A seventy year old man with intractable vomiting, parkinsonism, memory loss and ptosis

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Key Words

Multiple enhancing mass lesions, primary CNS lymphoma, structural parkinsonism.

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Case Presentation: Dr Suresh Kumar

A seventy year old businessman developed diarrhea which lasted for seven days and subsided with oral rehydration solution (ORS), antiemetics, and antibiotics. Two days after the onset of diarrhea, he developed nonprojectile vomiting. Though diarrhea subsided after seven days, vomiting persisted.

One week later, he developed unsteadiness and tendency to fall. Vomiting was not getting controlled with antiemetics. A Magnetic Resonance Imaging (MRI) brain was done on the 24th day of the illness. A gastroenterology work-up was inconclusive.

He was admitted into this hospital 53 days after the onset of his symptoms. He complained of intractable vomiting, unsteadiness, bilateral tremor, slowness of body movements, and mild dysarthria. He had prostatism detected three months ago. There was no history of weight loss, tuberculosis, fever, arthritis, diabetes mellitus, hypertension, dyslipidemia, liver disease, addictions, or extramarital sexual exposure.

On examination, his pulse was 80 per minute, blood pressure 120/80 and respiratory rate 16perminute. There was no pallor, icterus, cyanosis, clubbing, pedal edema or lymphadenopathy. Peripheral pulses, skin, cardiovascular, respiratory and skeletal systems, abdomen, thyroid and testis were normal.

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His Minimental state examination (MMSE) score was 24 upon 30. He missed two points on recall, two points in orientation to time, and two points in orientation to place. He had hypophonic dysarthria and facial hypomimia. There was no aphasia, apraxia, or agnosia. Rest of the cranial nerves were unremarkable at admission.

There was cogwheel rigidity and rest tremor of the left upper limb that disappeared with action and re-emerged on maintaining a posture. Motor power, deep tendon reflexes, and superficial reflexes were normal. The plantars were bilaterally flexor. Sensory and cerebellar systems were normal. He had a slow festinant gait and difficulty while turning. Tandem walking was difficult. His hemogram, urine examination, erythrocyte sedimentation rate (ESR), serum electrolytes, Calcium, Phosphorus, liver and renal function tests, and C-reactive protein (CRP) were normal except for hyponatremia. Prostate-specific antigen (PSA), human immunodeficiency virus (HIV) serology, and hepatitis B surface antigen (HBsAg) were negative. Electroencephalography (EEG) showed mild background slowing. He had hyponatremia, which rapidly worsened and was corrected with saline.

On the ninth hospital day, he developed bilateral ptosis and gaze palsy to left without any diplopia. An MRI was done on the ninth hospital day. Subsequently he deteriorated.

An investigation was done followed by a diagnostic procedure.

Discussion: Dr Jacob George

The discussion that follows is divided into four parts:

1. Localization of the problem
2. Clinical diagnostic possibilities
3. Imaging findings
4. Final diagnostic considerations.

Localization of the problem

The symptoms in this patient, in the order in which they appeared are:

a. Vomiting
b. Ataxia and difficulty to turn,
c. Parkinsonian tremor, hypophonic dysarthria, and bradykinesia,
d. Memory loss,
e. Bilateral ptosis, and
f. Gaze palsy to left.

Since the first four symptoms do not have good localizing value, it is better to see the localization of symptoms e and f mentioned above and to see whether the other symptoms can be explained by a lesion in that vicinity.

Ptosis

The ptosis could be due to bilateral Horner's syndrome or nuclear third nerve palsy. The ptosis of Horner's syndrome is mild, variable, and usually associated with miosis. An isolated lesion of the caudal central subnucleus of the third nerve may also produce bilateral ptosis without affecting other extraocular muscles or the pupils, since both levator palpebrae superioris muscles are innervated by a single midline subnucleus.[1] In this patient, a caudal central subnucleus lesion is more likely.

Gaze palsy to left

The gaze palsy to left indicates a lesion of the left paramedian pontine reticular formation (PPRF). Though a right frontal lesion is also possible, since the ptosis localizes to the brainstem, a PPRF lesion is more likely. Since the patient did not have diplopia, an infranuclear lesion is unlikely.

Vomiting

Since vomiting was the first symptom and persisted throughout the illness, it is likely that the vomiting is due to the disease itself. Vomiting could be due to raised intracranial pressure (ICP) or due to a lesion in the posterior fossa. Since the patient does not have headache or papilledema, raised ICP looks unlikely. Though there is no well-localized vomiting center, the essential neuronal circuitry for emesis is in the medulla. The chemoreceptor trigger zone (area postrema) is located in the floor of the caudal end of the fourth ventricle. Considering the localization of the other symptoms, the vomiting in this patient could be due to a posterior fossa lesion with compression on the area postrema.

Parkinsonian symptoms

The patient had tremor and cogwheel rigidity of left upper limb, facial hypomimia and slow hesitant gait.A structural or secondary cause of parkinsonism rather than idiopathic parkinsonism should be considered in this patient because of the many associated symptoms and signs. Since the patient has history of antiemetic use, drug-induced parkinsonism may be considered. But an attempt should be made to explain all symptoms in a given patient by the same lesion. Since the patient has a progressive illness, it would be worthwhile to consider whether mass lesions can produce parkinsonism. Surprisingly, tumors of the striatum presenting with parkinsonian features are rare.[2] Tumors in other brain areas (frontal, parietal, temporal, thalamus, midbrain and third ventricle), subdural hematoma, midbrain tuberculoma, vascular malformations and posterior fossa cysts are known to produce parkinsonism.[2] Most common feature of structural parkinsonism is gait disturbance. In this patient, it is possible that the parkinsonism is due to a structural lesion involving the midbrain.

Gait unsteadiness

The gait unsteadiness has no localizing value and could be due to parkinsonism, cerebellar dysfunction, metabolic disturbances producing myoclonus or postural imbalance due to subcortical dysfunction.

Memory loss

In MMSE, patient made mistakes in recall and orientation to place and time. This could be because of impaired attention and vigilance due to a subcortical lesion or it could be a deficit in episodic memory due to a hippocampal or diencephalic lesion.

To summarize, the patient has a lesion involving the caudal central subnucleus and PPRF, both of which are structures adjoining the aqueduct and the fourth ventricle in the brain stem. The area postrema also is a structure adjoining the cerebrospinal fluid (CSF) in the caudal fourth ventricle. The lesion hence seems to be adjoining the CSF pathways and extending through the medulla, pons and midbrain. The parkinsonism could be due to midbrain involvement, and the memory loss could be due to diencephalic involvement adjoining the third ventricle.

Clinical diagnostic possibilities

The diagnostic possibilities in a 70-year-old man with a progressive brainstem and subcortical lesion are neoplastic mass lesions (metastasis, glioblastoma, primary central nervous system lymphoma (PCNSL) etc) and infectious mass lesions (multiple tuberculomas, cysticercosis, brain abscesses, toxoplasmosis, fungal lesions such as histoplasmosis and aspergillus abscesses). The other differential diagnosis that may be considered include osmotic demyelination, Wernicke's encephalopathy, CNS vasculitis especially Neuro — Behcet's disease, demyelination (acute disseminated encephalomyelitis), and paraneoplastic brainstem encephalitis (anti Ma-2 encephalitis).

Imaging findings (Dr Gourisankar PT)

Two MRIs are provided, the first one done three weeks into the illness and the second done nine weeks into the illness.

The first MRI shows a single small lesion in the dorsomedial medulla corresponding to the region of area postrema visible only on T_2 and fluid-attenuated inversion recovery (FLAIR) images [Figure 1a]. This was when the patient was having only vomiting. Contrast images are not available. Though it is not possible to conclude what the lesion is, the lesion corresponds well with the symptoms of the patient and tells where the disease started.

The second MRI shows three lesions: One in the caudal fourth ventricle, one at the mesodiencephalic junction, and one involving the fornix [Figure 1b]. The imaging characteristics of all the three lesions are similar. The lesions are iso to slightly hypointense to the gray matter on T_1- and T_2-weighted images as well as in FLAIR images [Figure 2a-c] and show uniform intense

contrast enhancement [Figure 2d]. The lesions have invoked moderate amount of edema and on T_2 and FLAIR sequences [Figure 3a and b], the hyperintense edema contrasts sharply with the iso- to hypointense lesions. The lesions are hyperintense on diffusion-weighted images (DWI) and show restricted diffusion on apparent diffusion coefficient (ADC) maps [Figure 3c and d].

Retrospectively, the hyperintense lesion at the floor of the fourth ventricle in the first MRI is probably edema surrounding a minute lesion.

Final diagnostic considerations (Dr Jacob George)

The imaging findings correlate well with the clinical findings: Vomiting is explained by the lesion in the medulla, the gait unsteadiness and parkinsonism by the lesion in the mesodiencephalon, memory loss by the lesion in the fornix, ptosis and gaze palsy by the edema in the midbrain and dorsal pons, respectively.

The most striking feature on MRI is that the lesions are iso to hypointense on T_2-weighted and FLAIR sequences and show restricted diffusion on ADC maps. This indicates a densely packed and highly cellular tumor with high nuclear-cytoplasmic ratio. These signal intensity characteristics make conditions like demyelination, osmotic demyelination, Wernicke's encephalopathy, Neuro-Behcet disease and anti-Ma2 encephalitis unlikely. These conditions are all hyperintense on T_2-weighted and FLAIR sequence and do not produce such intense and uniform contrast enhancement. Though the lesions are diffusion restricting, the solid nature of the lesions on T_2-weighted images and the pattern of uniform rather than ring enhancement argue against multiple brain abscesses.

The final diagnostic considerations are based on the following five parameters viz. age of the patient, clinical features, lesion location on MRI, signal intensity characteristics, and contrast enhancement pattern.

Five diagnostic possibilities are discussed here, starting from the least possible through to the most possible diagnosis.

Diagnosis 5: Glioblastoma multiforme (GBM)

GBM may be considered because of the age of the patient, the progressive neurological deficit and the presence of mass lesions on imaging. The peak prevalence of GBM is between 65 and 75 years of age.[3] GBM may be multifocal.[4] Because GBM may disseminate by CSF pathways, lesions may occur adjoining the CSF as in this patient.

But there are many odd points for GBM. As was seen in the first MRI, the disease started in the medulla. This is unusual for GBM. Only less than 1% of GBM start in the posterior fossa.[5] The typical GBM has a heterogeneous appearance on T_1- and T_2-weighted images due to necrosis and hemorrhage with irregular enhancement,[4] very unlike in our patient who has homogeneously enhancing multiple lesions without necrosis or hemorrhage. The gradient echo (GRE) sequences also do not show any area of hemorrhage, whereas susceptibility artifacts are common in GBM due to blood products.[6] GBM does not typically produce diffusion restriction[6] unlike the lesions in this patient which show restricted diffusion.

Diagnosis 4: Multiple tuberculomas

Multiple tuberculomas may be considered as a differential diagnosis because tuberculomas may occur at any age; the clinical and radiologic findings suggest multiple enhancing mass lesions.

The MRI appearance of tuberculoma depends on the structure of the tuberculoma. Based on the structure tuberculomas may be of three types: Non-caseous granuloma, caseous granuloma with solid centre, and caseous granuloma with liquid centre.[7] The computed tomography (CT) and MRI features of tuberculoma are summarized in Table 1.

Though the lesions in our patient show homogeneous enhancement, the iso- to hypointense nature of the lesions on T_2-weighted images argues against non-caseous granuloma.

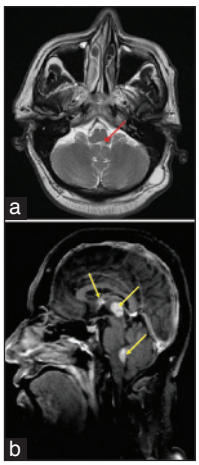

Figure 1: Magnetic resonance imaging (MRI) done 3 weeks into the illness showing subtle T_2 hyperintense lesion (red arrow) at the caudal end of the floor of the fourth ventricle (a) and MRI done 9 weeks into the illness showing three lesions, one at the floor of fourth ventricle, second at the mesodiencephalic junction, and third at the fornix (b). Yellow arrows point to the lesions

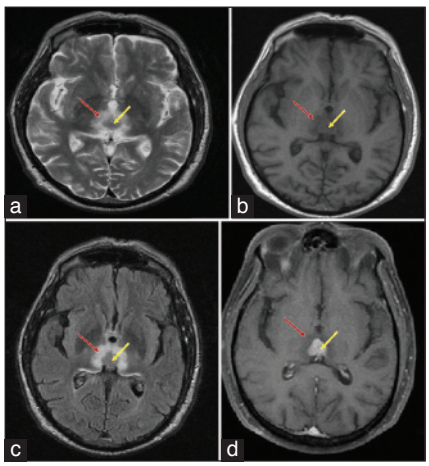

Figure 2: Signal intensity characteristics of the lesion at the mesodiencephalic junction: Isointense on T_2-weighted imaging (T_2WI) (a); isointense on T_1-weighted imaging (T_1WI) (b); isointense on T_2fluid-attenuated inversion recovery (FLAIR) (c); and homogeneous solid enhancement (d). Yellow arrow points to the lesion and red arrow to the edema surrounding

The enhancement pattern of the lesions in our patient does not fit with either of the two types of caseous granuloma.

Diagnosis 3: Multiple metastases

Multiple metastases need consideration because of the age of the patient and the presence of multiple enhancing mass lesions with edema on imaging.

Though the most common location for metastatic disease of the brain is the corticomedullary junction, any part of the brain may be involved.[8] Approximately 80% of metastases occur supratentorially, 15% in the cerebellum and 5% in the brainstem.[9] Gastrointestinal, prostate, and uterine cancers disproportionately metastasize to the posterior fossa.[9] The MRI appearance of metastases depends on whether the lesion is solid, necrotic, hemorrhagic, cystic, or rarely calcific. Metastases are usually hypo- or isointense on T_1-weighted images and hyperintense on T_2-weighted images, unless there is hemorrhage.[8] Contrast enhancement is usually present and the patterns of enhancement include homogeneous enhancement, ring enhancement, non-homogeneous enhancement, and no contrast enhancement.[8] Individual lesion contrast and the sensitivity of detecting newer lesions can be increased by administering triple dose gadolinium contrast.[10] On DWI, the signal intensity of non-necrotic components of metastases is variable. The necrotic components show marked signal suppression on DWI and increased ADC values. Increased signal intensity on DWI and a low ADC values are unusual but possible.[11]

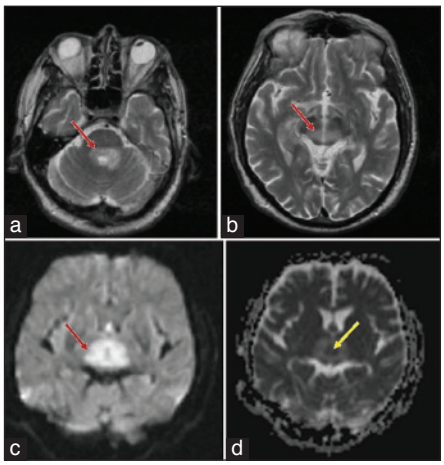

Figure 3: T_2WI showing edema in the dorsal pons (a) and midbrain (b). Lesion is hyperintense on diffusion-weighted imaging (DWI) (c) and shows restricted diffusion in apparent diffusion coefficient (ADC) maps (d). Yellow arrow points to the lesion and red arrow to the edema surrounding

Though a diagnosis of multiple metastases is possible, the following points are odd: The location is not classic; the isointense nature of lesions on T_2-weighted images; absence of necrosis or hemorrhage, though these are not mandatory; all lesions appear homogeneous, though this could be possible; and the lesions are diffusion restricting, which is rare in metastases.

Diagnosis 2: CNS toxoplasmosis

Toxoplasmosis should be considered in the differential diagnosis because of the presence of multiple enhancing lesions in the periventricular region.

On MRI, the lesions are usually hypointense on T_1-weighted images and typically hypo- to isointense on T_2-weighted images surrounded by high signal intensity vasogenic edema. Post contrast MRIshows multiple nodular lesions or ring enhancing lesions. Occasionally, a small eccentric nodule rests alongside an enhancing ring: The "target sign". This finding is highly suggestive of toxoplasmosis; however, it is relatively insensitive and is seen in less than 30% of cases.[12] Hemorrhage may be seen occasionally, a finding that can help differentiate toxoplasmosis from lymphoma, which typically does not hemorrhage before treatment.[13]

Points favoring toxoplasmosis in this patient are the isointense nature of the lesions on T_1- and T_2-weighted images and the nodular enhancement. The odd points are that the disease started in the medulla, that all lesions are adjoining the CSF and the lack of any immunocompromising condition. Though nodular enhancement may occur, ring enhancement is the classic pattern in toxoplasmosis. This also is a relative odd point.

Diagnosis 1: Primary CNS lymphoma

Primary CNS lymphoma (PCNSL) is a rare B-cell variant of non-Hodgkin lymphoma. Although PCNSL accounts for less than 7% of brain tumors, its incidence is increasing, particularly in immunocompetent individuals. The average age of onset is 58 years for immunocompetent patients.[14]

On CT scans, PCNSL lesions are typically hyperdense and virtually all lesions enhance with contrast.[15] On MRI, PCNSL lesions are well-demarcated and are typically isointense to slightly hypointense on T_1- and T_2-weighted images and produce only minimal edema. The appearance on T_2-weighted images likely reflects the high nuclear-cytoplasmic ratio in these densely packed and highly cellular tumors.[15] T_2-hypointensity when seen helps to differentiate PCNSL from gliomas, which are usually hyperintense on T_2-weighted images. Enhancement pattern of PCNSL lesions tend to be solid and homogenous in

Table 1: CT and MRI features of tuberculoma

Structure	Nonenhanced CT	Enhanced CT	MRI	MRI with contrast
Non-caseous	Hypo- or isodense	Nodular enhancement	T_1-hypointense T_2-hyperintense	Homogeneous enhancement
Caseous with solid center	Hypo- or isodense	Ringenhancement	T_1-hypo-isointense T_2-hypo-isointense	Ringenhancement
Caseous with liquid center	Hypo- or isodense	Ringenhancement	T_1-hypointense T_2-hyperintense	Ringenhancement

CT = Computed tomography, MRI = Magnetic resonance imaging

immunocompetent individuals.[15] The most common location of PCNSL is the cerebral hemispheres followed by the deep gray matter nuclei and the corpus callosum.[16] Lesions may also occur in the brainstem, cerebellum and spinal cord, cranial nerves, cavernous sinuses, cranial nerves pineal and pituitary glands.[17] Multiple lesions occur in 11-47% of patients with PCNSL, their prevalence being higher in immunocompromised patients.[15] One of the characteristic features of PCNSL is its tendency to abut the ependyma or the meninges or both. The finding of periventricular enhancement in acquired immunodeficiency syndrome (AIDS) patients is quite suggestive of PCNSL,[15] though this appearance may be mimicked by cytomegalovirus (CMV) ependymitis. Enhancement of the Virchow-Robin spaces, although not constant, is also a highly specific feature of PCNSL.[16] Pre-therapy PCNSL does not show calcification or hemorrhage. Peritumoral edema is typically mild or absent in immunocompetent patients.[17]

In immunocompromised patients, multiple lesions, necrosis, ring enhancement, and surrounding edema are more common than in immunocompetent patients.[16]

The high cellularity of lymphoma decreases the extracellular space and the random motion of water molecules. This causes hyperintensity on diffusion-weighted imaging and hypointensity on ADC maps. [17]

On proton MR spectroscopy, lymphoma typically shows decreased NAA, decreased creatine, increased choline, and a lipid, lactate peak. A similar spectral pattern may be seen with metastases and high grade gliomas. However, peritumoral choline is elevated for glioblastomas because of infiltrating tumor cells; whereas, the peritumoral choline is decreased for lymphomas and metastases, since these tumors are well-circumscribed.[17-19]

In patients with AIDS, in comparison with toxoplasmosis, lymphoma displays greater thallium uptake on SPECT images.[20]

The MRI features of GBM, metastasis, toxoplasmosis, and PCNSL are summarized in Table 2.

Overall, the age, clinical presentation, location of the lesions adjoining the CSF pathways, intensity characteristics of the lesions, uniform enhancement of the lesions, and absence of hemorrhage and necrosis are all typical for non-AIDS lymphoma. The only relative odd point for lymphoma is the edema that seems more than expected with lymphoma. But overall, primary CNS lymphoma is the first possibility.

The investigation that was done was probably a CSF examination for malignant cells and the diagnostic procedure a biopsy of the lesion.

Pathology findings (Dr Bindhu MR)

The patient underwent a CSF study followed by a biopsy from a new cerebral lesion which had appeared in a later MRI.

Microscopy of CSF cytospin preparation showed moderately cellular smear showing monotonous population of large round cells [Figure 4a].

The hematoxylin and eosin (H and E) stained sections showed a neoplasm comprising of large round cells arranged in sheets. These cells showed high N/C ratio open chromatin and prominent nucleoli and brisk mitosis [Figure 4b].

The immunohistochemistry done on these sections showed that the cells were positive for leukocyte common antigen (LCA)

Table 2: MRI features of GBM, metastasis, toxoplasmosis, and PCNSL

Sequence/ feature	GBM	Metastasis	Toxoplasmosis	PCNSLin immunocompetent
T_1WI	Heterogeneous	Iso- to hypointense. Melanoma and hemorrhagic metastases are hyperintense	Hypointense	Iso to hypointense
T_2WI	Heterogeneous	Variable, usually hyperintense	Hypo-, iso-, or hyperintense	Iso to hypointense
T_2 FLAIR	Heterogeneous	Hyperintense	Same as in T_2	Iso to hypointense
Contrast enhancement	Thick, irregular rim and central necrosis	Uniform, punctuate or rim	Nodular or ring enhancing. Target sign	Homogenous solid enhancement
Edema	+++	++++	++	+
GRE	Blood products present	Blooms if hemorrhage present	Hemorrhage may be present. Helps to differentiate from PCNSL	No blood products
DWI and ADC	No restriction	No restriction	Hyperintense rim on DWI. No restriction on ADC maps	Hyperintense on DWI. Restricted diffusion on ADC
MRS	Elevated choline, lipid lactate peak, decreased myoinositol; peritumoral choline elevated	Elevated choline peak without elevation in peritumoral area. Lipid/lactate if necrosis present.	Lipid/lactate peak. No choline peak.	Elevated choline. Peritumoral choline not elevated.
PWI (rCBV)	Increased	Increased	Not increased	Increased, but not to the extent of GBM and metastases
FDG PET	Hypermetabolic	Hypermetabolic	Hypometabolic	Hypermetabolic

GBM = Glioblastoma multiforme, PCNSL = Primary central nervous system lymphoma, T_1WI = T_1-weighted imaging, T_2WI = T_2-weighted imaging, FLAIR = Fluid-attenuated inversion recovery, GRE = Gradient echo, DWI = Diffusionweighted imaging, ADC = Apparent diffusion coefficient, MRS = Magnetic resonance spectroscopy, PWI = Perfusion-weighted imaging, rCBV = Relative cerebral blood volume, FDG PET = Fluorodeoxy glucose positron emission tomography

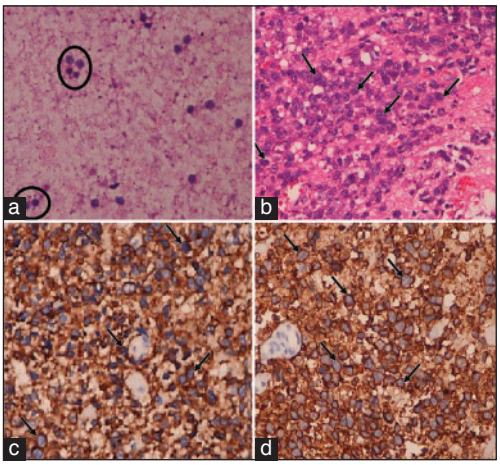

Figure 4: Cerebrospinal fluid (CSF) showing large round cells (encircled in a); hematoxylin and eosin (H and E) section showing large round cells with high nuclear cytoplasmic ratio (black arrows in b); immunohistochemistry showing positivity for leucocyte common antigen (LCA) (black arrows in c); cluster of differentiation-20 positivity (black arrows in d)

[Figure 4c] and cluster of differentiation (CD) 20 (B cell marker) [Figure 4d], but negative for cytokeratin (epithelial marker), GFAP,CD3,CD10,CD5,CYCLIND, and Bcl2; thus confirming the diagnosis of diffuse large B cell lymphoma.

Final diagnosis

Primary CNS lymphoma, diffuse large B cell type.

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