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Case report

Cognitive impairment in elderly population is not always a dementia

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ABSTRACT

INTRODUCTION AND AIM

Cognitive impairment is frequent disease in elderly people, prompting to the suspection of dementia. Considering the multi-disease nature and complexity of the treatment process for the geriatric population, it is crucial to exclude the several other disease processes and to actively search for causes of cognitive impairment, especially reversible conditions whose treatment can significantly improve cognitive function.

DESCRIPTION OF THE CASE

An 89-year-old man was admitted to the Geriatrics Unit due to progressive cognitive impairment and motor signs with the suspected dementia diagnosis. After a broad physical examination, laboratory tests and neuroimaging, the brain tumor was detected. The treatment included antiedematous drugs- Mannitol and Dexamethasone. The treatment resulted in an improvement of the contact with the patient and his general condition.

CONCLUSION

This clinical case demonstrates the relevance of searching for less common causes of cognitive impairment, as well as the necessity of careful investigation of concomitant coexisting symptoms, which can often lead us to the right diagnosis .

Keywords: cognitive impairment, dementia, brain tumor, geriatric population, antiedematous treatment

INTRODUCTION

With the significant increase in life expectancy and the subsequent aging of the population, cognitive impairment, which often affects older people, is becoming a growing problem [1]. The spectrum of cognitive impairment is broad, and depending on its severity, it can be considered normal for age, classified as Mild Cognitive Impairment (MCI), or as dementia [1].In clinical practice, it is cardinal to note that cognitive impairment is not only a symptom of dementias such as Alzheimer's disease, Parkinson's disease, or vascular dementia. It may also be a symptom of many other pathological conditions that should be considered in the differential diagnosis [1,2]. Cognitive impairment can be caused by infections, central nervous system (CNS) injuries, hypothyroidism, brain tumors and other systemic tumors, depression and other mood disorders, electrolyte disturbances, vitamin B12 deficiency, alcoholism, and other addictions, among others. Cognitive status can also be affected by the treatment being used, for example with anticholinergics, antihistamines, or sedatives and sleep medications such as benzodiazepines or 'Z' drugs[2]. Considering the multi-disease nature and complexity of the treatment process for the geriatric population, it is crucial to exclude the aforementioned disease processes and to actively search for causes of cognitive impairment, especially reversible conditions whose treatment can significantly improve cognitive function [3,4,5]. The clinical case presented below demonstrates the relevance of searching for less common causes of cognitive impairment, as well as the necessity of careful investigation of coexisting symptoms, which can often guide the diagnosis in the right direction.

CASE REPORT

An 89-year-old man with a multimorbidity burden with a history of hypertension, arteriosclerosis of the lower limbs, hyperuricemia, osteoarthritis, and prostatic hypertrophy was admitted to the Geriatrics Unit due to progressive cognitive impairment, significant deterioration in performance, balance disorders, falls and lower limb pain with worsening for 2 weeks and weakness in left limb strength for 2 weeks. The patient permanently used amlodipine 5mg, lisinopril 10mg, simvastatin 20mg, torasemide 10mg, tamsulosin 0.4mg, vitamin D 1000, paracetamol 3x500mg, nicergoline 10mg, buprenorphine 35mcg in a transdermal system. The patient was addicted to nicotine for many years. The patient’s increasing disability for 2 months forced him to live with children. He has required assistance with meals, mobility, and toilet use. Before this time, he was independent, lived alone, and drove a car.

On physical examination swelling of lower limbs, bleeding subluxations in the left hip region, systolic murmur over the aortic valve, and no pulse on the dorsal foot artery and left

posterior tibial artery were found. On neurological examination features of left-sided pyramidal syndrome were found. Patient presented weakness in left limb's strength, falling to the left side in a sitting position - left hemiparesis, bilateral positive Babinski's sign, disturbed upward eye movements, and slightly increased muscle tone.

Laboratory tests showed mild chronic disease anemia (Erythrocytes- 3.61 x106, Haemoglobin-12.10 g/dl, Haematocrit- 35.1%, MCV- 97.2 Fl) and elevated D-dimers (3359ug/dl).

A comprehensive geriatric assessment of the patient showed functional disability in basic and instrumental activities of daily living- Barthel scale - 25/100 pts, I-ADL scale - 1/12 pts. Also, the patient presented an increased risk of falls (Tinetti scale and balance assessment test - 5/28 pts) and weakness of hand grip strength. A result of the MNA questionnaire- 10/14 points indicated the suspected presence of/ indicates probability of malnutrition. The psychological assessment was performed and the results were: in the MMSE test (Mini-Mental State Examination)-15 points, the Clock Drawing Test- 0/7 points, and the Geriatric Depression Scale (GDS)- 12/30 points. The patient showed reduced orientation to place, lack of orientation to time, and difficulty in maintaining attention at a constant level. Also, an impaired constructional praxis and significant impairment of visuospatial processes were found, which together indicated the possibility of dementia in terms of information processing.

Doppler ultrasound of the carotid and vertebral arteries showed atherosclerotic lesions in both CCAs, in the bifurcations of both CCAs, and in the initial segments of both ICAs - atherosclerotic plaques of mixed echogenicity protruding into the lumen to a width of 4.4 mm - morphological narrowing of 30-40%. The following were also assessed: Doppler parameters of blood flow in the carotid arteries-normal; blood flow in the intracerebral vertebral arteries-normal; velocity and spectrum of blood flow in the vertebral arteries-normal.

During hospitalization patient's condition deteriorated. The patient was periodically confused, with impaired consciousness and verbal contact. The patient also received quetiapine because he was anxious and aggressive. Due to the confusion, buprenorphine was discontinued and the dose of torasemide was reduced. A CNS CT [Image 1] showed a heterogeneous tumor mass of approximately 38x27mm (ax), c-c 28mm in the right frontal-parietal lobe compressing the right corner of the lateral ventricle with an adjacent extensive area of hypodensity in the right cerebral hemisphere and features of oedematous changes- obliteration of grooves and furrows, displacement of the midbrain and ventricular structures to the left up to a width of about 9-10 mm, narrowing and displacement of ventricle III up to a width of 5 mm and the

lateral triangle with significant tightening of the temporal horn, with a tendency to wedge into the notch of the tentorium on the right side.

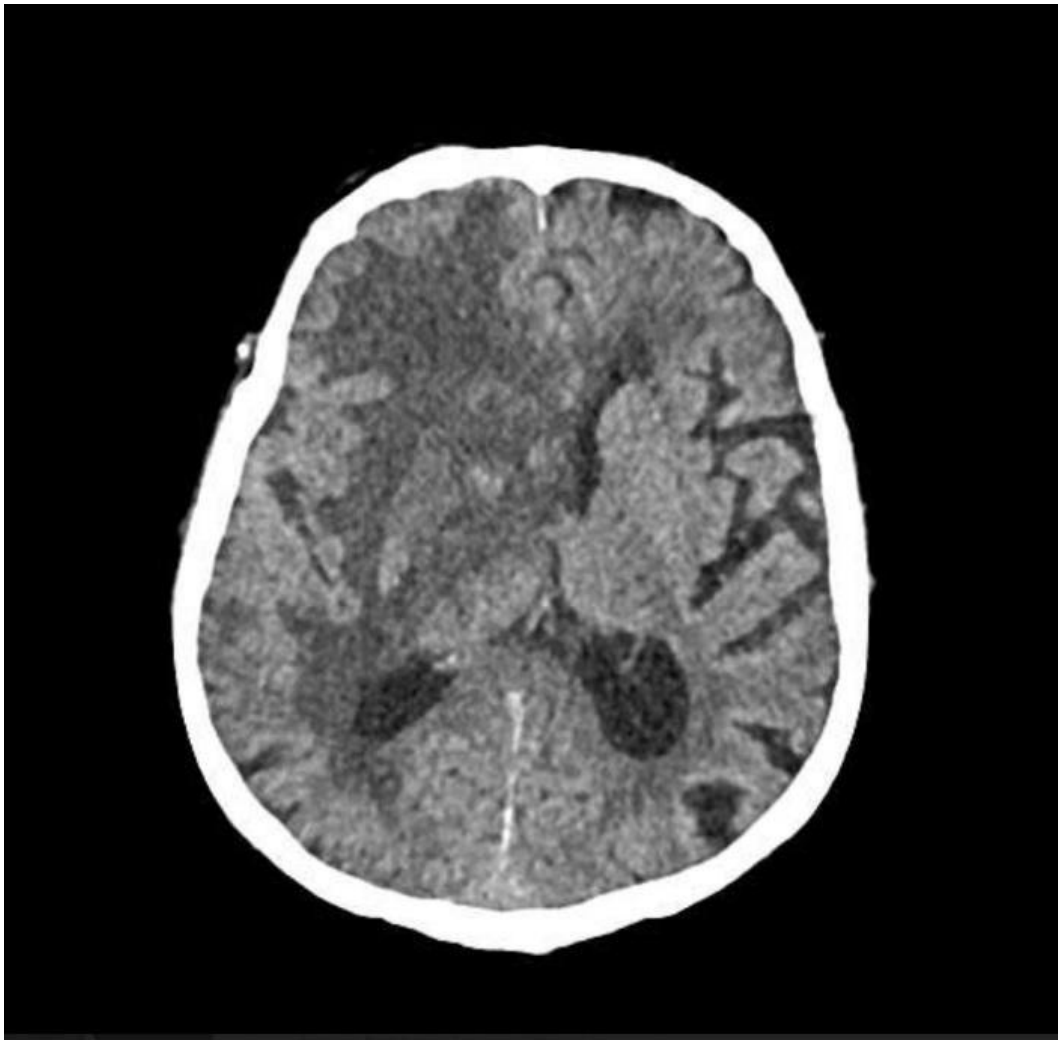

Image 1.

In addition, the CT visualized a dilatation and asymmetry of the ventricular system with displacement and distortion of the left frontal horn. In the left cerebral hemisphere were confluent areas of hypodensity in the white matter with cortical and subcortical atrophy and mediocre widening of the parietal-cerebral fluid spaces. Calcified atherosclerotic plaques were visualized in the intracranial segments of the internal carotid arteries and vertebral arteries. The CNS MRI imaging confirmed a diagnosis. It presented a nodular lesion with irregular contours, undergoing intense and heterogeneous contrast enhancement, measuring 33x24x27mm in deep

structures on the right side. The lesion was described to be surrounded by an extensive zone of edema, with shallowing of the furrows of the right cerebral hemisphere, and the mass effect exerted by the lesion - compression of the frontal horn and the body of the right lateral ventricle, displacement of the midline approximately 5mm to the left side, constriction and modeling of ventricle III. The MRI image was consistent with a neoplastic lesion. In addition, abnormalities described numerous vascular-like foci of varying size in the subcortical and periventricular white matter of both cerebral hemispheres and features of periventricular leukoaraiosis. The patient was consulted neurologically, oncologically, and neurosurgically. In addition, a multiphasic CT scan of 2 anatomical areas- thorax, abdomen, and pelvis was performed. No features of proliferative disease /metastases were found. During the neurosurgical consultation, it was recommended to consider a biopsy tumor's deep structures in the right cerebral hemisphere. The treatment included antiedematous drugs- 20 %Mannitol 100 ml 3x1 and Dexamethasone 8mg 2x1, which resulted in an improvement of the contact with the patient- contact became logical, and the patient followed the commands.

The patient was discharged home in a state of clinical improvement, with improved mobility and independence in eating and using the toilet, with the recommendation for the future admission to the neurosurgery clinic for metioned before biopsy the patient's family should report to the Neurosurgery Clinic for an appointment for admission to the Clinic - to consider a biopsy of the tumor lesion of the deep structures of the right hemisphere. Advised treatment: Vitamin D 1x1 tabl, Lisinopril 10 mg 1x1 tabl, Simvastatin10 mg 1x1 tabl, Tamsulosin 0.4 mg 1x1 tabl, Finasteride 1x1 tabl, Torasemide 5 mg 1x1 tabl, Dexamethasone 4mg 1x1, Quetiapine 25mg in case of delirium and nutritional treatment, oral nutritional supplements.

CONCLUSION

When cognitive impairment occurs in older people, the most commonly suspected diagnosis is dementia. The most probable types of dementia are: Alzheimer's disease, vascular dementia, Parkinson's disease, dementia with Lewy bodies, and frontotemporal dementia [6]. However, cognitive impairment also occurs in lotus of other pathological conditions that should be differentiated from dementia. In the geriatric population, several conditions [including reversible conditions] such as electrolyte disorders, endocrine disorders [especially thyroid function], vitamin deficiencies [especially vit.B12], neurological diseases, including brain tumors, psychiatric diseases, systemic tumors, infections, autoimmune diseases, treatment with sedative and sleep medications - especially from the benzodiazepine group, as well as drugs

with anticholinergic and antihistaminic effects, dependence on alcohol and other psychoactive substances, and many other less common conditions [2,3,4,5]. Therefore, the search for the cause of cognitive impairment and the differential diagnosis of dementia should be extensive and consist of medical history, physical examination, neuropsychological testing [such as e.g. MoCA - Montreal Cognitive Assessment and MMSE- Mini-Mental State Examination used as screening tests and advanced psychological testing], neurological examination, laboratory tests [including TSH and vitamin B12 levels] and structural brain imaging with CT or MRI [8]. Neuroimaging is mandatory during a diagnosis of dementia due to its high diagnostic value. It enables us to exclude neurological conditions causing cognitive impairment, such as stroke, normotensive hydrocephalus [Hakim's syndrome] or focal lesions causing pressure on the brain nerve tissue, e.g. hemorrhage within the brain [usually subdural hematoma], or - as in the case described here - brain tumor [9]. Neuroimaging can also present changes such as atrophy of brain tissue volume in specific areas [e.g. particularly in the hippocampus in Alzheimer's disease], vascular changes, assessment of flow, and perfusion. Another advantage is the gradual emergence of modern techniques with the ability to show characteristic changes in the brain for specific types of dementia. One of these is the study of brain tissue metabolism using fluorodeoxyglucose [FDG] in PET and the possibility of using ligands for amyloid in this study, showing changes in Alzheimer's disease or dopaminergic imaging with the possibility of visualizing changes in dementia with Lewy bodies [9]. Also, hybrid techniques such as imaging studies combined with functional studies, e.g. functional magnetic resonance imaging [fMRI], allow an in-depth examination of cognitive impairment in terms of disturbances in neuronal networks [9]. Currently, these modern techniques are for scientific research and are not included in the criteria for diagnosis of the disease entities in question, but in the future, they may play a decisive role in the diagnosis and treatment of these conditions [7,9,10]. In addition, if another etiology of dementia is suspected - extended investigations such as EEG; cardiovascular studies - ECG, ECHO, heart rate monitoring; vascular imaging studies - angio-CT/angio-MR of the head and neck; tests for autoimmune, metabolic, or infectious diseases are recommended [8]. Cerebrospinal fluid examination may also be useful in some diseases [8].

The most common cause of dementia worldwide is Alzheimer's disease, hence it is usually the first suspected diagnosis when cognitive impairment occurs [11]. According to WHO ICD-10 criteria, the diagnosis of Alzheimer's disease requires confirmation of dementia according to the criteria described above, as well as an insidious onset of the disease with a slow deterioration of functioning, exclusion of other conditions that may cause similar symptoms, and confirmation of the absence of focal symptoms at the onset of the disease [that

may appear later], such as hemiparesis or visual field loss, which are suggestive of other neurological conditions [12]. Alzheimer's disease, as a growing health and social problem in the geriatric population, is the subject of consideration by many scientific studies. More recent recommendations, such as studies by the NIA/AA society, introduce a biological definition of the disease, paying particular attention to the state of neurodegeneration in the CNS of sufferers [12] and the finding of biomarkers of neurodegeneration [12,13]. Of these, the most important are the metabolites associated with the pathological deposition of B-amyloid and tau protein in the cerebrospinal fluid and the characteristic changes described by PET imaging of the brain of patients with Alzheimer's disease [12]. Currently, testing of biomarkers of neurodegeneration mainly serves one research purpose but is not routinely used due to its high cost, lack of widespread availability, and invasive nature [12]. However, regardless of the initial suspicion of Alzheimer's disease at the onset of a patient's cognitive impairment, each case should be analyzed individually.

Depending on the location, brain tumors can be a reason of various symptoms [14]. The clinical presentation of a brain tumor can start with systemic symptoms resulting in increased intracranial pressure, such as epileptic seizures or headaches, as well as focal symptoms that result from the involvement of a specific area of brain nerve tissue [14]. The most common focal symptoms include unilateral hemiparesis, sensory disturbances, personality and behavioral changes, dementia, sensory and motor aphasia, unilateral visual disturbances, ataxia, dysmetria, nystagmus, cranial nerve dysfunction, and autonomic nervous system dysfunction [14]. The symptoms resulting from frontal lobe involvement may initially point towards a diagnosis of dementia, particularly in geriatric populations, as in the case of the patient described [15]. The frontal lobe of the brain is a key area responsible for cognitive functions such as memory, decision-making, language functions, emotions, and executive functions [14]. According to studies, all these areas of intellectual functioning can be impaired by the presence of a brain tumor [14]. In the case described here, features of left-sided pyramidal syndrome combined with significant cognitive decline initially raised the suspicion of a history of cerebral ischaemic stroke, the diagnosis of which was ruled out by imaging of brain tissue. At the same time, a tumor in the frontal and parietal lobes was visualized by brain imaging. The most common brain tumor in the adult population, including the geriatric population, is glioblastoma multiforme [16]. Advanced age is a recognized risk factor for this tumor, as well as a factor for poor prognosis, indicating the need for particular vigilance in the geriatric population [16]. In addition, a significant aspect in terms of the subsequent course of treatment is that the prevalence of cognitive dysfunction in people with brain tumors is not only due to the location

of the area of neural tissue involvement but also to subsequent therapeutic interventions [16,17]. The elderly population is at particular risk of developing cognitive dysfunction, thus it is crucial to choose the appropriate treatment method. Currently, the standard approach to glioblastoma multiforme, depending on the advancement stage of the tumor and the individual circumstances of the patient, includes surgery, radiotherapy, and chemotherapy. We observe a thriving research into innovative treatments such as immunotherapy, nanotherapy, and tumor growth inhibitor therapy [18]. The geriatric population is usually not included in large clinical trials, and therefore the management of glioblastoma multiforme in this patient group is not fully described [16]. Surgical treatment options in this group are often severely limited due to the multi-morbidity of the patients and the possible range of postoperative complications [16]. On the other hand, radiotherapy at standard doses, compared to younger patients, is less tolerated by geriatric patients and is more likely to cause cognitive deficits [17]. Chemotherapy for glioblastoma multiforme is based on alkylating agents, the most relevant of which is Temozolomide. While the Temozolomide treatment prolongs patient survival, it is not always effective due to the molecular profile of the tumour. In addition, it causes haematological complications , which could be extremely dangerous for the elderly [17]. Often the only possible therapeutic interventions remain biopsy and palliative treatment [17]. According to analyses, patients with frailty syndrome do not benefit from aggressive treatment and symptomatic treatment is recommended in their case [16].

AUTHOR'S CONTRIBUTION

Conceptualization -K.N, A.K., Z.W., Formal Analysis-K.N., A.K., Z.W., Investigation- K.N., M.N.,A.K., Writing- through preparation- K.N.,M.N., P.D.,L.M., Writing- review and editing- A.K., Z.W., Supervision- Z.W. All authors have read and agreed with the published version of the manuscript.

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REFERENCES

1. Jongsiriyanyong S, Limpawattana P. Mild Cognitive Impairment in Clinical Practice: A Review Article. Am J Alzheimers Dis Other Demen. 2018 Dec;33(8):500-507. doi: 10.1177/1533317518791401. Epub 2018 Aug 1. PMID: 30068225; PMCID: PMC10852498.

2. Tangalos EG, Petersen RC. Mild Cognitive Impairment in Geriatrics. Clin Geriatr Med. 2018 Nov;34(4):563-589. doi: 10.1016/j.cger.2018.06.005. Epub 2018 Aug 21. PMID: 30336988.

3. Samuels M. H. (2008). Cognitive function in untreated hypothyroidism and hyperthyroidism. Curr. Opin. Endocrinol. Diabetes Obes. 15, 429–433. 10.1097/MED.0b013e32830eb84c

4. Markun S, Gravestock I, Jäger L, Rosemann T, Pichierri G, Burgstaller JM. Effects of Vitamin B12 Supplementation on Cognitive Function, Depressive Symptoms, and Fatigue: A Systematic Review, Meta-Analysis, and Meta-Regression. Nutrients. 2021 Mar 12;13(3):923. doi: 10.3390/nu13030923. PMID: 33809274; PMCID: PMC8000524.

5. Cabé N, Laniepce A, Ritz L, Lannuzel C, Boudehent C, Vabret F, Eustache F, Beaunieux H, Pitel AL. Troubles cognitifs dans l'alcoolodépendance : intérêt du dépistage dans l'optimisation des prises en charge [Cognitive impairments in alcohol dependence: From screening to treatment improvements]. Encephale. 2016 Feb;42(1):74-81. French. doi: 10.1016/j.encep.2015.12.012. Epub 2016 Jan 14. PMID: 26774623.

6. Wilbur J. Dementia: Dementia Types. FP Essent. 2023 Nov;534:7-11. PMID: 37976169.

7. World Health Organization (1993) The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research. WHO.

8. Arvanitakis Z, Shah RC, Bennett DA. Diagnosis and Management of Dementia: Review. JAMA. 2019 Oct 22;322(16):1589-1599. doi: 10.1001/jama.2019.4782. PMID: 31638686; PMCID: PMC7462122.

9. The use of neuroimaging techniques in the early and differential diagnosis of dementia. Mol Psychiatry. 2023 Oct;28(10):4084-4097. doi: 10.1038/s41380-023-02215-8. Epub 2023 Aug 22. PMID: 37608222; PMCID: PMC10827668.

10. Varesi A, Carrara A, Pires VG, Floris V, Pierella E, Savioli G, Prasad S, Esposito C, Ricevuti G, Chirumbolo S, Pascale A. Blood-Based Biomarkers for Alzheimer's Disease Diagnosis and Progression: An Overview. Cells. 2022 Apr 17;11(8):1367. doi: 10.3390/cells11081367. PMID: 35456047; PMCID: PMC9044750.

11. . Knapskog AB, Engedal K, Selbæk G, Øksengård AR. Alzheimers sykdom – diagnostikk og behandling [Alzheimer's disease – diagnosis and treatment]. Tidsskr Nor Laegeforen. 2021 Apr 29;141(7). Norwegian. doi: 10.4045/tidsskr.20.0919. PMID: 33950641.

12. Jack CR Jr, Bennett DA, Blennow K, Carrillo MC, Dunn B, Haeberlein SB, Holtzman DM, Jagust W, Jessen F, Karlawish J, Liu E, Molinuevo JL, Montine T, Phelps C, Rankin KP, Rowe CC, Scheltens P, Siemers E, Snyder HM, Sperling R; Contributors. NIA-AA Research Framework: Toward a biological definition of Alzheimer's disease. Alzheimers Dement. 2018 Apr;14(4):535-562. doi: 10.1016/j.jalz.2018.02.018. PMID: 29653606; PMCID: PMC5958625

13. Chahal M, Thiessen B, Mariano C. Treatment of Older Adult Patients with Glioblastoma: Moving towards the Inclusion of a Comprehensive Geriatric Assessment for Guiding Management. Curr Oncol. 2022 Jan 14;29(1):360-376. doi: 10.3390/curroncol29010032. PMID: 35049706; PMCID: PMC8774312

14. A, Liu G. Primary Brain Tumors in Adults: Diagnosis and Treatment. Am Fam Physician. 2016 Feb 1;93(3):211-7. PMID: 26926614

15. Shen C, Bao WM, Yang BJ, Xie R, Cao XY, Luan SH, Mao Y. Cognitive deficits in patients with brain tumor. Chin Med J (Engl). 2012 Jul;125(14):2610-7. PMID: 22882948. Wilbur J. Dementia: Dementia Types. FP Essent. 2023 Nov;534:7-11. PMID: 37976169

16. Cohen-Inbar O. Geriatric brain tumor management part II: Glioblastoma multiforme. J Clin Neurosci. 2019 Sep;67:1-4. doi: 10.1016/j.jocn.2019.05.064. Epub 2019 Jun 18. PMID: 31227406.

17. Chahal M, Thiessen B, Mariano C. Treatment of Older Adult Patients with Glioblastoma: Moving towards the Inclusion of a Comprehensive Geriatric Assessment for Guiding Management. Curr Oncol. 2022 Jan 14;29(1):360-376. doi: 10.3390/curroncol29010032. PMID: 35049706; PMCID: PMC8774312.

18. Wu W, Klockow JL, Zhang M, Lafortune F, Chang E, Jin L, Wu Y, Daldrup-Link HE. Glioblastoma multiforme (GBM): An overview of current therapies and mechanisms of resistance. Pharmacol Res. 2021 Sep;171:105780. doi: 10.1016/j.phrs.2021.105780. Epub 2021 Jul 21. PMID: 34302977; PMCID: PMC8384724.