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Darpan Gupta,
Chinmaya Srivastava,
Sudhanshu Agrawal

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Mass lesion in cases of cerebral arteriovenous malformations post gamma knife radiosurgery or embolization. Pathophysiology and management algorithm

Darpan Gupta[1], Chinmaya Srivastava[2], Sudhanshu Agrawal[3]

[1] Command Hospital Pune, INDIA
[2] Command Hospital Kolkata, INDIA
[3] Best Hospital, Jabalpur, INDIA

ABSTRACT

Background: Cerebral Arteriovenous Malformations (AVM) have been conventionally treated with surgery, embolization and/or Gamma Knife Radiosurgery (GKRS). This article is to present a rare complication after Embolization/ Gamma Knife Radio Surgery for Cerebral AVMs.

Method: 05 patients with cerebral AVMs presenting with an unusual complication of a mass lesion at the site of the treated lesion were treated. Two modes of index treatment were used: endovascular embolization and/or GKRS. These patients developed new-onset neurological deficits at varying intervals after index treatment. They were investigated radiologically, revealing a mass lesion at the site of the treated AVM. The pathophysiology of this complication, along with the management algorithm, has been studied and is presented.

Results: All the patients in the series responded well to surgery. The histopathological examination revealed vascular elements in all cases without any evidence of neoplasm.

Conclusion: Delayed presentation as a mass lesion of a treated AVM is unusual. The mass lesion in cerebral arteriovenous malformations with suspicion of malignant transformation or with unresponsive raised intracranial pressure may mandate craniotomy and excision.

Keywords
cerebral AVM,
gamma knife radiosurgery,
radiation necrosis,
mass lesion

Corresponding author:
Darpan Gupta

Command Hospital Pune, India

drdarpangupta@gmail.com

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INTRODUCTION

Cerebral arteriovenous malformations (AVMs) are abnormal connections of arteries and veins of the brain, resulting in arteriovenous shunting of blood. The population prevalence of brain AVM is estimated to be 10–18 per 100,000 adults with a new detection rate (i.e., incidence) of approximately 1 per 100,000 person-years[1]. Overall mortality rates in AVM patients range from 0.7%–2.9% per year[2]. The commonest presentation of the cerebral AVMs is intracranial haemorrhage. Seizure is the second most common presentation of AVM, which occur in 20%–45% of patients[3].

The various modalities conventionally used to treat cerebral AVMs are embolization, Gamma Knife RadioSurgery (GKRS) and surgical excision4. The choice of treatment depends upon the size of the lesion, location of the lesion and venous drainage of AVM, consolidated and summarized as Spetzler Martin Grading.

Physiology of AVM treatment with embolization and Gamma Knife Radiosurgery

With the cyno-acrylate glue is deposited in the feeders of the AVM, the venous channels collapse, vascular channels undergo constriction and the nidus shrinks[5, 6]

Gamma Knife Radiation induces intimal necrosis with resultant obliteration of the abnormal vascular channels and shrinkage of the AVM nidus. Immediate post procedure perinidus oedema after Gamma Knife Radiation is not uncommon and can be bothersome, especially in cases, where the nidus size is big and maximum radiation dosage is used.

Adverse Effect Profile of AVM embolization and Gamma Knife RadioSurgery

Adverse effects after embolization

Some minor side effects may be observed shortly after embolization of an AVM by some patients. The side effects are usually temporary and should subside within a few days to weeks[7].

Headaches are not infrequently reported. Other possible complication is stroke like symptoms such as weakness in one arm or leg, numbness, tingling, speech disturbances and visual problems. The risk of permanent stroke or death after embolization is low.

The risk of bleeding reduces, but doesn't become zero after embolization of AVM cases. Residual risk of bleeding is directly proportional to the initial size of the AVM and the size of residual nidus post embolization.

Adverse effects after Gamma Knife RadioSurgery

The common short term complications, which can happen are:

a) Rebleeding
b) Incomplete obliteration
c) Encephalomalacia/encephalocele
d) Fresh neurological and cranial nerve deficits
e) Seizures
f) Persistent headaches
g) Alopecia at the local irradiated site

Majority of these events occurs within 3 years of radio-surgical treatment[9] The most common long term complication of Gamma Knife Radiosurgery are Radiation necrosis, cyst formation, Haemorrhage (CEIH) and increased seizure frequency. Radiation necrosis effects almost 33% of all patients, however clinical manifestations owing to Radiation Necrosis effect about 1.7 – 7.6% of all patients treated with GKRS.

We here, present an unusual complication which was noticed in few of our patients following embolisation/ GKRS, which has not been widely reported so far and has not been studied. We here present a series of five patients who underwent transarterial embolization for cerebral arteriovenous malformation, were asymptomatic in the initial post procedure period. On follow up, they presented with a space occupying lesion with surrounding oedema at the site of initial nidus, resulting in local and hemispheric mass effect.

All these patients presented with clinical features of raised Intracranial pressure, which didn't respond to decongestants and needed to be treated with decompressive craniectomy, to which they promptly responded with improvement in sensorium. The CEMRI Brain did not show any bleeding from the AVM or any evidence of a fresh acute event except the finding of fresh onset perilesional oedema with mass effect. The Histopathological examination of the mass revealed only vascular tangles and was devoid of any tumour tissue, even when each thin section of the excised mass was deliberately examined. The consent of the patients has been obtained for publishing this data.

Case No. 1

Ms IS

13yrs old girl presented with history of fall from treadmill with LOC for about 15 min followed by severe headache and vomiting in August 2012. MRI with MRA revealed Left sylvian fissure SAH due to left frontal AVM with intranidal aneurysm (Fig 1). Endovascular coiling of the intranidal aneurysm was done on 03 Oct 2012 (Fig 2) followed by GKRS for the left frontal AVM on 02 Feb 2013. DSA, 3yrs Post GKRS in Dec 2016 showed complete obliteration of AVM and no residual sac of intranidal aneurysm (Fig 3). She was asymptomatic for 9yrs Post GKRS. In Sep 2021, she presented with complaints of headache, vomiting and nuchal pain. NCCT Head + CT

Angiography followed by CEMRI Brain showed mass lesion in left sylvian fissure region with no aneurysm (Fig 4a). She was initially managed with Mannitol, Lasix and Steroid therapy. She responded favorably with reduction in headache and vomitings. She was given steroids for 6 weeks and gradually tapered off. Within one week of stopping steroids, she presented again with features of raised intracranial pressure. She was readmitted and underwent excision of the mass lesion in Oct 2020 in view of recurrent features of mass effect and diagnostic dilemma between radiation necrosis and malignant transformation. Intraoperatively, it was found to be a well-defined, firm, vascular mass lesion localized in the left sylvian fissure with well-defined planes abutting the coiled aneurysm.

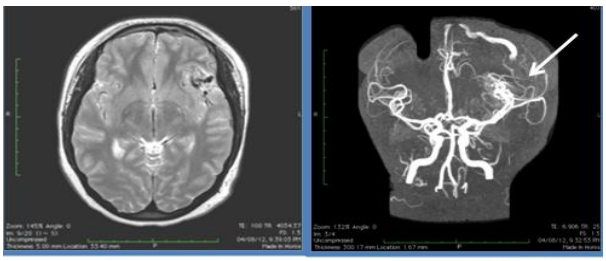


Fig 1 : MRI Brain and MR Angio at presentation (Aug 2012)

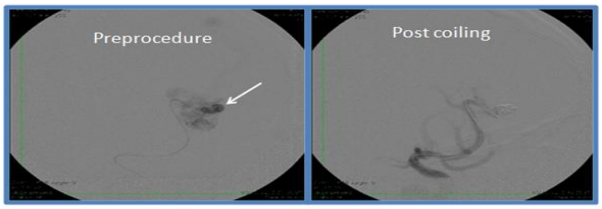
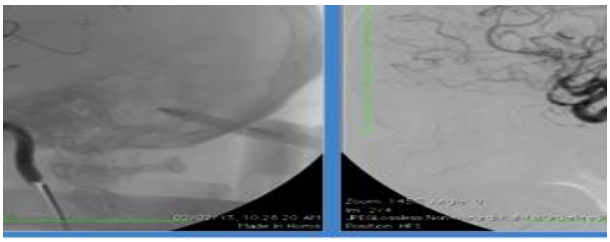


Fig 2 : DSA showing AVM with intranidal aneurysm and post coiling



entation **Fig 3B** Interval

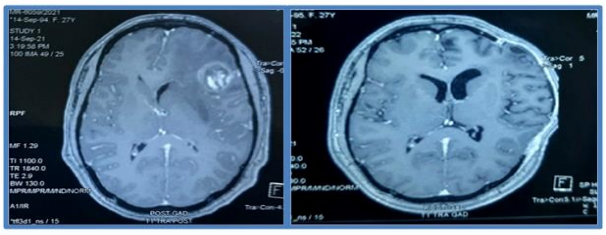
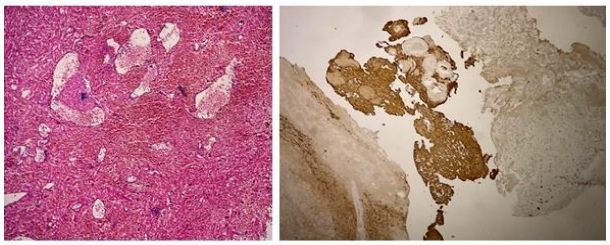


Fig 4 : Preop and postop MRI (Sep 2021)



5A : Large areas of haemorrhage, d necrosis, ectatically dilated blood **Fig 5B** GFAP IHC highlights gliotic brain tissue vessels between the hyalinised blood vessels

HPE – vascular elements interspersed with gliotic brain (Fig 5A & B)

Case No. 2

Ms YK

This 39yrs old lady patient presented with left sided hemiparaesis in Apr 2013. Investigations revealed Right Parietal AVM sized 46 x 38 x 49mm. The patient underwent Gamma Knife Radiosurgery for this AVM at Army Hospital R & R on Jun 2013. Post procedure angiogram runs revealed 80% reduction in the AVM nidus size (Nidus size 10 x 8mm on MRI Angiogram in Dec 2016 and 6.2mm diameter in MRI with Angiogram in Nov 2017). The patient remained asymptomatic for about 7yrs post treatment (till Jan 2020). In Jan 2020, patient presented with progressive left hemiparaesis. Investigations revealed an ill defined 16 x 12 x 14mm SOL in right parietal region with perilesional oedema and adjacent cystic lesions (tumefactive cysts) with toal conglomerulate measuring 44 x 41 x 55mm. CT angiogram and DSA revealed no patency of vessels at the location of the nidus. The patient was managed with decongestants and conservative measures, and the hemiparaesis improved (and so did the oedema radiologically). She presented again in Apr 2020 to a nearby hospital in Dehradun (due to COVID restrictions on inter-state travel) with left hemiplegia and altered sensorium. NCCT Head and MRI Brain revealed 30 x 20 x 10mm mass lesion in the right parietal region and surrounding oedema with adjacent tumefactive necrotic cysts (total volume 7 x 5 x 5cm) with mass effect and midline shift to the left side. She was initially given a trial of elective ventilation and decongestants, however there was no improvement in the neurological status and hence, Right Parietal Craniotomy and excision of the lesion was done.

Histopathology - blood clots, areas of infarction, multiple variable sized vessels and gliosed brain tissue with no features of any neoplasm (Fig 6A).

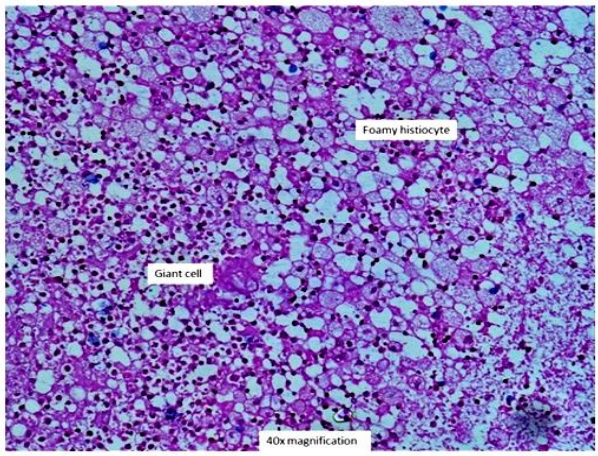


Fig 6A : HPE of case 2 Sheets of foamy histiocytes, inflammatory cells and an occasional giant cell

Case No. 3

Mr NK

This 43yrs old male patient was treated for AVM in Left Parietal region. Patient first presented with throbbing headache in Mar 2017. He was detected to be having superior sagittal sinus thrombosis with no abnormality on MR Angiogram. On follow up MRI with MR Angiogram and Venogram, he was found to be having partial recanalisation of superior sagittal sinus with Dural based AVM in right parietal region. He underwent glue embolisation in Dec 2018. Follow up MRI in Aug 2019 revealed an ill-defined mass lesion in the left parietal region (21 x 33 x 22mm) with mild perilesional edema. Follow up imaging (Aug 2020) revealed an increase in the size of this lesion (now 37 x 37 x26mm).

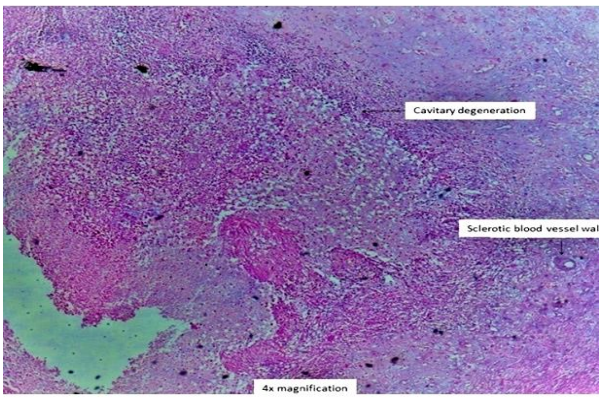


Fig 6B HPE of case 3 :- Cavitary degeneration containing foamy histiocytes and inflammatory cells with surrounding thickened sclerotic blood vessel walls

CT angiogram and DSA Cerebral vessels did not show any recanalisation of vascular components of the nidus. In view of progressive left hemiparaesis, radiological progression of the lesion and doubt in diagnosis, the patient was subjected to craniotomy and excision of the lesion on 26 Aug 2020.

Histopathology- glial brain tissue with foam cells and vascular elements consistent with AV malformation (post embolisation) (Fig 6B).

Case No 4

Mr SK

This 29yrs old male patient had an AVM in Lt Temporo-parietal region. Patient presented with seizures in Feb 2007. Investigations revealed AVM sized 65 x 63 x 66mm in Lt Temporo-parietal region. The patient underwent coil embolization for this AVM at Army Hospital R & R in 2007. Post procedure angiogram runs revealed 80-90% reduction in the AVM nidus size. He subsequently underwent Gamma Knife Radiosurgery in staged fashion on May 2017 and Feb 2018. The patient remained asymptomatic for 2yrs post treatment. In Jul 2019, patient presented with Right Hemiparaesis and dysphasia. NCCT Head revealed an ill defined SOL in the left Temporo-parietal region with perilesionaloedema, with adjacent tumefactive necrotic cysts and midline shift to Right side of 12mm. CT angiogram and DSA revealed 65 x 63 x56mm vascular lesion with minimal revascularisation. The patient was managed with decongestants and conservative measures, however the symptoms intensified and to relieve the raised Intracranial pressure the patient was subjected to Left frontotemporoparietal decompressive craniectomy and excision of the lesion on 27 Jul 2019.

Histopathology revealed a tangle of vessels and no features of any neoplastic transformation (Fig 6C).

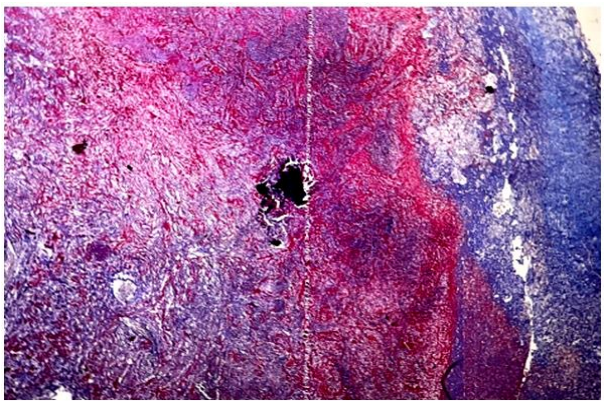
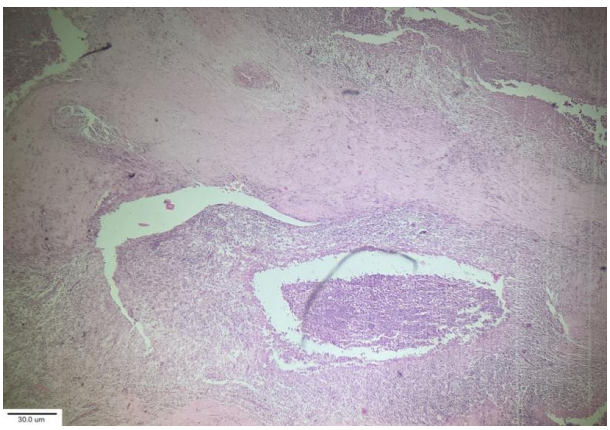


Fig 6C : HPE slide of Case 4, Masson Trichrome stain highlights Fibrin (reddish areas) and areas of fibrosis (Blue)

Case no 5

Mr HD

This 29yrs male presented with sudden onset right hemiparaesis on 04 Aug 24. His NCCT Head revelaed large bleed on left frontal region extending towards left basal ganglia. His DSA cerebral vessels revealed Lt frontal AVM 9x9.1x11mm in size with feeders from Lt MCA and draining vein draining to SSS. He was managed conservatively for the bleed and was taken up for endovascular embolisation of the AVM on 29 Aug 2024. He recovered symptomatically and his post procedure angiographic runs showed 80% obliteration of the nidus. He was discharged with follow up advice. He remained well till 16 Feb 2025 (~ 6months from initial treatment), when he developed right hemiparaesis and was admitted. His MRI Brain revealed a large 38 x 34 x 30mm mass lesion at the site of treated nidus. He was managed with decongestants and had slow, but appreciable symptomatic recovery. He was discharged and remained well for 3months, however in May 2025, he again had similar symptoms and was admitted. NCCT Head revealed mass lesion with massive surrounding oedema. He was given a trial of decongestants, however, he didn't show much improvement and was ultimately taken up for decompressive craniectomy with excision of the lesion on 19 Jun 2025. Histopathological examination of the sample revealed chronic inflammatory changes within congested blood vessels. (Fig 6d)



Result

We have described a clinical entity which effected five patients of cerebral AVM treated at our centre with embolisation or GKRS or both (in a sequential fashion) (**Table 2**), (out of total 106 cerebral AVM patients treated in the given time frame). At varying intervals from the index treatment, they presented with features of raised intracranial pressure and their management algorithm has been elucidated. The pathophysiology of the said complication has also been studied. In view of unresponsive cerebral oedema, they needed decompressive craniectomy and excision of the nidus. The histopathological examination of the excised lesion revealed vascular elements, glial brain tissue and no neoplastic transformation. They all responded well to the treatment.

Discussion

The ideal goal of cerebral AVM treatment is to completely obliterate the AVM nidus and normalization of cerebral hemodynamics[8]. This is to emphasise the possibility of delayed radiation necrosis at the site of treated AVM, presenting as a mass lesion with mass effect. Earlier, there have been isolated case reports for delayed presentation with increased intracranial pressure after Radiosurgery[10], however similar entity following embolisation has not been reported. The management of such an entity should be initially with decongestants, however in non-responsive cases decompression of the lesion with or without decompressive craniectomy may be needed.

The preoperative evaluation of such cases is aimed at:

a) Detecting whether there is a residual or recanalised AVM nidus with or without rebleed
b) If there is no AVM nidus or rebleed identified, then to differentiate between radiation necrosis and post radiation malignant transformation

Various modalities which can be instituted to differentiate between radiation necrosis and malignant tranformation are:

a) NCCT Head and CEMRI Brain – indirect evidences like cystic degenration more common with radiation necrosis, enhancing lesions and distant non contiguous lesions more suggestive of malignant transformation
b) MR Perfusion Imaging – increase in local perfusion is indicative of malignant transformation
   (i) PET Scan - FDG PET has a sensitivity of 65-81% and specificity of 40-94% to differentiate between radiation necrosis and tumour recurrence.

Other PET modalities:

(ii) F-F DOPA: 3,4 dihydroxy 6,18F-fluoro-L-Phenylalanine has a specificity of 86% and sensitivity- 98% to differentiate between radiation necrosis and tumour recurrence.

(iii) F-FET: O-2-18F-fluoroethyl-L-tyrosine has specificity of 100% and sensitivity- 93%

(iv) C-METL-methyl-11C-methionine has specificity- 83% and sensitivity- 88%

However, it is important to note that these statistics of the PET Scan modalities have been validated for known cases of neoplastic conditions, which underwent radiation therapy and are on follow up and may not hold true for the Post GKRS cases of cerebral AVMs.

Suggested Pathophysiology of this complication after embolisation is likely to be related to the normal perfusion pressure breakthrough (NPPB), impaired venous drainage, and embolization-induced angiogenesis. This in turn means formation of collaterals around the embolised arterial elements. These may be vessels with abnormal wall and permeability, thus causing gradual extravasation around the nidus, ultimately leading to cumulative oedema and presentation as a mass lesion. Pathophysiology following GKRS is more elusive and may be related partially to radiation necrosis and partially to neoangiogenesis.

Even after a deliberate investigatory protocol, it may not be possible, at least in few cases, to conclusively differentiate between post radiation necrosis and malignant transformation, and diagnostic dilemma may form an independent indication to operate, especially in younger individuals. Increasing, non-responsive oedema with raised intracranial pressure and consequent neurological deficits may warrant surgical decompression with removal of the conglomerate at the AVM site, in its own merit.

This is to generate familiarization about this entity, which may either be under treated considering it to be a post treatment effect or over treated considering it to be malignant transformation of a benign lesion. However, when there is a persistent diagnostic dilemma, compounded with the non-responsive clinical condition of the patient, surgical excision should be readily offered. The epidemiological profile of the patient may also play a role in decision making.

This highlights the need of cautious follow up of these patients for a longer duration as two of these five patients developed the complication with raised intracranial pressure more than 5years after the embolisation procedure. To gain insight into the pathophysiology of such delayed cerebral reaction to a procedure, more cases of similar kind need to be studied with inclusion and exclusion of all scientifically important information.

Table 1. Spetzler Martin Grading

Spetzler- Martin Grading	Points	Supplementary Grading
Size, cm		**Age (in yrs)**
<3cm	1	<20
3-6cm	2	20-40
>6cm	3	>40
Venous Drainage		**Bleeding**
Superficial	0	Yes
Deep	1	No
Eloquence		**Compactness**
No	0	Yes
Yes	1	No
Total	**5**	

Conclusion

The follow up of a group of AVM patients treated with embolization/ GKRS revealed that, after a duration ranging from 1.5-12yrs, these patients presented with a mass lesion with significant perilesional oedema at the site of preexisting AVM. CT Angiogram and DSA revealed no significant flow in the vascular malformation. They were subjected to decompressive craniectomy and excision of the nidus. The histopathological examination of the excised lesion revealed vascular elements, glial brain tissue and no neoplastic transformation. This description is to notify a rare, serious and hitherto unexplained complication of cerebral AVM patients undergoing embolisation/ GKRS. Further studies with larger patient subsets are required to establish the pathophysiology of such a complication and also predictive factors at the time of index treatment.

Table 2. Summary of cases with course of illness and treatment rendered

S No	Age	Sex	Location of lesion	Size of lesion	Time of Treat-ment	Details of treatment rendered	Time lag for Symptom appearance after embolization	Deficits	Resolution of symptoms after craniotomy
1	22y	F	Left Temporal	6.5cc	2012	GKRS	9yrs	Headache, vomiting nuchal pain	Recovered
2	39y	F	Rt Parietal region	46 x 38 x 49mm	Jun 2013	Gamma Knife Radiosurgery	7yrs	Left hemiparaesis, LOC	Consious, oriented, hemiparaesis improving
3	43y	M	Lt parietal region	21 x 12 x 18mm	Dec 2018	Embolisation	1.5yrs	Right hemiparaesis	hemiparaesis improving
4	30y	M	Lt temporo-parietal region	65 x 63 x 66mm	2007 Feb 2018	Embolisation Gamma Knife Radiosurgery	12yrs post embolisation 1.5yrs post GKRS	Right hemiparaesis, Dysphasia	Partial resolution
5	29y	M	Lt Frontal		Oct 2024	Embolisation (onyx)	6months	Rt hemiparaesis	Weakness completely recovered

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