



Metabolic Profiling in Diffuse Intrinsic Pontine Glioma: Imaging and Molecular Analysis

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DESCRIPTION

Diffuse Intrinsic Pontine Glioma (DIPG) is a highly aggressive and fatal pediatric brainstem tumor, primarily affecting children between the ages of 5 and 10. Unlike other brain tumors, DIPG is inoperable due to its critical location within the brainstem, which controls essential functions such as breathing and heart rate. Traditional therapies, including radiation and chemotherapy, provides limited efficacy. Consequently, understanding the unique metabolic profile of DIPG is potential for developing new therapeutic strategies. Measuring tumor metabolism can provide insights into the tumor's biology, identify potential metabolic vulnerabilities, and guide the development of targeted treatments.

Importance of tumor metabolism in DIPG

Tumor metabolism refers to the altered biochemical processes within cancer cells that support their rapid growth and survival. In DIPG, as in other cancers, metabolic reprogramming is a symbolic feature. Cancer cells often exhibit increased glucose uptake and glycolysis, a phenomenon known as the Warburg effect, even in the presence of sufficient oxygen. Additionally, these cells may rely on alternative substrates such as glutamine and fatty acids to sustain their proliferation and resist apoptosis.

Methods for measuring tumor metabolism

Several techniques can be employed to measure tumor metabolism in DIPG, each providing different insights into the metabolic state of the tumor.

Positron Emission Tomography (PET) imaging is a non-invasive technique that can quantify the metabolic activity of tumors *in vivo*. 18F-Fluorodeoxyglucose PET (FDG-PET) is the most common PET tracer, FDG, is a glucose analog. Cancer cells, including those in DIPG, take up FDG at higher rates due to their increased glucose metabolism. FDG-PET can thus highlight regions of high metabolic activity within the tumor,

providing a functional imaging perspective. In addition to FDG, other tracers like 18F-Fluorothymidine (FLT) for cellular proliferation and 11C-acetate for lipid metabolism can be used to study different aspects of DIPG metabolism.

Magnetic Resonance Spectroscopy (MRS) is an extension of MRI that provides metabolic information about tissues by detecting the presence of specific metabolites. Proton MRS (1H-MRS) technique can measure the concentrations of metabolites such as lactate, choline, and N-acetyl aspartate. Increased lactate levels, indicative of enhanced glycolysis, are often observed in high-grade gliomas, including DIPG. Phosphorus MRS (31P-MRS) can assess the energy status of cells by measuring high-energy phosphates like ATP and phosphocreatine.

Metabolomic profiling in advanced analytical techniques such as Mass Spectrometry (MS) and Nuclear Magnetic Resonance (NMR) spectroscopy can provide comprehensive metabolic profiles of tumor tissues. Metabolomic profiling can be performed on biopsied tumor tissues, Cerebrospinal Fluid (CSF), or blood samples. These analyses can identify altered metabolic pathways and potential biomarkers for DIPG.

Biopsy and histopathological analysis are used for obtaining biopsy samples from DIPG is challenging, recent advances in surgical techniques have made it more feasible. Histopathological examination combined with immunohistochemistry can reveal the expression of metabolic enzymes and transporters, providing insights into the metabolic adaptations of DIPG cells.

Metabolic characteristics of DIPG

Recent studies have begun to understand the metabolic landscape of DIPG, highlighting several key features such as enhanced glycolysis, glutamine metabolism, and lipid metabolism. DIPG cells exhibit high rates of glycolysis, reflected by increased glucose uptake and lactate production. This dependence on glycolysis, even under aerobic conditions, supports rapid ATP generation and provides intermediates for biosynthetic processes. DIPG cells also show dependency on

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glutamine, an amino acid that serves as a carbon and nitrogen source for biosynthesis and supports the TriCarboxylic Acid (TCA) cycle. Alterations in lipid metabolism, including increased fatty acid synthesis and beta-oxidation, have been observed in DIPG. These changes support membrane biosynthesis and energy production.

Implications for therapy

Understanding the metabolic dependencies of DIPG facilitates for therapeutic intervention.

Targeting glycolysis inhibitors of key glycolytic enzymes, such as hexokinase and lactate dehydrogenase, can potentially disrupt the energy supply of DIPG cells.

Glutamine inhibition drugs that inhibit glutamine uptake or metabolism, such as glutaminase inhibitors, may starve DIPG cells of critical nutrients.

Lipid metabolism inhibitors targeting enzymes involved in fatty acid synthesis or oxidation could impair the ability of DIPG cells to generate essential lipids and energy.

Measuring tumor metabolism in DIPG provides valuable insights into the unique biochemical adaptations of this aggressive cancer. Techniques such as PET imaging, MRS, and metabolomic profiling are essential tools in characterizing the metabolic phenotype of DIPG. By understanding the metabolic vulnerabilities of DIPG, researchers can develop targeted therapies aimed at disrupting the metabolic networks that sustain tumor growth and survival. This approach holds potential for improving outcomes in children afflicted with this devastating disease.