Helicobacter pylori* infection is associated with mucosa-associated lymphoid tissue lymphoma in the gastrointestinal mucosa, and Sjogren syndrome is linked to extranodal marginal zone lymphoma of the salivary gland.<sup>25 26</sup> One case series described 20 patients diagnosed with both marginal zone lymphoma and amyloid depositions, of whom 6 had a history of autoimmune disorders (such as Sjogren disease or lupus or a lupus-like syndrome; no multiple sclerosis).<sup>25</sup> Another case report describes a patient with a cerebral amyloidoma accompanied by Sjogren disease and another amyloidoma in the hip.<sup>27</sup> The relationship between localised amyloidosis and autoimmune disorders is yet unclear.

An extensive study including 293 patients with localised light chain amyloidosis showed a concomitant autoimmune disorder in 21% of the patients.<sup>28</sup> This was most frequent in patients with skin localised amyloidosis. None of the patients (N=7) with brain amyloidosis in this study had a concomitant autoimmune disorder. However, these data indicate that there might be an association between localised amyloidosis and autoimmune disorders. Another study showed a few patients with initially clinically definite multiple sclerosis who were diagnosed with primary cerebral AL amyloidoma during neuropathological examination.<sup>10 29</sup> Furthermore, in rare multiple sclerosis cases, amyloid deposition (typed as immunoglobulin light chain, lambda) was found in demyelinated plaques of the patients.<sup>30 31</sup> This strengthens the importance to remain alert for another diagnosis in patients with an atypical course of multiple sclerosis. Our case report and the literature support the concept that diagnosis of primary cerebral AL amyloidoma is challenging, especially in case of another neurodegenerative disease, such as multiple sclerosis. Further research is necessary to unravel the role of multiple sclerosis on the development of primary cerebral AL amyloidoma based on a local cerebral marginal zone lymphoma.

### Patient's perspective

The patient has written his perspective on the diagnosis and treatment. The own words of the patient are translated by the authors.

'I was diagnosed with multiple sclerosis and my symptoms kept getting worse. I thought it was due to the multiple sclerosis. After examinations, the lesion in my brain (which was always there) turned out to be growing. I was also retaining more fluid in my brain. I had function failure of the left side of my body, memory problems, and coming up with words became also more difficult. Furthermore, lying down made speaking more difficult. The brain biopsy went well. I received 12x radiotherapy, which went well and my left arm also improved. After a few weeks, this became less again and I needed a few months to recover from the radiotherapy. At the moment I feel fairly stable, also in terms of fatigue. I do regularly suffer from nerve pain in the left leg, which is probably due to the multiple sclerosis.'

### Learning points

- ▶ Clinical symptoms of primary cerebral immunoglobulin light chain (AL) amyloidoma can be similar to those of multiple sclerosis.
- ▶ Primary cerebral AL amyloidoma is a rare entity which can be successfully treated with locally applied radiotherapy.
- ▶ Primary cerebral AL amyloidoma can only be diagnosed by MRI investigation of the brain and histopathological examination.
- ▶ Multidisciplinary teamwork between neurologists, radiologists, haematologists and pathologists is necessary to reduce the delay between onset symptoms and diagnosis, and to decide the most effective treatment.

**Twitter** Stephan T F M Frequin @STFM Frequin

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MJMT, KC, RJL, STFMF and MCM. The following authors gave final approval of the manuscript: MJMT, KC, RJL, STFMF and MCM.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

#### ORCID iD

Marissa J M Traets <http://orcid.org/0000-0003-0821-7995>

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