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The Effect of Deuterium-Depleted Water on the Quality of Life and Lifespan of an Astrocytoma Patient

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ABSTRACT

Diffuse astrocytomas (WHO grade II) are slow-growing yet ultimately progressive primary brain tumors with a substantial risk of malignant transformation. When surgical resection is not feasible, long-term observation becomes a key component of clinical management. In this context, deuterium-depleted water (DDW) has been proposed as a potential adjunctive therapeutic strategy. We present the case of a 34-year-old female patient, diagnosed in 2005 with a supratentorial diffuse astrocytoma following the development of right-sided neurological deficits and seizure activity. Neuroimaging demonstrated a large, irregular lesion suggestive of early anaplastic transformation; however, histopathological analysis confirmed a WHO grade II astrocytoma. Given the tumor's location, surgical resection was not feasible, and the patient was managed conservatively with radio- and chemotherapy and long-term clinical and radiological monitoring. The patient had 4 courses of DDW treatment in 4 years. Follow-up of the patient's condition demonstrated that deuterium depletion counteracted the growth of the tumor. The data also revealed that an early break in DDW consumption promoted progression of the disease, as confirmed by MRI scan. This study confirms that deuterium depletion is an effective adjuvant therapy when used alone and prolongs life when conventional treatments have been exhausted.

Keywords: Astrocytoma; Hemiparesis; Deuterium (D); Deuterium-depleted water (DDW); Radiotherapy; Chemotherapy; Case study

INTRODUCTION

In addition to established cancer treatment modalities such as surgery, radiotherapy, and chemotherapy, growing attention has been directed toward metabolic strategies that target tumor cell biology. Among these, the use of deuterium-depleted water (DDW) has emerged as a novel, still debated approach.

Since the early 1990s, increasing attention has been devoted to elucidating the biological functions of naturally occurring deuterium (D, the heavy isotope of hydrogen) particularly in relation to cell proliferation[1]. It is now widely recognized that naturally occurring D plays a significant role in various biological processes, including

controlling the cell cycle, influencing gene expression, and (consequently) being involved in the development of cancer[2]. In addition to these effects, deuterium also affects a broad range of physiological processes, such as aging[3], long-term memory[4], cellular metabolism[5,6], and physical performance[7]. Reduction in D levels is typically achieved by consuming DDW, the apoptosis-triggering effect of which has been observed both *in vitro* [8] and *in vivo*[9-12]. Furthermore, reducing D availability can inhibit tumor cell proliferation by increasing oxidative stress[10,11] and also inhibits cell migration[13]. An earlier study suggested that combining D depletion with conventional therapy can increase median survival in patients with glioblastoma multiforme; indicating that DDW could function as a supportive adjunct, potentially improving treatment outcomes[14]. Such detailed longitudinal case analyses may provide valuable insights into potential biological effects and clinical relevance.

The incidence of astrocytomas is relatively low compared with other malignancies, yet they represent a significant proportion of primary central nervous system tumors. Population-based studies report an overall incidence of astrocytomas from approximately 0.2 to 3.2 cases per 100,000 persons per year, varying by population and tumor subtype[15]. Diffuse astrocytomas, or IDH-mutant diffuse astrocytomas, account for approximately 20–25% of adult gliomas. They most commonly present in patients aged 30-40 years, with a slight male predominance and a predilection for the frontal lobe. These tumors are generally slow-growing; however, prognosis is strongly influenced by tumor grade, extent of resection, and molecular features, including CDKN2A/B status[16]. Although classified as low-grade based on histopathology, these tumors have a notable tendency to undergo malignant progression to higher-grade gliomas, such as anaplastic astrocytoma and glioblastoma, which is associated with a significant reduction in overall survival[16]. In this report, we present the case of a 34-year-old female patient with a tumor classified as a diffuse astrocytoma (WHO grade II) according to the fourth edition of the World Health Organization (WHO) Classification of Tumors of the Central Nervous System (CNS)[16-18]. The tumor, due to its location and poor response to conservative therapy was managed for limited time after diagnosis. The aim of this study was to assess the impact of deuterium depletion on the patient's outcome, with particular emphasis on the period following the initiation of DDW consumption and its potential association with tumor progression dynamics and overall clinical course.

CASE DESCRIPTION

The 34-year-old female patient weighing 52 kg was diagnosed with brain cancer on 23 May, 2005.

At first, she sought medical attention, with right-sided hemiparesis. She was hospitalized following a right-sided seizure and was unable to walk independently due to increased muscle tone in the right leg and impaired movement of the right foot, requiring walking aids. Neuroimaging revealed a left-sided central hypodense tumor. MRI showed an irregular supratentorial mass located on the parietal convexity, measuring approximately 52 x 33 x 46 mm (Figure 1), with a garland-like appearance.

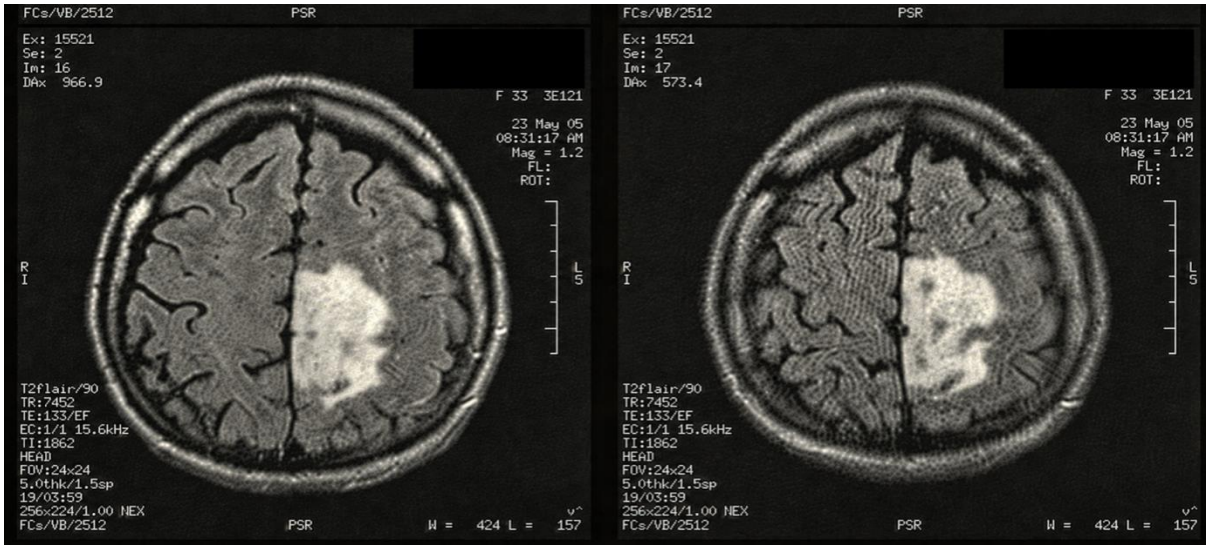

Figure 1: MRI sections of the brain tumor are acquired at different levels at the time of diagnosis (23 May, 2005).

Imaging findings suggested early anaplastic transformation, but the stereotactic biopsy confirmed a WHO grade II (A2) diffuse astrocytoma. Due to the tumor's location and relatively mild symptoms, surgical intervention was not planned.

The patient was subsequently closely monitored through regular clinical evaluations as well as personal and telephone follow-ups. For easier understanding, her medical history from diagnosis to death is presented here as divided into two main periods: from diagnosis to initiation of DDW consumption, and from the start of DDW intake until death. The latter period was further subdivided into three phases according to clinical status and treatment interventions.

First period

The first period from diagnosis to DDW, covers a total of 191 days.

From 13 June 2005 to 16 August 2005, the patient received fractionated photon radiotherapy using 3D CT-planned fields, delivered with a Mevatron KD2 device. The total radiation dose was 60 Gy, administered in daily fractions of 2.0 Gy. Following these treatments, she reported cold sensations in the lower extremities and hip pain. On 15 November 2005, she was rehospitalized after experiencing a generalized epileptic seizure at home. Her seizure activity had begun one month earlier and three attacks occurred in total. Laboratory tests revealed mild hypokalemia. A follow-up MRI performed on 14 November 2005 indicated disease progression. Between 21 November 2005 and 13 February 2006, she received three cycles of chemotherapy with 300 mg Bis-chloroethylnitrosourea **(**BCNU).

Second period

The second period began with initiation of DDW consumption on 30 November 2005, 191 days after diagnosis, between the first and second BCNU chemotherapy cycles. It includes periodic intakes of DDW over the

subsequent four years (a total duration of 1,306 days) up to the time of death. During this time, the patient met her daily water demand by drinking 1.5 to 2 L of Preventa DDW of 105, 85, 65, and 45 ppm D concentration, instead of normal water. The three cycles of her BCNU were followed by Temozolomide therapy by the end of May 2006, when all conventional therapy due to progression of the disease was stopped. Table 1 and Figure 2 summarize the DDW consumption intervals during this period, including the corresponding concentrations of DDW.

Table 1: DDW consumption during the four-year follow-up period

Duration of DDW consumption	DDW concentration/ppm	Number of days
30.11.2005-26.02.2006	105	89
27.02.2006-15.01.2007	85	323
16.01.2007-07.01.2008	65	357
08.01.2008-18.01.2008	45	11
19.01.2008-09.02.2008	break	22
10.02.2008-02.03.2008	105	22
03.03.2008-24.03.2008	85	22
25.03.2008-15.04.2008	65	22
16.04.2008-09.05.2008	45	24
10.05.2008-20.06.2008	break	42
21.06.2008-12.07.2008	105	22
13.07.2008-03.08.2008	85	22
04.08.2008-24.08.2008	65	21
25.08.2008-30.09.2008	break	37
01.10.2008-16.10.2008	105	16
17.10.2008-01.11.2008	85	16
02.11.2008-16.11.2008	65	15
17.11.2008-27.06.2009	45	223

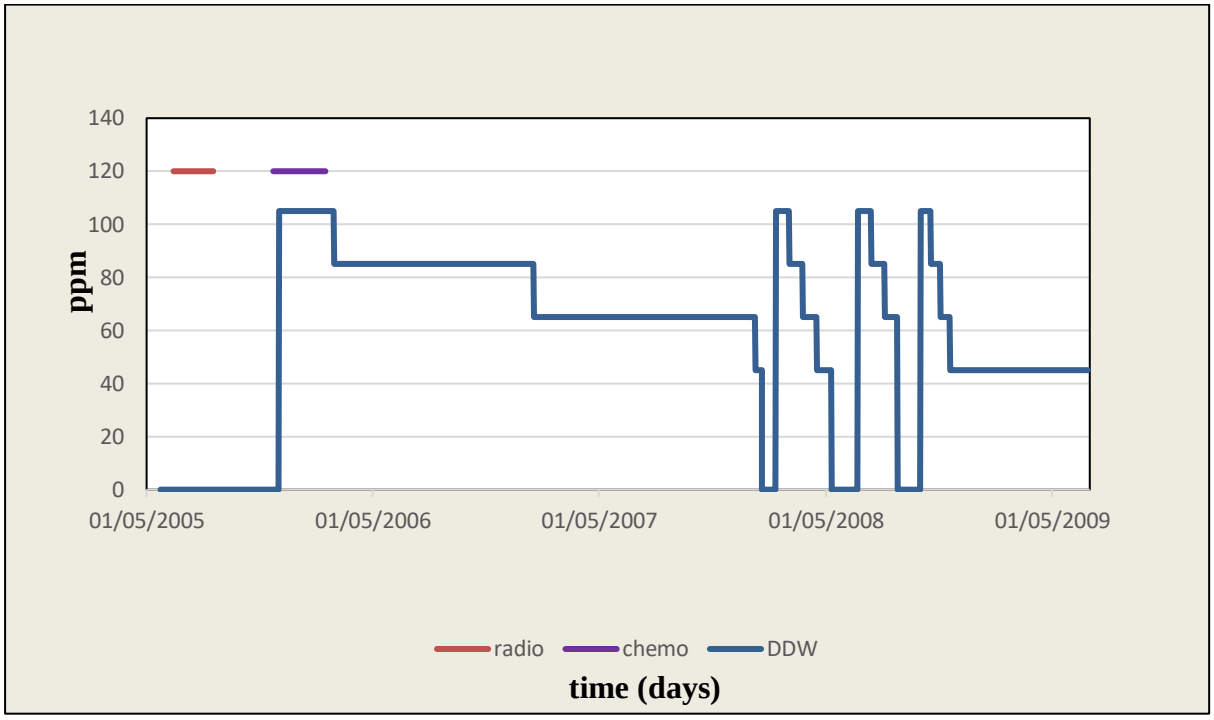

Figure 2: Indicating radiotherapy, chemotherapy, and DDW consumption during the four-year follow-up period.

Second period - first stage

The first stage of the second period covers the time during which the patient was already consuming DDW while still receiving chemotherapy, from 30 November 2005 to 23 May 2006, a total of 175 days.

During this period encompassing the two BCNU chemotherapy cycles, the patient experienced multiple episodes of malaise, including twitching of the right leg and hand with preserved consciousness. In some instances, attacks occurred in succession without complete resolution, evolving into low-amplitude tremors resembling chills, accompanied by painful muscular tension and subsequent worsening of leg paresis. She also reported severe, diffuse tension-type headaches involving the entire head, described as intense, with a burning sensation and a persistent feeling of elevated temperature. On 27 February 2006, three months after initiation of DDW consumption, follow-up MRI demonstrated slight progression compared with the previous scan. The posterior fronto-central lesion appeared minimally enlarged, with increased necrotic content, and a new, more caudal nodular lesion was identified. These findings were interpreted in the clinical context as potentially reflecting necrosis-associated inflammatory and immune processes. In light of them, BCNU chemotherapy was discontinued, and antiepileptic medication was modified. Following this adjustment, the patient reported no further acute episodes of illness. On 6 March 2006, laboratory results were within normal limits, and treatment with temozolomide (Temodal, 5 x 200 mg) in combination with ondansetron (Zofran) was continued. Subsequently, she developed anxiety disorder associated with fatigue, general malaise, and recurrent headaches accompanied by a burning sensation. From 22 March 2006 onward, her general condition deteriorated, with urinary difficulties and persistent headaches. On 3 April 2006, temozolomide therapy was postponed due to leukopenia. One month later, she experienced seizures, limb rigidity, and severe cramps, which were managed with antiepileptic medication and glycerol-based dehydration therapy during hospitalization. By 23 May 2006, due to her significantly worsened condition, further temozolomide treatment was no longer administered.

Second period - second stage

The second stage of the second period covers the time during which the patient was receiving only DDW and experienced clinical regression. This phase lasted from 24 May 2006 to 27 September 2008, totaling 857 days. Figure 3 shows the MRI scan performed on 29 May 2006, demonstrating the initial condition at the beginning of the second period of the second stage. The MRI report stated that the left-sided centro-parasagittal tumor regressed.

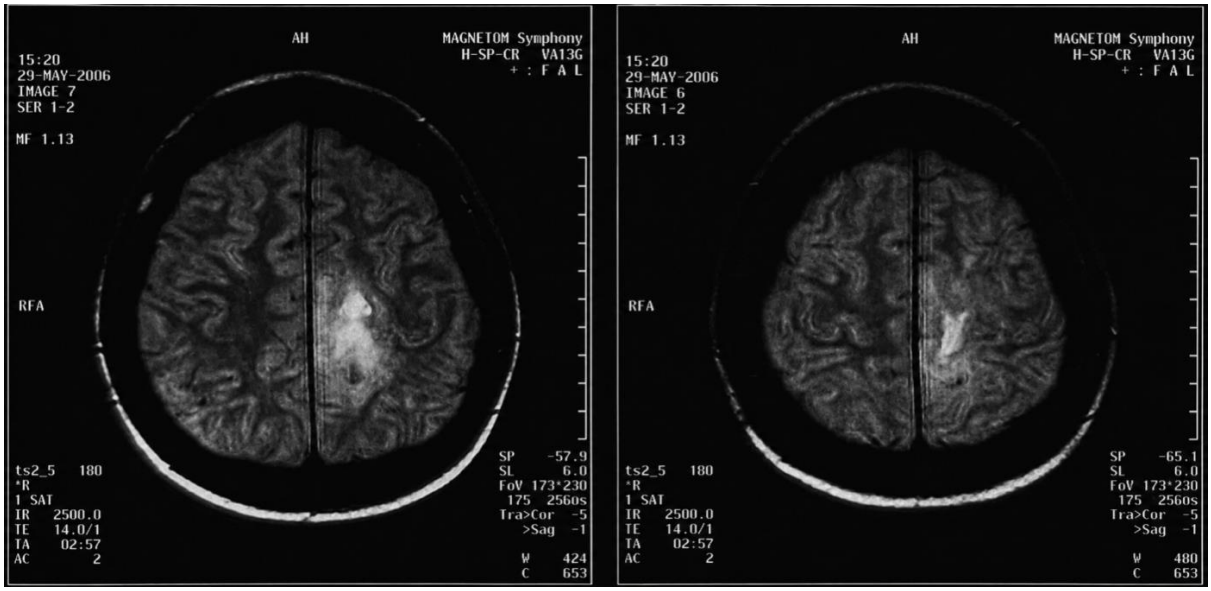

Figure 3: MRI sections of the same brain tumor acquired at different levels taken at the time of the beginning of the second period's second stage (29 May 2006).

The patient's condition showed continuous, gradual improvement. On 21 June 2006, she was able to walk independently, without assistance, within her home. In July, she reported improved sleep quality, along with good mood and overall well-being. On 21 August 2006, MRI findings (Figure 4) demonstrated moderate tumor regression, with a reduction in the necrotic components of the left central tumor and further regression of the ring-like enhancement. In August, she regained the ability to walk downstairs independently.

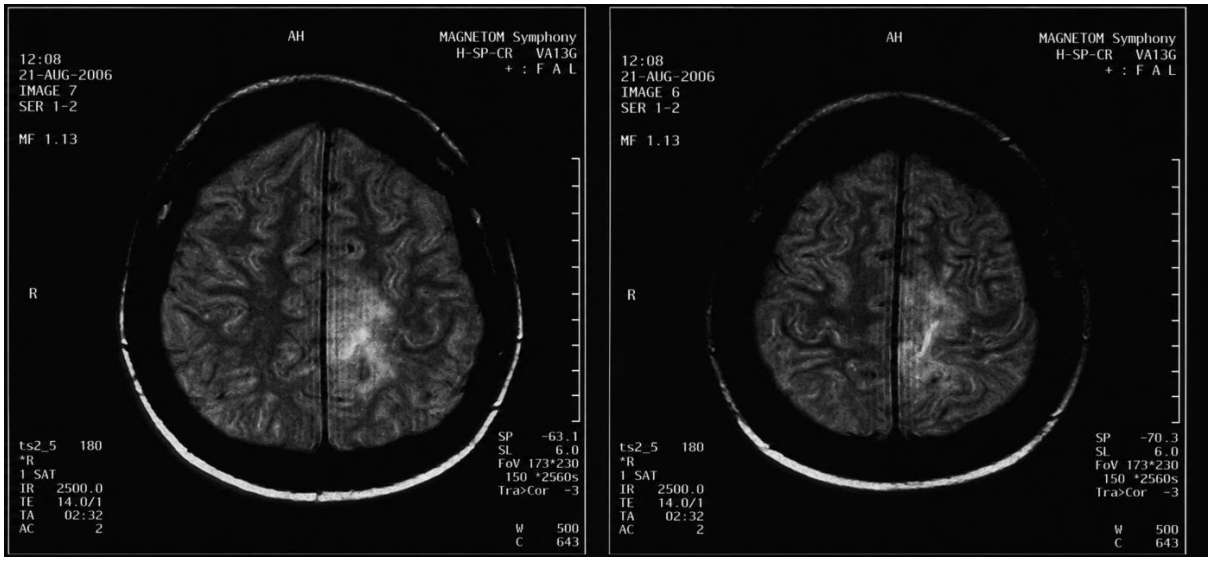

Figure 4: MRI sections of the same brain tumor acquired at different levels. The state of the brain in an MRI scan taken on 21 August 2006.

The patient reported in January 2007 that her walking ability had significantly improved, and she was able to go outside into the garden independently. However, she experienced marked anxiety, nocturnal teeth grinding, and back and neck pain. Hair loss was also noted at the radiation site, and she reported an inability to maintain a positive mood. A sedative medication was therefore prescribed. In February 2007, she regained the ability to move her toes. Two months later she reported that she had not required sedative medication for one week, was walking frequently. A clinical examination on 24 April 2007 confirmed functional improvement. In June 2007, MRI findings demonstrated regression of the left fronto-central lesion and reduced contrast enhancement. By August 2007, she was able to perform more extensive movements, actively push off with her foot, and climb stairs using an alternating gait pattern. Under ongoing medication, she remained free of epileptic seizures. From October 2007, she underwent musculoskeletal rehabilitation. Her mood and general condition were stable, and she was able to move independently, although her gait remained mildly spastic-paretic but stable. On 20 November 2007 (Figure 5), further clinical improvement was observed, and MRI findings showed no change compared with the previous examination in June 2006. Irregularly shaped edema was observed around the lesion.

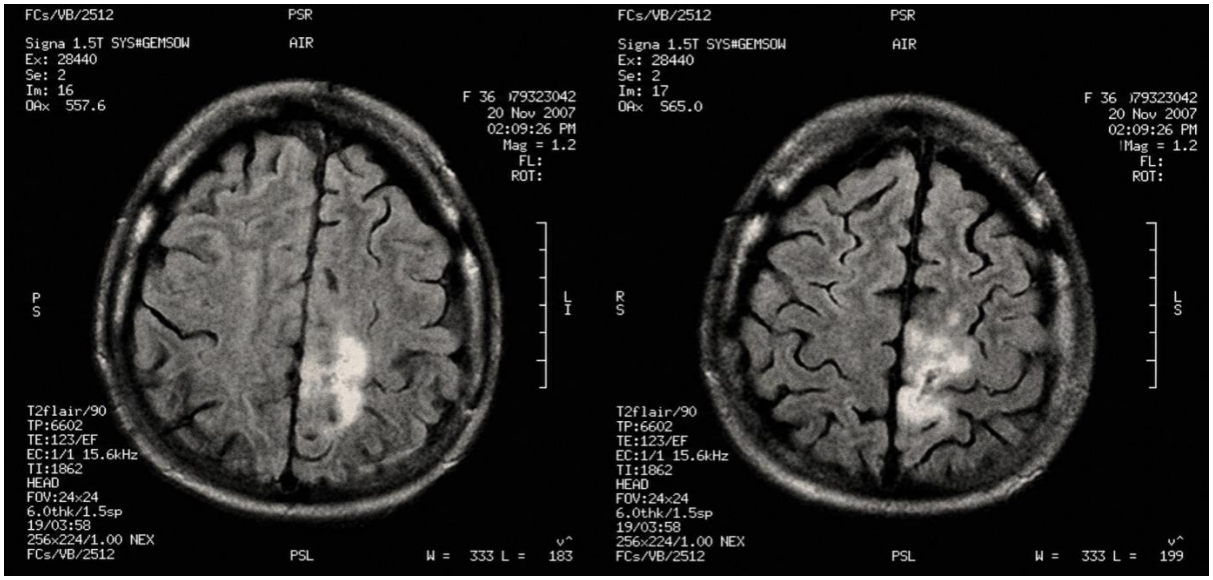

Figure 5: MRI sections of the same brain tumor at different levels, showing the brain's condition on scans obtained on 20 November 2007.

(The MRI images presented in this paper were acquired at two different clinics. The images in Figures 1 and 5 were obtained with one MRI device, and those in Figures 3 and 4, using another machine. Mind this when comparing images from different dates.)

In April 2008, the patient reported further significant improvement, including better foot mobility, the ability to stand on her toes, and a substantial increase in functional capacity, allowing her to ride a bicycle and to clean windows. Clinical examinations performed on 25 April, 10 June, and 10 July 2008 demonstrated continued improvement compared with previous assessments, and no epileptic seizures were reported during this period. A notable factor associated with her condition was a vacation abroad to Turkey with her husband, which she experienced as highly positive and without complications.

Second period - third stage

The third stage of the second period covers the time during which the patient was consuming only DDW and experienced disease progression. In the previous DDW courses, she had consumed 45 ppm water at the end; however, she finished the third course in August 2008 with to 65 ppm DDW (Table 1 and Figure 2). Her condition began to deteriorate. This stage lasted from 28 September 2008 to 27 June 2009 (date of death), totaling 273 days.

On 28 September 2008, the patient required emergency hospital care due to general malaise, numbness of the right side of the face, and increased muscle tone in the lower limb. She also experienced nausea accompanied by chest pressure, followed by muscle twitching in the right thigh. Treatment with alprazolam (Xanax) was administered. A follow-up MRI performed on 5 October 2008 revealed a new cystic and contrast-enhancing lesion in the left precentral region, suggesting tumor recurrence. In November 2008, the patient reported

recurrent seizure-like episodes occurring every 2–3 days. However, clinical evaluation suggested that these events were non-epileptic in nature, and symptomatic treatment with sedative medication was initiated.

On 23 January 2009, the patient experienced clinical deterioration, including difficulty speaking and impaired self-expression. She developed right-sided numbness with a subjective inability to move the affected side. An emergency cranial CT scan revealed a space-occupying lesion with extensive perifocal edema in the left fronto-temporo-parietal region, consistent with disease relapse. Within the edematous area, a 20 x 15 x 30 mm hypodense region was identified. An MRI on 23 February 2009 demonstrated marked progression on the left side, with the lesion measuring approximately 50 mm in diameter, surrounded by moderate edema. Part of the mass effect was attributable to a cystic component, next to the lesion. On 2 March 2009, a cyst puncture was performed, and a CT-guided stereotactic biopsy of the lesion was carried out in local anesthesia, and approximately 20 ml of cyst fluid was evacuated. Thirty minutes post-procedure, the patient experienced an abortive right-sided Jacksonian seizure, which resolved after administration of diazepam (Seduxen). Neurological function on the right side subsequently improved transiently. Histopathological examination revealed post-irradiation necrosis with small foci of tumor tissue, consistent with anaplastic astrocytoma. From 28 April 2009 onward, further clinical deterioration was observed, including increasing weakness of the right extremities and intermittent dysarthria. Cranial CT demonstrated extensive perifocal edema surrounding the known lesion with mild midline shift. Dehydration therapy resulted in only minimal improvement. On 5 May 2009, a repeat cyst puncture was performed, resulting in temporary clinical improvement followed by renewed deterioration. MRI on 5 June 2009 revealed a new recurrent left-sided intracranial mass lesion. The parietal lesion compressed the left lateral ventricle and brainstem, with peripheral contrast enhancement. The lesion measured approximately 60 x 40 x 55 mm, surrounded by extensive edema up to 100 mm in diameter. The posterior wall of the left lateral ventricle was displaced inferiorly, and the lateral ventricles were otherwise dilated. Further neuro-oncological treatment was no longer considered feasible. The patient died on 27 August 2009.

CONCLUSION

This case report presents a uniquely detailed longitudinal observation of a patient with diffuse astrocytoma (WHO grade II), highlighting the complexity and variability of the disease regression and progression. The structured analysis of clinical and radiological changes across distinct phases provides valuable insight into the natural history of the disease and the potential impact of conventional and non-conventional therapeutic approaches.

The study suggests that appropriately scheduled oral DDW consumption – a procedure shown to be safe in preclinical drug safety studies[19] may achieve a prolonged progression-free period and improved quality of life. During follow-up, repeated DDW treatment and break cycles, altogether four, were applied, with consuming water of increasing level of deuterium depletion. The first two treatments ended with 45 ppm D, and the following breaks did not result in significant changes in the patient's condition. However, the patient finished the third teratment with 65 ppm DDW, not reaching 45 ppm before the break. It is possible that omitting the strongest depletion contributed to the deterioration of her condition.

Overall, the above case suggests that DDW was associated with notable clinical improvement and delayed disease progression in a brain tumor patient with no remaining conventional therapeutic options, supporting the conclusion that deuterium depletion provides additional benefit and plays a significant role in delaying the progression of brain tumors.

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