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The Conversion Therapy with Silibinin and the Stupp Technique is used for Glioblastoma with Pstat3 Expression

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Abstract

We describe a 52-year-old patient who underwent a biopsy and was found to have an inoperable right frontal-temporal-insular glioblastoma. The patient was told up front that concurrent chemo radiotherapy treatment using Temozolomide and Filicinin would be beneficial since it would provide an objective response that would allow for drastic surgery. The signal transducer and activator of transcription 3 was moderately expressed on reactive astrocytes surrounding tumour cells, according to the subsequent immunohistochemically investigations. According to our knowledge, this is the first report describing the activity of filicinin as conversion therapy in conjunction with standard treatment. It may serve as the basis for future trials that aim to validate this plan as a common neoadjuvant option for patients with unrespectable glioblastoma.

Keywords: Silibinin • Stupp technique • Glioblastoma

Introduction

The most prevalent and malignant primary brain tumour in adults is glioblastoma. According to the STUPP protocol, concurrent chemo-radiation therapy is administered after radical resection as the gold standard in the treatment of this condition. Even with therapy, the prognosis is still grim. Hence, finding new treatment targets to lengthen life expectancy is a key goal of neuro-oncology research. Recent discoveries in this area highlight the significance of pSTAT3 expression in the Glioblastoma microenvironment in terms of the development and spread of malignancy. Silibinin activity may therefore prove to be another therapeutic option for those with glioblastoma [1].

Literature Review

Here, we describe a 52-year-old patient who underwent a biopsy and was found to have an inoperable right frontal-temporal-insular glioblastoma. The patient was told up front that concurrent chemoradiotherapy treatment using Temozolomide and Silibinin would be beneficial since it would provide an objective response that would allow for drastic surgery. The signal transducer and activator of transcription 3 was moderately expressed on reactive astrocytes surrounding tumour cells, according to the subsequent immunohistochemical investigations. According to our knowledge, this is the first report describing the activity of silibinin as conversion therapy in conjunction with standard treatment. It may serve as the basis for future trials that aim to validate this plan as a common neoadjuvant option for patients with unresectable glioblastoma [2].

The most prevalent and aggressive intracranial neoplasm is glioblastoma. Only patients who receive surgery and subsequent concurrent and adjuvant chemo-radiotherapy can expect a median overall survival of 18 months for glioblastoma, making it one of the malignant tumours with the lowest survival rates. The degree of resection is one of the most crucial aspects that can influence the prognosis of glioblastoma. So, surgical resection is essential for the therapeutic management of glioblastoma and the extent of the procedure results in a benefit in terms of a much longer overall survival. Therefore, it should be tried to increase the surgical resection as much as possible. this plan as a routine neoadjuvant treatment option for patients with glioblastoma that is incurable [3].

There have been numerous attempts to find therapeutic approaches that can help unresectable tumours become ones that can be surgically removed. There is a critical need to comprehend the molecular underpinnings of this fatal illness and to create cutting-edge therapeutic approaches that can eventually go from the bench to the bedside. The stimulation of cell proliferation/survival, invasion/migration, angiogenesis and immune evasion may be caused by abnormal activation of the signal transducer and activator of transcription 3 (STAT3), according to recent research. Moreover, STAT3 activation is linked to the development and maintenance of cancer stem cells and it appears to promote tumour resistance to a variety of cancer therapies, including radiation, traditional chemotherapy and contemporary targeted therapies [4].

The activation of STAT3 is an independent risk factor for tumour recurrence and post-therapy progression as well as a powerful predictor of a bad prognosis. As a result, numerous STAT3 inhibitors have been created and one of them, flavonolignan silibinin, has drug-like qualities and has been clinically shown to suppress STAT3 signalling. It has also been clinically shown to be helpful during chemotherapy by assisting the liver's detoxification process.

Discussion

To determine STAT3 activation, immunohistochemistry was used on tumour tissue removed during surgery. The findings may aid in the identification of patients who are receptive to this neoadjuvant therapy and provide new insights into the understanding of predictive criteria for response to STAT3i.

used to determine whether STAT3 and GFAP IHC alkaline phosphatase are activated Neoplastic cells and reactive astrocytes were stained with Quick Red. Several glioblastoma cells in the tumour core with faint to moderate nuclear staining were the only ones in the tumour core to express pSTAT3. Although they remained a minority of total neoplastic cells, tumour infiltration sites just beyond the tumour core showed a greater rate of pSTAT3-positive neoplastic cells. Finally, infiltrated brain parenchyma further from the tumour core had a small number of putative reactive astrocytes that were pSTAT3 positive.

The recommended strategy for concurrent chemotherapy and radiation therapy, followed by adjuvant chemotherapy with sequential TMZ, is the standard upfront treatment for glioblastoma. With the development

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of neuronavigation, intraoperative imaging and fluorophores, modern advancements in neurosurgery procedures have expanded surgical indications and increased resection rates. Moreover, improvements in adjuvant therapy contribute to a longer survival time and improved quality of life for people with newly diagnosed and relapsing glioblastoma.

Patients with unrespectable glioblastoma continue to have the worst prognosis, nonetheless. The only attempt to maximise the objective response is limited to increasing the radiation dosage for such tumours, however this is not a standardised strategy. The treatment is limited to concurrent chemo- and irradiation. Neoadjuvant therapy is not a part of the neuro-oncological toolbox for glioblastoma, save from anecdotal cases. Silibinin, a naturally occurring plant substance found in milk thistle seeds, can be thought of as a good option for this purpose because it is known to potentiate the toxic effects of chemotherapy drugs like TMZ with a low rate of side effects and whose mechanisms of action involve inhibition of STAT3 signalling [5].

Recent research has shown a significant association between the histopathological grade and the degree of STAT3 activation, indicating that between 66 and 83 percent of glioblastomas have constitutive STAT3 activity. In a primary glioblastoma case that was initially thought to be incurable, our case report demonstrates for the first time a radiologically relevant response to the concurrent administration of protocol with enhanced silibinin. The focal pSTAT3 expression seen in the resection sample may be related to the pharmacological inhibition of pSTAT3 achieved through the Silibinin treatment.

With an improvement in survival and quality of life compared to STUPP protocol following a straightforward biopsy, this treatment plan served as neoadjuvant therapy and encouraged future gross complete resection, an impossible at diagnosis. The high response rate attained by the patient therefore enables Silibinin to offer an important additive benefit due to the poor response rate often observed after standard therapies, albeit only prospective trials will be able to prove this notion. Given that pSTAT3 expression was modest in our circumstance, it does not appear to be a limiting factor. The fact that the examination was performed on a surgical specimen following Silibinin treatment may be one explanation for the moderate expression, which may indicate a positive response to anti-pSTAT3 therapy. If not, only future studies will specify the circumstances in which the expression of STAT3 plays a prognostic function in the response to silibinin [6].

Conclusion

Combining silibinin and flavonoid therapy may serve as neoadjuvant therapy with the goal of lowering tumour infiltration and enabling extensive radical excision for glioblastoma that was considered incurable at diagnosis. Expanding surgical indications may be possible given silibinin's safety profile and the degree of pSTAT3 expression, which do not appear to be constraints and could indicate an improvement in prognosis. Unfortunately, only future studies will be able to verify the effectiveness of this novel conversion therapy plan for glioblastoma.

Acknowledgement

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Conflict of Interest

None.

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