

Catalytic Nanomedicine: Ganglioglioma Treated with Bionanocatalysts in Mexico

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ABSTRACT

Gangliogliomas are rare, low-grade neuroglial tumours predominantly affecting children and adolescents, frequently presenting with drug-resistant epilepsy. Despite surgical resection and adjuvant therapies, recurrence is common, especially when total resection is not feasible. This case study reports on a 12-year-old female with a recurrent ganglioglioma treated with conventional therapies—surgery, chemotherapy, and radiotherapy—followed by a novel nanomedicine intervention using platinum-based bionanocatalysts. These bionanocatalysts (Pt/TiO₂), synthesized via sol-gel methods, were engineered for high selectivity and catalytic activity within malignant cells. Comprehensive characterization through TEM, SEM, EDS, FTIR, Raman spectroscopy, and XRD confirmed their nanoscale structure, anatase crystalline phase, and high surface area (515 m²/g). The patient received intratumoral implantation of these nanoparticles during her final surgical procedure. Subsequent follow-up over two years revealed no recurrence of tumour or neurological sequelae. The catalytic mechanism involves radical generation (O₂•⁻, HO₂•, OH•), facilitating oxidative degradation of cancerous tissue. Histopathology confirmed the biphasic nature of the ganglioglioma, and imaging studies tracked tumour progression and response to treatment. This report underscores the potential of heterogeneous catalytic nanomedicine as a complementary approach to conventional treatments for refractory brain tumours. The case demonstrates that bionanocatalysts offer targeted tumour destruction with minimal systemic toxicity, presenting a promising avenue for future neuro-oncological therapies.

Keywords: Bionanocatalyst; Ganglioglioma; Platinum; Catalytic Nanomedicine; Neuro-oncology

INTRODUCTION

Gangliogliomas are slow-growing tumors of neuroglial origin, histologically characterized by biphasic components comprising dysplastic neurons and neoplastic glial cells.^[1] These tumors represent the second most common cause of drug-resistant focal epilepsy, with a peak incidence during the first two decades of life.^[2] Predominantly located in the supratentorial region—most commonly the temporal lobe (approximately 68% of

cases)—gangliogliomas typically present with chronic, pharmaco-resistant epilepsy and focal neurological deficits.^[3] According to the World Health Organization (WHO) classification of central nervous system (CNS) tumors, gangliogliomas are classified as low-grade (Grade I–II);^[4] however, several clinical and pathological features are associated with poorer outcomes. These include tumor location near critical structures, size, and early age at presentation, particularly when involving regions proximal to the brainstem.^[5]

Histologically, gangliogliomas are generally well-demarcated from the adjacent brain parenchyma.^[6] The glial component often contains calcifications, extensive perivascular or intratumoral lymphoid infiltrates, and a pronounced capillary network.^[7] While no single mutation defines gangliogliomas, molecular studies have identified recurrent alterations such as BRAF V600E mutations—where valine is substituted by glutamic acid at position 600—and MAPK pathway fusions.^[8] These oncogenic drivers are shared among several neuroepithelial tumors, including pilocytic astrocytoma and dysembryoplastic neuroepithelial tumor.^[9]

Complete surgical resection remains the treatment of choice, offering the best prognosis and reduced morbidity.^[10] However, partial resection may be unavoidable in eloquent or high-risk regions, increasing the risk of recurrence and necessitating long-term surveillance.^[11] Adjuvant chemotherapy and radiotherapy are typically reserved for progressive or malignant cases.^[12] In a retrospective study by Jallo et al. (2004) involving 56 pediatric patients, a 5-year progression-free survival rate of 67% was reported following surgical intervention.^[13]

Recent advances in oncology have led to the development of novel chemotherapeutic strategies that offer high selectivity and reduced systemic toxicity.^[14] Nanomedicine—a field at the intersection of nanotechnology and medicine—offers promising approaches to cancer therapy, particularly through the controlled, localized delivery of chemotherapeutic agents from nanoparticle-based carriers.^[15] Examples of such nanotechnology-enabled therapies are summarized in [Table 1](#).

Table 1: Approved nanotechnology-based cancer drug therapies (EMA: European Medicines Agency; FDA: US Food and Drug Administration)[†]

Approval (year)	Product	Company	Nanoparticles material	Drug/Mechanism	Indication
EMA (2019)	Hensificar (NBTXR3)	Nanobiotix	Hafnium oxide nanoparticle	Radiotherapy	Locally advanced soft tissue sarcoma (STS)
EMA (2019)	Pazenir	Ratiopharm GmbH	Albumin bound to nanoparticles	Paclitaxel	Metastatic breast cancer, metastatic pancreatic adenocarcinoma, non-small cell lung cancer
FDA (2017) EMA (2018)	Vyxeos	Celator/Jazz Pharma	Liposome	Cytarabine/daunorubicin	Acute myeloid leukemia
FDA (2015)	Onivyde	Merrimack Pharmacy	Liposome	Irinotecan	Pancreatic cancer, colorectal cancer

European Medicines Agency (2010, 2013)	Nanoterm	MagForce Nanotechnologies AG	Iron oxide nanoparticles	Thermal ablation with magnetic field	Glioblastoma, prostate and pancreatic cancer
FDA (2012)	Marquibo	Talon Therapeutics/ Spectrum Pharmaceuticals	Liposome	Vincristine	Acute lymphoblastic leukemia
European Medicines Agency (2009)	Mepact	Takeda Pharmaceuticals	Liposome	Mifamurtide MTP-PE	Osteosarcoma
South Korea (2007)	Genexol-PM	Samyang Biopharmaceutical Products	PEG-PLA polymeric micelle	Paclitaxel	Breast, lung, and ovarian cancer
FDA (1994, 2006)	Oncaspar	Enzon-Sigma-tau	Polymer-protein conjugate	Pegaspargase/ L-asparaginase	Acute lymphoblastic leukemia
FDA (2005)	Abraxane	Abraxis/Celgene	Albumin bound to nanoparticles	Paclitaxel	Breast and pancreatic cancer, non-small cell lung cancer
FDA (1999)	Citrate depot	Pacira Pharmaceuticals	Liposome	Cytarabine	Neoplastic meningitis
FDA (1996)	DaunoXome	Gilead Sciences	Liposome	Daunorubicine	Kaposi's sarcoma
FDA (1995, 1999, 2007), EMA (1996, 2000), Taiwan (1998)	Doxil, Caelyx, Myocet y Lipo-Dox	Johnson & Johnson, Schering-Plough, Teva UK and TTY Biopharm	Liposome	Doxorubicine	Metastatic breast cancer, ovarian cancer, Kaposi's sarcoma, multiple myeloma

† Retrieved from Wicki et al. (2015)^[16], and Rodríguez et al. (2022).^[17]

Controlled-release nanoparticles offer a significant therapeutic advantage by enabling the localized delivery of active agents directly to the tumor site, thereby minimizing systemic exposure and reducing associated side effects.^[18] However, unlike conventional nanopharmaceuticals associated with controlled-drug release, bionanocatalysts exert their therapeutic effect through catalytic mechanisms that directly target the DNA of malignant cells.^[19] These agents are composed of ultrananometric structures synthesized via a sol-gel process, consisting of titania (TiO₂) coated with 1% platinum (Pt).

Bionanocatalysts demonstrate high tumor selectivity (>90%), attributed to their receptor-mediated endocytic uptake that is specific to malignant cells.^[20] This specificity arises from the differential expression of surface receptors between healthy and cancerous tissues, allowing the coated Pt/TiO₂ (1%) bionanocatalysts to selectively penetrate and act within the tumor microenvironment.

This study presents a clinical case involving a pediatric patient diagnosed with ganglioglioma, who has been under continuous follow-up at the Federico Gómez Children's Hospital of Mexico since the age of 12. The patient underwent multiple therapeutic interventions, including surgery, chemotherapy, radiotherapy, and subsequently, a combined approach incorporating novel catalytic therapy as part of a Phase 1 clinical trial. Following partial tumor resection, the patient received treatment with Pt/TiO₂ bionanocatalysts. The

physicochemical properties of the materials were rigorously characterized using transmission electron microscopy (TEM), scanning electron microscopy (SEM), energy-dispersive X-ray spectroscopy (EDS), Fourier-transform infrared spectroscopy (FTIR), and X-ray diffraction (XRD), confirming their structural and compositional integrity for biomedical application.

MATERIALS AND METHODS

Surface-Coated Bionanocatalysts Synthesis

The Pt/TiO₂ bionanocatalysts were synthesized via a sol-gel method, which involves a series of hydrolysis and condensation reactions of metal alkoxides under tightly controlled conditions.^[21] Key synthesis parameters—including pH, solvent composition, precursor concentration, and water content—were optimized to ensure homogeneity and reproducibility. The sol-gel process facilitates the formation of a catalytic network through the nucleation and aggregation of nanometric sol particles; continuous stirring was employed to promote uniform dispersion and interaction of suspended species throughout the reaction medium. In brief, a titanium precursor hydrolysed with a mixture of water and acetylacetone. Once it gelled, the compound was dried and the stoichiometric amount of a platinum precursor was dissolved in acetylacetonate to achieve a final concentration of 1% platinum and mixed with the titania gel. Functionalizing agents were also introduced during the sol-gel process to enhance biocompatibility and facilitate receptor-mediated targeting, thereby mimicking biological interactions relevant to tumour cell uptake. Following synthesis, the gel was subjected to vacuum drying at 70 °C to remove residual solvents and promote structural stabilization. The resulting bionanocatalysts were then prepared for subsequent physicochemical characterization and biological evaluation.

Physicochemical Characterization

Transmission electron microscopy (TEM). The sample was transferred under high vacuum protection to the TEM. Images were obtained using a JEOL 2010, operated at 120 kV, equipped with an energy-dispersive spectroscopic (EDS) microanalysis system (OXFORD) and a Mega Vision (III) CCD camera for elemental analysis. *Scanning electron microscopy (SEM).* The study was carried out to investigate the porous and nanostructured morphology of the NPt/TiO₂ samples. For this purpose, a Hitachi-4800 field emission microscope operated at 3 kV was used. Cryogenic conditions were maintained with a constant flow of liquid nitrogen through the SEM analysis chamber. The aqueous phase on the upper surface of the frozen sample bubble was removed by maintaining a temperature gradient (10°C) for 5 minutes between the sample and the anti-contamination plate. Subsequently, the sample was transferred under high vacuum to the microscope chamber at an accelerating voltage of 2 kV and a working distance of 5 to 6 nm. *Fourier transform infrared spectroscopy (FTIR).* The spectra were recorded at room temperature using KBr, with a Shimadzu model FTIR IR Affinity-1 spectrometer, covering the range of 4000-400 cm⁻¹. *Raman spectroscopy.* This study was carried out on a HORIBA Jobin Yvon HR800 UV spectrometer using an excitation line of 514.5 nm and a 600 g mm⁻¹ grating. All spectroscopy was taken with an exposure time of 1 s. *X-ray diffraction (XRD).* It was performed on a Siemens D-5000 diffractometer with CuK α radiation and a monochromatic graphite beam. The intensity was measured along 2 θ between 18 and 130° with a step size of 0.02° and a measurement time of 2 seconds per step. The atomic scattering factors of neutral atoms reported in the international tables for X-ray crystallography were used in the analysis. The spectra were refined using the Rietveld technique and the DBWS-9006PC program.

BET analysis. The specific surface area of the bionanocatalysts was analysed by the BET method, starting with the nitrogen adsorption isotherms at 77 K in Micromeritics ASAP 2000 automatic equipment. The mean pore size distribution was calculated from the BET isotherm by applying the BJH method before chemisorption, and the samples were reduced to 400° C in a hydrogen flow for 4 hours.

Histological Analysis

Haematoxylin and Eosin staining were used. They are techniques that use two types of dyes to differentiate a cell's structures and characteristics under a light microscope.^[22] Haematoxylin stains ribosomes, chromatin within the nucleus, and other structures in a deep blue-violet. Eosin stains the cytoplasm, collagen, connective tissue, and other structures surrounding and supporting the cell a pinkish orange.

CLINICAL CASE

A 12-year-old female with a history of chronic epilepsy presented to her local healthcare center with a six-month history of progressively worsening focal seizures. According to her mother, the episodes were characterized by sudden jerking of the right upper limb, lasting 1–2 minutes, and followed by postictal confusion and fatigue. Initially occurring 2–3 times per week, the seizures increased in frequency and were accompanied by headaches and vomiting. In the two weeks preceding presentation, she developed daily morning headaches and mild nausea, which escalated to intolerable levels. Due to the deterioration in her condition, she was referred to the tertiary care facility at Federico Gómez Children's Hospital of Mexico City. On arrival at the emergency department, physical examination revealed asthenia, adynamia, mild right upper limb paresis, hyperreflexia, and ipsilateral dysmetria. She was stabilized with anticonvulsants, analgesics, and antiemetics. Magnetic resonance imaging (MRI) and computed tomography (CT) revealed a lesion in the left occipital region, suggestive of a diffuse glioma. She was admitted for surgical intervention and monitoring.

An intraoperative biopsy confirmed the diagnosis of ganglioglioma. A second procedure was performed to achieve partial tumor resection, after which she was discharged with scheduled follow-up in pediatric neuro-oncology. The patient received adjuvant chemotherapy with carboplatin and fractionated radiotherapy (25 Gy in 10 sessions). Over the next three years, two additional surgeries were performed, along with further chemotherapy and radiotherapy; however, her condition continued to deteriorate.

Due to disease progression, she was referred to palliative care. As a final therapeutic option, she and her mother consented to an experimental treatment with Pt-functionalized TiO₂ (NPt) bionanocatalysts. The nanoparticles were surgically implanted into the tumor bed. Following this intervention, the patient was monitored over a two-year period, during which she remained clinically stable, with no evidence of neurological deficits or tumor recurrence.

RESULTS AND DISCUSSION

Physicochemical Properties of Bionanocatalysts

Particle and conglomerate size. Figure 1A and B show micrographs of 1-3 nm aggregated NPt nanoparticles. SEM images show spherical agglomerates with an anatase crystal structure determined using EDS. The pore

size distribution in NPt was also confirmed. Figure 1C and D show the minimal particle size (1-3 nm) due to the established chemical and physical conditions.

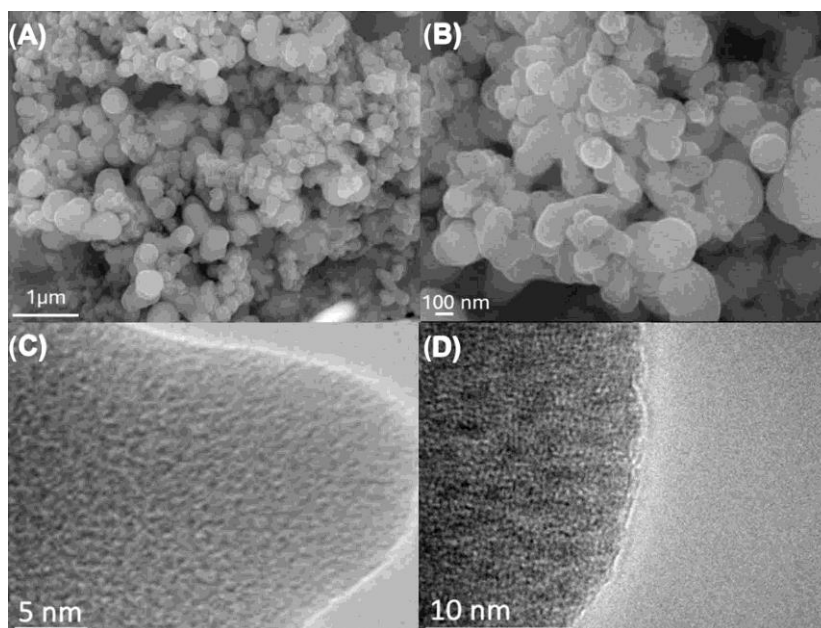


Figure 1: (A, B) Scanning Electron Micrographs and (C, D) Transmission Electron Micrographs of bionanocatalysts at different magnifications. (A) and (B) show the presence of agglomerated nanoparticles in which each agglomerate has an approximate diameter of 0.5 to 1 μm , and individual particles range from 1-5 nm. In (C), it can be observed that TiO_2 has an anatase crystal structure, while platinum copies the lattice structure of titania.

Acid Sites, Functional Groups, and crystalline structure. The surface acidity of the NPt sample was evaluated using Fourier-transform infrared (FTIR) spectroscopy with pyridine adsorption. The resulting spectra exhibited distinct vibrational bands corresponding to both Brønsted and Lewis acid sites.^[23] A characteristic band at 1543 cm^{-1} was attributed to pyridine coordinated to Brønsted acid sites (Figure 2A). Peaks observed at 1576 , 1489 , and 1444 cm^{-1} were assigned to pyridine interacting with Lewis acid sites. Additionally, a band at 1604 cm^{-1} was associated with pyridine hydrogen-bonded to surface hydroxyl groups, indicating the presence of adsorbed water and surface hydroxyl functionalities contributing to the overall surface reactivity.

FTIR analysis of the NPt sample (Figure 2B) revealed a broad absorption band around 3431 cm^{-1} and a distinct band at 1632 cm^{-1} , indicative of adsorbed water and surface hydroxyl groups—features commonly observed in freshly synthesized or atmospherically exposed TiO_2 . These surface hydroxyls are known to enhance the reactivity of titania, thereby improving its photocatalytic performance. Additional peaks at 606 cm^{-1} and 485 cm^{-1} correspond to Ti-O bond vibrations characteristic of the anatase crystalline phase.^[24] The spectral region between 2919 and 2700 cm^{-1} exhibited bands attributable to C-N and N-H stretching vibrations, while weak bands and shoulders between 1500 and 1000 cm^{-1} were associated with C-C scissoring, C-H bending, and C-C stretching modes—arising from organic functional groups introduced during surface modification.

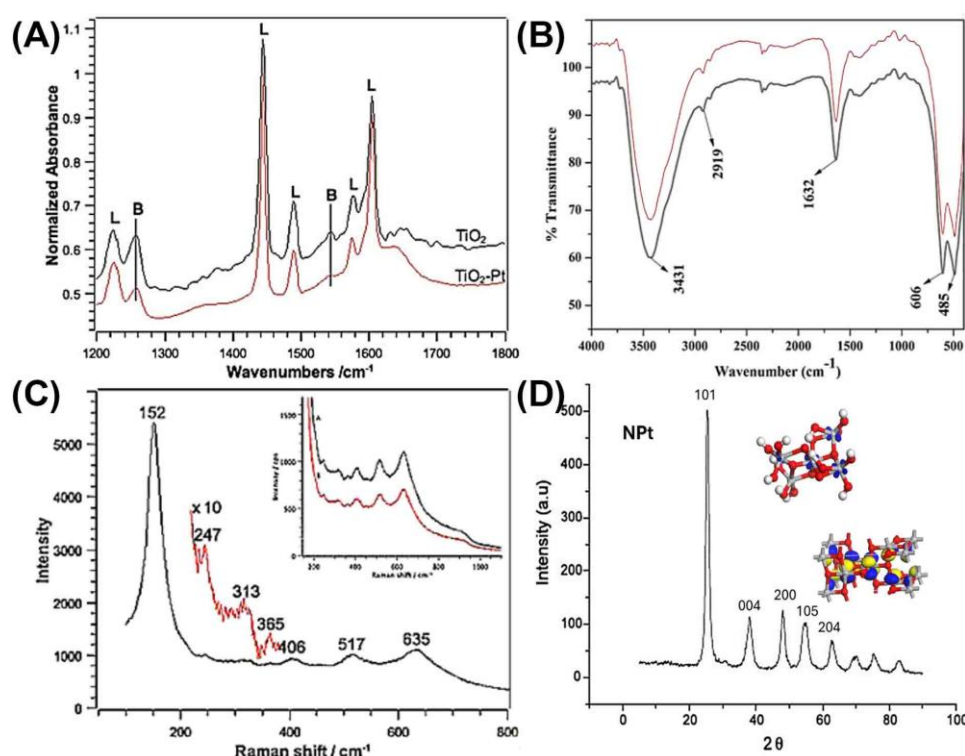


Figure 2: (A) FTIR with pyridine adsorption to study Brönsted and Lewis acidity. Black line Titania Reference, red line NPt. (B) FTIR spectroscopy of Titania Sol-Gel of NPt. (C) Raman spectroscopy of the NPt bionanocatalyst (black) and the titania support (red). (D) X-ray diffraction of the NPt bionanocatalyst: the titania support remains anatase even after adding platinum in situ.

Raman spectroscopy further confirmed the presence of anatase TiO₂ through the identification of five Raman-active optical phonon modes at 152 cm⁻¹ (*Eg*), 247 cm⁻¹ (*Eg*), 313 cm⁻¹ (*B1g*), 406 cm⁻¹ (unresolved *A1g* + *B1g*), and 635 cm⁻¹ (*Eg*) (Figure 2C).^[22] The *Eg* mode in the 143–152 cm⁻¹ range has been shown to vary with stoichiometric deviations in anatase; thus, the measured peak position and full width at half maximum (FWHM) were used to assess the stoichiometry of the anatase phase in both TiO₂ and NPt samples. Functionally, heterogeneous catalytic nanomedicine employing NPt bionanocatalysts induces tumor degradation via catalytic generation of reactive oxygen species (ROS), including O₂•⁻, HO₂•, and OH• radicals. These highly reactive intermediates mediate the oxidative and reductive breakdown of tumor tissues, yielding end products such as CO₂(g), H₂O(g), N₂(g), K₂SO₄, and Na₃PO₄, thereby facilitating localized and selective tumor ablation.

In the XRD pattern of NPt (Figure 2D), diffraction peaks corresponding to the anatase phase are seen at 2θ = 25°, 30°, 37°, 47°, 53°, 62°, 69°, and 75°.^[25]

Porous structure. Figure 3 shows the adsorption-desorption isotherm for NPT, as well as the pore size distribution. BET analysis of the isotherms showed that the sample exhibited a specific surface area (*S*_{BET}) of 515 m²/g and an average pore diameter (*d*_p) of 2.40 nm, with an average pore volume (*V*_p) of 0.3080 cm³/g. According to the IUPAC classification, the values obtained for the Pt/TiO₂ sample are characteristic of

mesoporous materials, approaching the boundary with microporous structures.^[26] Among the phases analyzed, the anatase phase exhibited the lowest specific surface area.

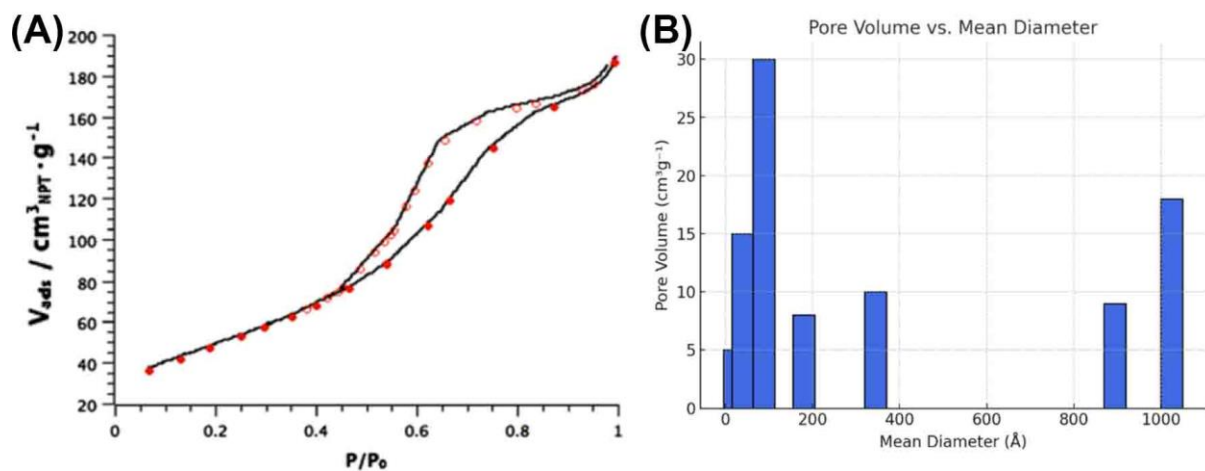


Figure 3: (A) NPt adsorption isotherm, **(B)** NPt pore size distribution.

CASE REPORT: MAGNETIC RESONANCE IMAGING

The initial magnetic resonance imaging (MRI) was performed in November 2012, revealing an intra-axial lesion associated with gliosis in the left inferior parietal lobe, specifically within the angular gyrus. The lesion measured approximately $3.5 \times 3.0 \times 2.5$ cm and exhibited a heterogeneous mixed-density appearance, comprising both solid and cystic components. On T1-weighted axial imaging (**Figure 4A**), a hyperintense signal was observed, attributable to the presence of blood products. Sagittal T1-weighted sequences (**Figure 4B**) demonstrated a mixed solid-cystic mass in the left parietal lobe. T2 FLAIR imaging (**Figure 4C**) showed peripheral vasogenic edema surrounding the lesion, evident as a hypointense halo, suggesting mass effect. Additionally, small peripheral calcifications were suspected based on the hyperintense border observed. Following histopathological confirmation of the diagnosis, a surgical intervention was undertaken, with partial resection of the tumour to reduce mass effect and obtain definitive tissue samples.

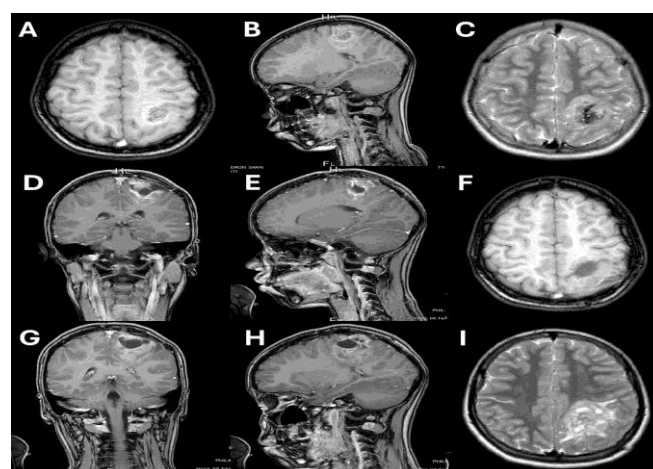


Figure 4: Contrast-enhanced MRI. November 2012: (A) Axial slice in T1-weighted sequence, **(B)** Sagittal slice in T1W sequence, **(C)** Axial slice in T2 FLAIR sequence. September 2013: **(D)** Coronal slice in T1-weighted

sequence, **(E)** Sagittal slice in T1-weighted sequence, **(F)** Axial slice in T2-weighted sequence. March 2015: **(G)** Coronal slice in T1-weighted sequence, **(H)** Sagittal slice in T1-weighted sequence, **(I)** Axial slice in FLAIR sequence.

The first follow-up MRI was conducted in September 2013. Coronal T1-weighted imaging (**Figure 4D**) revealed a well-demarcated lesion composed of mixed solid and cystic elements. The solid component demonstrated contrast enhancement, indicative of increased vascularity and heightened tumor activity. Although the lesion exerted mass effect on adjacent structures, it did not display features suggestive of infiltrative growth. In sagittal T1-weighted images (**Figure 4E**), extension into cortical and subcortical regions was evident without significant midline shift, consistent with preserved intracranial pressure. Axial T2-weighted sequences (**Figure 4F**) showed heterogeneous signal characteristics, with a hyperintense cystic component and slight perilesional vasogenic edema, yet without extensive invasion of surrounding brain parenchyma. Post-surgical changes, including gliosis and persistent perilesional edema in the white matter of the left angular gyrus, were noted.

Subsequent imaging performed in March 2015 (**Figures 4G–I**) demonstrated an irregularly enhancing solid portion within the lesion, suggestive of slow nodular progression. The lesion continued to display the well-defined cystic morphology typical of ganglioglioma, with no significant diffusion restriction or signs of aggressive behaviour.

In April 2017, a minimally invasive surgical procedure was performed for the intratumoral placement of NPt nanoparticles. Postoperative MRI (**Figure 5A**) revealed a hypointense cavity in the left parietal lobe corresponding to the resection site, consistent with postsurgical gliosis and scar formation. Mild dilation of the ipsilateral lateral ventricle was observed, likely secondary to regional brain volume loss following multiple surgical interventions. Cortical thinning adjacent to the lesion was also evident, reflecting chronic postsurgical changes.

Importantly, no pathological contrast enhancement was detected, suggesting complete resection with no evidence of residual tumour tissue. **Figures 5B and 5C** further depicted an irregular surgical cavity with hyperintense signals consistent with inflammatory changes, and hypointense punctate foci corresponding to calcifications and residual blood products. Additional findings included periventricular hyperintensity, attributable to reactive gliosis resulting from repeated surgical manipulations.

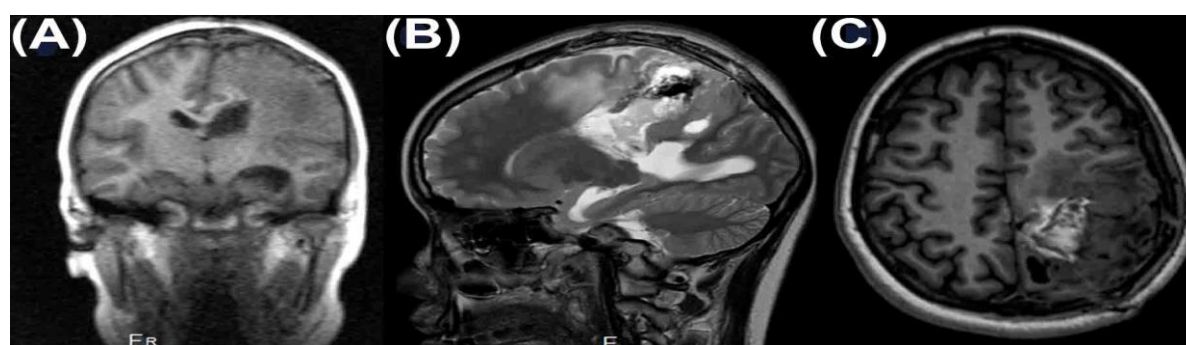


Figure 5: **(A)** Coronal slice T1 sequence, **(B)** Sagittal slice T2 sequence, **(C)** Axial slice T1W sequence.

CASE REPORT: HISTOLOGICAL ANALYSIS

Histopathological analysis played a crucial role intraoperatively in establishing a timely diagnosis and was performed using haematoxylin-eosin staining. Examination revealed a biphasic morphological pattern, characterized by dysmorphic ganglion cells exhibiting abnormal clustering, cytomegaly, and disrupted cytoarchitectural organization (Figure 6). The glial component was well demarcated from the surrounding brain parenchyma and showed prominent calcifications and extensive lymphoid infiltration along the perivascular spaces.

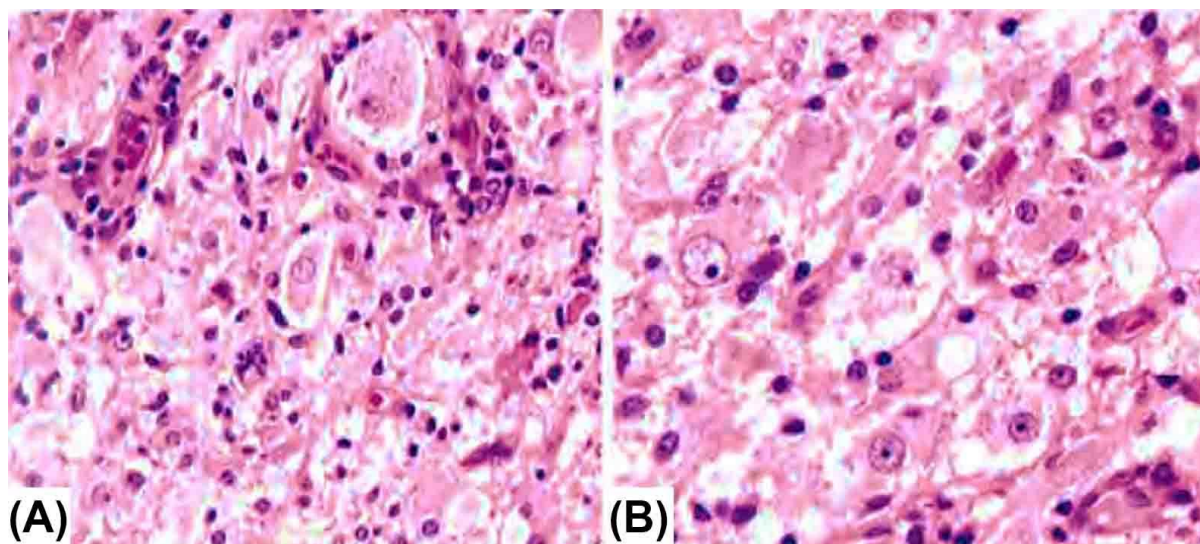


Figure 6: Ganglioglioma histology obtained from the first surgical intervention.

CONCLUSIONS AND OUTLOOK

This study highlights the potential of catalytic nanomedicine as an innovative therapeutic approach for recurrent ganglioglioma. The intratumoral implantation of platinum-coated titania (Pt/TiO₂) bionanocatalysts demonstrated high tumour selectivity, effective localized tumour degradation, and excellent biocompatibility, with no observed recurrence or neurological deficits during a two-year follow-up period. The catalytic generation of reactive oxygen species by the nanoparticles enabled a targeted cytotoxic effect limited to dysplastic cells, minimizing damage to surrounding healthy tissue.^[27] Comprehensive physicochemical characterization confirmed the structural integrity, high surface area, and anatase crystalline phase of the bionanocatalysts, critical features that underpinned their therapeutic performance. The clinical outcome observed in this case suggests that bionanocatalyst-based interventions could complement or, in specific contexts, potentially replace conventional adjuvant therapies such as chemotherapy and radiotherapy, which are often associated with systemic toxicity and limited efficacy in recurrent disease. These results support the further investigation of catalytic nanomedicine within controlled clinical trials to validate its safety, optimize delivery methods, and assess its efficacy across a broader range of neuro-oncological indications. Future studies will be essential to establish standardized protocols and fully elucidate the mechanisms underlying nanoparticle-tumor interactions to maximize therapeutic outcomes.

AUTHOR STATEMENTS

The authors declare no competing financial interest.

All authors contributed equally. All authors have given approval to the final version of the manuscript.

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ETHICS AND CONSENT

The authors affirm that all aspects of the research involving human patients in this study were carried out with the appropriate ethical approval from relevant bodies, and such approvals are duly acknowledged within the article. The study adhered to the guidelines set forth by the National Institutes of Health and was specifically approved by the Ethics Committee of the Children's Hospital of Mexico under Protocol Number HIM 2017-072 entitled "Estudio del Efecto Antineoplásico del Nanomaterial Organoplatino al 1% Soportado en Titanita Funcionalizada (NPt-Ped) en Niños con Tumores de Sistema Nervioso Central con Pronóstico Reservado en el Hospital Infantil de México 'Federico Gómez' y la Universidad Autónoma Metropolitana".

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