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Research Article

Molecular-based Implication of Epstein–Barr Virus in Gliomatous tissues from a group of Iraqi Patients: A PCR Study

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A B S T R A C T

Background: Gliomas account for around 60% of all primary brain tumors. Gliomas' aetiology is unknown, yet neurotropic viruses, including the oncogenic Epstein–Barr virus (EBV), have been implicated recently in the pathogenesis of various brain tumors, including primary CNS lymphoma and glioma.

Objective: To analyze the rate of neurotropic EBV infection in cerebral gliomas from a group of Iraqi patients with primary gliomas.

Patients and methods: This prospective study was conducted on 65 brain tissues, 40 of them from gliomas, while the rest 25 brain tissues showed unremarkable pathological changes (and were used as an apparently healthy control group). The polymerase chain reaction was done for detecting EBV-DNA sequences,, and the main clinicopathological features were recorded.

Results: 32% (8 out of 25) of the examined brain tissues were positive for EBV genome detection. The most infected brain tumor tissues with EBV-DNA are related to the age stratum (41-60 years), which accounted for 7.5 per percent, while the age strata of (2-20 years), (21-40 years), and (61-82 years) accounted for 2.5 per cent, 5 per cent, and 5 per cent,, respectively. The positive EBV-PCR results from brain tumour tissues show that accounted for 62.5% while females accounted for 37.5%. Positive EBV-PCR detection results from patients with various types of brain tumours were found as 20%, 8%, and 4% in glioblastoma, astrocytoma, and anaplastic astrocytoma, respectively.

Conclusion: Altogether, the apparently too high rate of EBV detection in the studied glioma samples can be considered, in respective parts, in brain tumour induction. We recommend the use of advanced molecular studies to be done to confirm the currently obtained data to unravel further the importance of this viral infection in such tumours.

Keywords: EBV, Brain tumor, Glioma, Astrocytoma, Anaplastic Astrocytoma, Glioblastoma, PCR

Introduction

Gliomas, which arise from glial cells, are the most prevalent primary central nervous system (CNS) cancers, accounting for approximately 60% of all primary CNS malignancies despite their rarity in the cancer spectrum [1]. Astrocytomas, oligodendrogliomas, and ependymomas are the three main types of gliomas. Glioblastoma multiforme (GBM) is the most common and aggressive glial tumour in adults, with a poor prognosis and low 5-year survival rates, according to the World Health Organisation's (WHO) classification of gliomas.

Glioma aetiology has been linked to genetic predisposition, chemical and radiation exposure, and, more recently, viral agents [2,3]. Gliomas have been shown to harbour a wide variety of infectious agents, including HPV 16/18, EBV, CMV, HHV8, HBV, HCV, HTLV1, JCV, BK virus, and SV40, all of which have been linked to primary brain carcinogenesis to varied degrees of certainty [4,5].

Epstein-Barr virus (EBV) is of special relevance since it has been linked to many distinct types of cancer, including lymphomas, nasopharyngeal and gastric carcinomas, thymomas, and central nervous system diseases and neuropathies [6,7]. Although EBV is extremely common (affecting nearly 90% of the world's population), it remains largely undetected by the immune system because it can establish asymptomatic latency in epithelial and certain immune cells by expressing specific viral latency genes [8-10]. Recent research has shown promise for a connection between EBV and gliogenesis, with some evidence suggesting the expression of EBV cellular receptor (CR2) on astrocytes [8, 11, 12]. This may shed light on the role of this virus in a variety of brain tumours, including glioblastoma, astrocytoma, and anaplastic astrocytoma.

The current research intends to examine EBV detection rates in a cohort of Iraqi patients with a variety of brain tumours, including glioblastoma, astrocytoma, and anaplastic astrocytoma. Using rigorous scientific methods, we want to shed light on the possible link between EBV infection and the development of these brain malignancies, therefore adding important new information to the growing body of knowledge in this area.

Patients and Methods

This study is designed as case-control study.

Clinical Specimens

A total of 40 gliomatous tissue samples were obtained from individuals aged (2 to 82) who had been diagnosed with brain tumours. There were 24 glioblastomas, 9 astrocytomas, and 7 anaplastic astrocytomas found. The control group consisted of 25 tissue specimens from autopsied brains of healthy adults aged 21 to 75. The criteria established by the WHO were used for the tumour categorisation process. Institutional review board and local ethics committee permission was obtained for the project.

Viral Genome Isolation and Identification

Extraction of Epstein-Barr virus nucleic acid from glioma patient brain tissue samples utilising the virus DNA/RNA Extraction Kit (Pathogene-Intron/Korea) for viral genome isolation and identification. The recovered viral genetic material was carefully monitored for purity and integrity, following strict criteria throughout the isolation process.

The PCR analysis for EBV

A PCR was performed using 500 nanograms of DNA extracted from freshly frozen gliomatous tissue, which coded for EBV. To prevent contamination during the PCR procedures for EBV, great caution was used.

After that, polymerase chain reaction (PCR) was used to verify the presence of the Epstein-Barr virus in each DNA sample (5 microlitres).

Primer sets were used in this study to detect the EBV forward 5'-CCAGTGCTGTGATCGAGCATCT-3', and the EBV reverse primer, 5'-CTGCTGACAAACTGCTGCATTC-3'.

The PCR conditions for EBV were as follows:

Step 1: 95ºC, 4 min.; Step 2: 95ºC, 30 sec.; Step 3: 60ºC, 1 min.; Step 4: 72ºC, 2 min. Cycles 40; Step 6: 72°C, 5 min.; Step 7: 4°C, forever.

Statistical analysis

The significance of the variables of the study was determined by statistical analysis. Chi-square analysis was utilized for this purpose. A relevant correlation between the variables was considered to exist if the p-value was less than 0.05, and all statistical analyses were conducted in SPSS (Version 23).

Results

Anatomy and physiology of the study groups

Age distribution

Patients with glioma brain tumors were included in the research, and their ages ranged from 2 to 82, with a mean of 48.2±13.2. The age distribution for the control group was (44.4+14.1) years. As can be seen in Table 1, the ages of glioma patients and controls were quite comparable. There was no statistically significant difference in age between the groups (p>0.05). These findings underscore the need of establishing appropriate age matching of participants in comparative studies.

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Table 1.The distribution of the age of research groups

Studied Group	No.	Mean Age (years)	Standard deviation	S. E	Minimum	Maximum
Brain Tumors Patients	40	48.2	13.2	2.5	2	82
Apparently Healthy Control (AHC)	25	44.4	14.4	2.9	21	75
Statistical Analysis	Non-significant (P > 0.05) = 0.09					

Sex Distribution

There were 23 men (57.5%) and 17 females (42.5%) in the glioma patient group, for a male-to-female ratio of 1.3:1. While there were only 11 (46%) women in contrast to 14 (56%) men who accounted for the majority of the control group Table 2 shows that there is a statistically significant ($p<0.05$) sex difference between glioma patients and healthy controls. This sex-based difference might shed light on what sets off gliomas.

Table 2.Distribution of study groups according to their sex

Sex	Glioma Tumors		Control		P-value
	No.	%	No.	%	
Male	23	57.5	14	56	0.004*
Female	17	42.5	11	44	
Total	40	100	25	100	

Age Distribution of Glioma Brain Tumor Patients

Patients diagnosed with glioma showed a complex pattern throughout the age spectrum. There were 7 (17.5%) cases of gliomas between the ages of 2 to 20 years, of them 4 (10%) were male and 3 (7.5%) were female. In addition, there were 8 (20%) people between the ages of 21 and 40 who were afflicted, including 5 (12.5%) men and 3 (7.5%) women (20% of the total). 7 (17.5%) men and 5 (12.5%) women, or 30%, were between the ages of 41 and 60. 7 (17.5%) men and 6 (15%) women, or 32.5% of the total, were diagnosed between the ages of 61 and 82.

Sex -Specific Distribution

Seven men and six women were diagnosed in the age category of 61 to 82, making this the age range with the greatest incidence of patients overall. This extensive breakdown provides useful insights into the demographic features of individuals afflicted by this disorder (Table 3), highlighting the necessity of examining age and gender intersections in glioma epidemiology. The development of diagnostic and treatment techniques for the glioma patient population that is unique to age and sex demographics requires such advanced analysis.

Table 3.Patients with brain tumors according to their age and sex

Age	Sex		Total
	Male	Female	
2-20 years	4 (10%)	3 (7.5%)	7 (17.5%)
21-40 years	5 (12.5%)	3 (7.5%)	8 (20%)
41-60 years	7 (17.5%)	5 (12.5%)	12 (30%)
61-82 years	7 (17.5%)	6(15%)	13 (32.5)
Total Glioma	23 (57.5%)	17 (42.5%)	40 (100%)

Distribution of studied glioma group according to their grading

Histopathological study of the glioma patients allowed for classification according to disease stage. The majority of gliomas, 55 percent, were classified as grade I, represented by 22 individuals 15 (37.5%) men and 7 (17.5%) women. 7 individuals (17.5%), 4 (10%) men and 3 (7.5%) women, were found to have grade II gliomas. 6 (15%) patients, 4 (10%) men and 2 (5%) women, had gliomas of grade III, accounting for 15% of the total. Grade IV gliomas, the most serious kind, were found in 12.5% of patients, or 5 total 3 (7.5%) males and 2 (5%) females; (Table 4).

There were statistically significant differences ($p\leq0.05$) between the four glioma grading groups. These statistically significant differences highlight the therapeutic importance of histopathological grading as a vital prognostic factor and assist in the development of individualized treatment options for glioma patients.

Table 4.Distribution of gliomas according to their grading

Grading of Gliomas	Gender		Total	P-value
	Male	Female		
I	15	7	22 (55%)	0.03
II	4	3	7 (17.5%)	
III	4	2	6 (15%)	
IV	3	2	5 (12.5%)	
Total brain tumors	23	17	40 (100%)	

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Frequency distribution of brain tumors according to their types and the gender of patients

The histopathological categorisation of brain tumours is shown in Table 5. A total of 24 tumours were glioblastoma, 14 in men and 10 in women, accounting for 60% of the cases. Furthermore, astrocytoma was identified in 22.5% of patients (9 tumours), with 5 men and 4 females affected. The incidence of anaplastic astrocytoma was 17.5% (7 tumours), with 4 males and 3 females affected. The various forms of brain tumours were shown to be statistically distinct from one another (P=0.03). Since glioblastoma, astrocytoma, and anaplastic astrocytoma all have very different pathology characteristics, this finding highlights the need for accurate categorisation.

Table 5.Frequency distribution of brain tumors according to their types and the sex of patients

Type of Glioma	Sex		Total	P-value
	Male	Female		
Glioblastoma	14	10	24 (60%)	0.03
Astrocytoma	5	4	9 (22.5%)	
Anaplastic astrocytoma	4	3	7 (17.5%)	
Total	23	17	40 (100%)	

Polymerase Chain Reaction results for Epstein–Barr virus detection in gliomatous tissue specimens

Using Viral DNA/RNA Extraction Kit to Extract Epstein–Barr Viral Nucleic Acid

Our analysis of 40 glioma tissue samples from our patient cohort revealed that viral genomes were present in 60% of cases (Figure 1). This research shows that there is a strong correlation between glioma histology and viral infections, suggesting that viruses may play a role in glioma initiation and progression.

Epstein-Barr virus nucleic acid was found in only 8% of instances (2 out of 25) in postmortem materials from the control group (Table 6). When compared to the control group, glioma tissues had a significantly higher prevalence of viral genomes (p = 0.03). This difference highlights the fact that glioma patients have different viral profiles than the general population, suggesting that viral elements may contribute to the aetiology of gliomas. More research into the exact functions of the viral components in glioma formation is warranted in light of these results, which may lead to new opportunities for targeted treatment therapies.

Table 6.Epstein–Barr Viral genome extraction from Brain tumors tissues

Epstein–Barr Viral Genome		Brain Tumors Patients Group	Apparently Healthy Control Group	p value
Positive	N	25	2	0.03
	%	62.5%	8%	
Negative	N	15	23	
	%	37.5%	92%	
Total	N	40	25	
	%	100%	100%	

Figure 1.Viral genome extraction from brain tumor tissues, electrophoresed in 1% agarose gel with a TBE of 1X, and run at 75 Volt for 45 minutes: the lanes 1-20 are positive samples

Epstein–Barr Viral Genome Detection Using Conventional PCR

According to Table 7 and Figure 2, 32% (8/25) of our glioma patients tested positive for EBV genome detection using PCR, whereas 68% (17/25) showed negative findings. This finding highlights the variability of viral involvement in glioma patients and the varying prevalence of EBV genomes within the patient group.

A much lower incidence was seen in the control group, with just 4% (1 of 25) of cases being positive for EBV genome detection. When compared to a control group, glioma patients showed a statistically significant difference in these results (p = 0.04). This striking difference provides further evidence that EBV may play a role in glioma etiology by highlighting the much higher incidence of EBV genome present in glioma patients compared to control peers. These findings shed light on the particular viral elements that may play a critical role in glioma formation and give vital insights into the disease's molecular complexity. The role of the EBV in the development of gliomas is complex, and further research is needed to understand the underlying processes and clinical consequences.

EBV-PCR Results in Patients with Brain Tumors by Age Strata

DNA-EBV prevalence in tissues from various types of brain tumours was shown to exhibit age-related differences. The greatest infection incidence, 7.5% (3 out of 12 tissues), was seen in people between the ages of 41 and 60. Rates

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of infection were 5%, 5%, and 5% among those aged 61–82 compared to 2.5%, 5%, and 5% among those aged 2–20. These results demonstrate a significant age-related variation in the susceptibility to EBV infection among the population under study.

There are statistically significant variations in EBV infection rates across different age groups, as shown in Table 8 at the P0.05 level of significance. The significance of age in determining the presence of EBV in brain tumour tissues is highlighted by this finding. There may be age-related susceptibility or biological variables that contribute to EBV infection in those between the ages of 41 and 60, as shown by the increased prevalence in this age group.

Table 7.Positive Conventional - PCR results of Epstein–Barr Viral Genome Detection in brain tumors tissue samples

Conventional - PCR Results of EBV Genome	Glioma Tissue Samples Group	Apparently Healthy Control Group
Positive	8 (32%)	1 (4%)
Negative	17 (68%)	1(96%)
Total	25 (100%)	2 (100%)

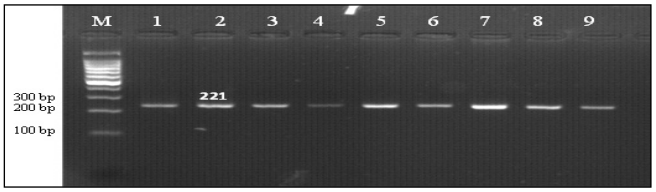

Figure 2.The detection pattern of the electrophoresis for EBV DNA (221bp) in tissues from glioma after using PCR run. Lanes 1-8 refer to EBV DNA; the electrophoresis conditions were: 5 µl in each well, 1% concentration agarose, 75 Volt, 20 mA for 1h, staining by safe red solution

EBV results in brain tumor tissues according to the gender of the patients

Table (9) shows the percentage of brain tumours tissues that have positive EBV -PCR results based on the gender of patients, with males accounting for 62.5% (5 out of 8 cases) and females accounting for 37.5% (3 out of 8 cases). In the brain tumors group, statistical analysis revealed significant differences in gender in relation to positive EBV- PCR (P<0.05).

Association of EBV Infection with types of brain tumors

Table (10) shows positive EBV-PCR detection results from patients with various types of brain tumours, where 20%, 8%, and 4% glioblastoma, astrocytoma, and anaplastic astrocytoma, respectively, showed positive PCR results for EBV detection. The statistical analysis regarding the EBV-positive showed significant differences between the different brain tumours types (p<0.05) (Table 4-10).

Table 9.Positive EBV percentage in glioma patients based on their gender

Glioma patients	EBV- Infection Positive (%)
Men	5 (62.5%)
Women	3 (37.5%)
Statistical analysis	(P<0.05) = 0.03

Table 10.Frequency of brain tumor types in relation to the EBV-positive PCR results

Brain Tumor Types	Brain Cancer			P-value
	Total Brain tumors	EBV-Negative	EBV-Positive	
Glioblastoma	24	19	5 (20%)	0.03
Astrocytoma	9	7	2 (8%)	
Anaplastic astrocytoma	7	6	1 (4%)	
Total	40	32	8(32%)	

Table 8.Frequency of EBV-PCR results among brain tumor tissues according to age strata

	Years	EBV-PCR Results			P value
		No.	Positive	Negative	
Age Stratum	2-20	7	1	6	Anova test P=0.03 (P<0.05)
		17.5%	2.5%	15%	
	21-40	8	2	6	
		20%	5%	15%	
	41-60	12	3	9	
		30%	7.5%	22.5%	
	61-82	13	2	11	
		32.5%	5%	27.5%	
Total %100		40	8	32	
		32%	68%		

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Discussion

Brain tumors account for 2% of all tumours in the body, yet their aetiology is still mostly unknown. The most common kinds of these tumours are gliomas and meningiomas [13]. Epstein-Barr Virus (EBV) identification in glioma tissues was the focus of our research because of the established viral connections to brain carcinogenesis [12].

Since its identification in 1964 as the first human oncovirus, EBV has been linked to many types of cancer arising in lymphocytes, epithelial cells, and mesenchymal cells [14]. EBV was detected in 60% of all brain tumours patients by PCR in our investigation, which is much higher than the 8% incidence seen in postmortem control material [13]. This frequency is consistent with reports from other international investigations that have shown EBV to be significantly present in gliomas.

Several different molecular methods have been used in the past to identify EBV in malignant brain tumors. Mixed infections have been seen in patients with glioblastoma multiforme (GBM) when immunohistochemistry and in situ hybridisation methods were employed to identify EBV (20). Rates of EBV identification in various tumours types have also been reported by research using PCR methods. The frequency of glioblastoma multiforme was 44.4%, whereas that of astrocytomas was only 30% [15].

Although PCR and immunohistochemistry are useful, they have significant limits due to their specificity. In order to investigate viral sequences in gliomas without bias, next-generation sequencing (NGS) has emerged as a viable approach [16-20]. The use of NGS has the potential to provide hidden information about the possible variety of EBV strains and their interactions with host genomic landscapes.

Notably, investigations conducted in various parts of the world found conflicting outcomes. According to one study, EBV was found in 14.7% of gliomas. This percentage was greatest in low-grade gliomas. However, research out of Slovenia has shown that EBV-positive is more common in high-grade gliomas [21]. These discrepancies may be attributable to geographical variations, genetic predispositions, or even methodological differences.

Our glioma samples are noteworthy for the high prevalence of EBV identification. Although EBV is common, its significant presence in brain tumours prompts researchers to speculate about its possible involvement in glioma production. Validation of these results and elucidation of the complex relationships between EBV and gliomas need further advanced molecular research. The hope for better results for patients fighting these terribletumours rests on our understanding of the underlying processes, which may lead to innovative treatment strategies.

Conclusion

In conclusion, our research contributes new information on the frequency of EBV in glioma tissues. The high incidence of identification highlights the need for further investigation, encouraging researchers to investigate further the role of EBV in the genesis of gliomas. The elusive nature of gliomas may be better understood and novel targeted treatments developed with the use of cutting-edge molecular methods, including next-generation sequencing (NGS).

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Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: None

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