CLINICAL PICTURE:

The patient is a 37 years old female who was presented clinically in years 2000 with Parinaud syndrome, The patient first noticed abnormalities in eye movement while she was 12 years old, the patient did not receive medical care since that time and the patient condition remained stable until she was asked to seek medical advice at the age of 37 years. No evidence of increased intracranial pressure is noticed either by history or clinical examination. Parinaud syndrome, which is secondary to compression of the tectum, is the most important clinical presentation of tectal compression. The triad of Parinaud syndrome includes palsy of upward gaze, dissociation of light and accommodation, and failure of convergence. In addition, findings secondary to hydrocephalus resulting from aqueductal compression might be seen in midbrain tumors (not present in this case). Nothing was done to the patient and the patient was followed up by MRI every two years. The patient condition is stable till now (2010) (To inspect the patient’s full radiological study, click on the attachment icon (The paper clip icon in the left pane) of the acrobat reader then double click on the attached file)

RADIOLOGICAL FINDINGS:

RADIOLOGICAL FINDINGS:

Figure 1. Precontrast MRI T1 images showing an isointense tectal plate glioma. The tumor is showing posterior exophytosis to the left side of the quadrigeminal cistern. The posterior parts of the tumor are showing some hyperintense zones, probably representing hemorrhagic spots.
Figure 2. Postcontrast MRI T1 images showing dense ring enhancement within the tectal tumor mass. The mass is seen protruding to the left side of the quadrigeminal cistern.

Figure 3. The tectal plate glioma is hyperintense in the MRI T2 images, with central hypointensities
Figure 4. The tectal plate glioma on postcontrast MRI T1 image (A) and MRI T2 image (B)

**DIAGNOSIS:**

**TECTAL PLATE GLIOMA (FOCAL MIDBRAIN GLIOMA)**

**DISCUSSION:**

The diagnosis of brainstem glioma was long considered a single entity. However, since the advent of magnetic resonance imaging in the late 1980s, neoplasms within this anatomic region are now recognized to include several tumors of varying behavior and natural history. More recent reports of brainstem tumors include diverse sites such as the cervicomedullary junction, pons, midbrain, or the tectum. Today, these tumors are broadly categorized as either diffuse intrinsic gliomas, most often in the pons, or the nondiffuse brainstem tumors originating at the tectum, focally in the midbrain, dorsal and exophytic to the brainstem, or within the cervicomedullary junction. Although we briefly discuss the nondiffuse tumors, we focus specifically on those diffuse brainstem tumors that regrettably still carry a bleak prognosis.

- **Epidemiology**

  Brainstem gliomas account for approximately 20% of all CNS tumors among children younger than age 15. This increase from 10% in the late 1970s reflects an increase in detection of the nondiffuse brainstem tumors by magnetic resonance imaging (MRI) rather than a true increase in incidence. The median age at presentation for all brainstem gliomas in children is 6 to 7 years, with males and females equally affected. Among adults, brainstem gliomas are considerably less common, but reported up to age 70 years, sometimes with a less aggressive course. There is an increased frequency of brainstem tumors among patients with neurofibromatosis type 1, and these tumors have a more indolent behavior.

- **Anatomy and function of the brain stem**

  The human brainstem comprises three connected parts: the midbrain, pons, and medulla, each composed of numerous axonal tracts (white matter), cranial nerve nuclei, and noncranial nerve brain-stem nuclei (gray matter). Thus, the brainstem serves as the conduit through which axonal tracts pass to the spinal cord, cerebrum, or exit as cranial nerves. Within the brainstem, axonal tracts course in longitudinal, oblique, and transverse directions and may synapse within the brainstem nuclei. Newer imaging techniques such as diffusion tensor imaging allow these white matter tracts to be visualized. Neurologic deficits resulting from brainstem tumors result from mass effect or invasion of white matter tracts or nuclei. Midbrain gray matter includes the periaque-ductal gray circumferential around the aqueduct of Sylvius and reticular formation in the tegmentum. Tumors arising in this area tend to be lower grade and focal, and may cause aqueductal obstruction and subsequent hydrocephalus. The reticular formation extends the length of the dorsal brainstem, controlling vital functions such as blood pressure and respiration, while also regulating general level of alertness. Oculomotor (III) and trochlear nuclei (IV) reside in the midbrain. The third nerve exits ventrally from the medial aspects of the crus cerebri in the interpeduncular fossa. Lesions of the oculomotor nerve result in external strabismus, ptosis, and dilation of the pupil from loss of parasympathetic innervation of the radial muscles of the iris. The fourth cranial nerve exits the posterior brain-stem near the lower margin of the inferior colliculus. An isolated lesion of the trochlear nerve results in failure of downward gaze when the eye is turned to the nose and a patient may complain of vertical or skew (diagonal) diplopia. The tectum of the mid-brain is dorsal to the aqueduct of Sylvius and contains nuclei within the superior and inferior
colliculi, which participate in visual and auditory processing, respectively. Tectal tumors are typically low-grade lesions, often affecting the cerebral aqueduct.

In addition to the reticular formation, gray matter structures in the pontine tegmentum include the trigeminal (V), abducens (VI), facial (VII), and vestibulocochlear (VIII) cranial nerve nuclei. Numerous unnamed pontine nuclei reside in the basilar pons. The nuclei of the trigeminal nerve are large, with a motor nucleus in the pons and sensory nuclei extending from the midbrain to the upper cervical spinal cord. The three divisions of the trigeminal nerve exit the lateral pons superior to the middle cerebellar peduncle. The motor nucleus controls muscles of mastication while larger sensory components distribute to the face, mouth, nasal cavity, orbit, anterior half of the scalp, and dura matter.

Cranial nerves VI, VII, and VIII exit the brainstem anteriorly near the pontomedullary junction. The abducens nerve is susceptible to injury because it has the longest intracranial course passing between two layers of dura on the floor of the posterior fossa, to the cavernous sinus, and innervates the lateral rectus muscle after passing through the superior orbital fissure. Injury to the abducens nerve results in horizontal diplopia. Injury to the facial nerve results in ipsilateral facial weakness and loss of corneal reflex when the lesion is peripheral. In addition, when seventh nerve injury is proximal or in the cerebellopontine angle, ipsilateral hyperacusis, absent taste sensation in the anterior tongue, and disturbed secretion of tears and saliva are present. Tumors within the pons are often diffuse, infiltrative of white matter, and effect adjacent cranial nerves and white matter tracts passing through the brainstem.

Grey matter structures in the medulla include the dorsal sensory nuclei and the nucleus ambiguous, in the anterolateral part of the reticular formation. Motor fibers from this nucleus leave the medulla as fibers of the glossopharyngeal nerve (IX), vagus (X), and bulbar accessory (XI) nerves. Lesions of these nuclei or nerves affect swallowing and vocalization and exit the medulla in the dorsolateral medullary groove. The hypoglossal nucleus (XII) is located medially in the inferior medulla and innervates ipsilateral striated muscle of the tongue after exiting through the superior orbital fissure. Injury to the hypoglossal nerve results in ipsilateral dysarthria and weakening of the tongue. The dorsal motor nucleus of the vagus nerve (X) is located lateral to the hypoglossal nucleus and is an important part of the parasympathetic nervous system. Inferior vestibular nuclei (VIII) are positioned dorsolateral in the medulla near the anterior fourth ventricle.

- **Pathology of brain stem glioma**

  Diffuse intrinsic tumors account for approximately 80% of all brain-stem gliomas. [5] These tumors are generally high-grade anaplastic astrocytoma (WHO grade 3), glioblastoma multiforme (WHO grade 4), or occasionally, well-differentiated diffuse astrocytoma (WHO grade 2). [2] At autopsy, most pontine tumors are high-grade gliomas and may disseminate within the neuraxis. [6,7] In contrast, the focal, the dorsal exophytic, and the cervicomedullary tumors are pilocytic astrocytomas (WHO grade 1) or, less often, ganglioglioma (WHO grade 1) or diffuse astrocytoma (WHO grade 2). [2,8] Because of their anatomic location, it is often difficult to obtain tissue for histopathologic confirmation of tumor type and grade.

  The differential diagnosis for a brainstem tumor at an atypical age, or with an unusual presentation or neuroimaging also includes the atypical teratoidrhabdoid tumor as well as the embryonal tumors, (ie, primitive neuroectodermal tumor).9,10 Hemangioblastoma is less common, and occurs in adolescents or adults, particularly in association with von Hippel-Lindau disease. Vascular malformation, demyelinating disease, and focal brainstem encephalitis seldom masquerade as a brainstem tumor.

  - **Clinical presentations of brain stem glioma**

    The most common presentation of a brainstem glioma is that of the diffuse brainstem glioma, rising in the pons and causing diffuse enlargement. Patients typically report a short duration of symptoms, median one month, and have a triad of signs including cranial neuropathy, long tract signs (hyperreflexia, a Babinski sign, and weakness), and cerebellar signs (ataxia, dysmetria, or dysarthria). The cranial nerve impairment is often with multiple, unilateral or bilateral, cranial nerve deficits, particularly cranial nerve VI and VII paresis. The tumor commonly engulfs the basilar artery, and many have axial or exophytic extension to the midbrain, cerebral peduncles, cerebellum, or medulla. Hydrocephalus and metastatic disease from diffuse tumors of the pons are rare.

    In contrast, patients with a focal brainstem tumor have a more insidious presentation, with a long history of localizing signs, such as isolated cranial nerve deficit and contralateral hemiparesis. Elevated intracranial pressure is uncommon. These tumors are usually discrete, well circumscribed, and often times dorsal and exophytic, adjacent to or arising from the floor of the fourth ventricle. They may also involve the medulla or the midbrain, and may present with emesis and failure to thrive. Tumors arising in the tectal region obstruct the aqueduct of Sylvius, causing increased intracranial pressure and hydrocephalus.
Figure 1. Sagittal T2 weighted magnetic resonance image demonstrating a well circumscribed, low-grade, dorsally-exophytic brainstem tumor. (*) Indicates the solid portion of the tumor enhanced homogeneously on T1 weighted images, while the cystic portions of the tumor above and below enhanced less.

Figure 2. Sagittal T1 magnetization prepared rapid gradient echo (A) and axial T2 weighted; and (B) magnetic resonance images showing the infiltrative nature of a diffuse brainstem glioma as it involves the entire pons. The left pons is more expanded as the tumor infiltrates between transverse bands of darker, myelinated white matter tracts.

- Neuroimaging of brain stem glioma

High-quality MRI of brainstem tumors is essential in clinical management to localize the lesion and to differentiate focal from diffuse tumor involvement. A focal tumor is typically well marginated, enhances with contrast, and occupies less than 50% of the axial diameter of the midbrain or medulla (Fig 1). A diffuse tumor is poorly marginated, rarely enhances, occupies more than 50% of the axial diameter of the pons and commonly engulfs the basilar artery (Fig 2). The diffuse tumor, which infiltrates and expands the brainstem, is shown as increased signal on T2-weighted MRI sequences.

Treatment-related changes may be difficult to differentiate from progressive disease. Early cystic or necrotic changes within 3 to 4 months of radiation therapy often represent treatment effects. As intralesional hemorrhage may occur spontaneously or following irradiation, imaging studies should include pulse sequences sensitive to detecting intratumoral bleeding (eg, gradient echo sequences). Computed tomography brain scans can also be useful in detecting and characterizing intralesional bleeding.
Prospective studies testing new imaging technology such as perfusion and diffusion MR sequences, MR spectroscopy (MRS), and positron emission tomography (PET) are accumulating data helpful to differentiate treatment change from disease progression. These imaging methods provide quantitative physiologic and functional information to complement the anatomic visualization provided by conventional imaging. Imaging protocols addressing perfusion and metabolic characteristics of diffuse brainstem tumors are under study by the Pediatric Brain Tumor Consortium.

MR perfusion measures regional blood volume and flow reflecting the vascular nature of neoplasms. Serial changes in tumor vascularity may be a valuable method to monitor effectiveness of therapy, especially as new biologic agents with antiangiogenic properties are introduced. Diffusion tensor imaging provides visualization and quantitative characterization of the major white matter pathways in the brain (Fig 3). It has been used to study supratentorial tumors, and recent studies demonstrate its value in brainstem lesions to guide operative intervention in focal low-grade gliomas, and to characterize white matter tracts in the diffuse gliomas. Figure 3 shows diffusion tensor imaging with color hue demonstrating the principal direction of anisotropic water diffusion in white matter tracts: normal axonal tract orientation in thepons; corticospinal tracts, transverse pontine (TP) tracts, middle cerebellar peduncles, and tracts of the central tegmentum; tumor within the fourth ventricle changing tract orientation in the tracts of the central tegmentum and middle cerebellar peduncles; tumor expanding the TP tracts, but not affecting direction; and tumor expanding the pons with loss of direction in the TP tracts suggesting invasion and destruction of axon fibers.

Proton MRS may provide insight into the biology of brainstem tumors based on the small set of metabolites detected in the human brain. The most prominent peaks in the brain spectrum are N-acetyl aspartate, creatine, and choline. NAA is a neuronal marker and is generally decreased in tumors. The creatine peak includes creatine and phosphocreatine and is often equal to, normal or diminished in neoplastic tissue. Choline reflects the metabolism of membrane turnover, and often is increased in tumors. In addition, peaks from lactate and mobile lipids are often elevated in malignant tumors, especially with necrosis. MRS is being used to differentiate brainstem glioma from brainstem enlargement associated with neurofibromatosis type 1.

[18F]fluorodeoxyglucose (FDG) -PET scanning has not been widely applied to the study of brainstem tumors. The principle indications for FDG-PET are to distinguish viable tumor from posttherapeutic changes and to detect tumor transformation to a higher grade. High FDG accumulation compared with adjacent brain indicates residual or recurrent tumor, whereas low or absent FDG uptake is observed in areas of necrosis. However, FDG-PET findings are not consistent in differentiating tumor from treatment effects. Other tracers may prove more useful in the study of brainstem gliomas. Carbon-11 methionine PET images show higher tumor to nontumor ratios in CNS tumors than FDG images do. Other tracers for future study include 18F-fluoro thymidine, a marker of DNA synthesis and cellular replication.
Management of brain stem glioma

The choice of therapy for a brainstem glioma rests largely on whether the tumor is diffuse or nondiffuse. Prognosis for a patient with a diffuse brainstem tumor is poor, with median survival less than 1 year and less than 20% of children alive by 2 years. [5] Conventional radiotherapy is standard treatment; the role of chemotherapy, radiosensitizing, and other biologic agents remain unclear. Among the nondiffuse tumors, there is a relatively good outcome with more than half of the patients surviving beyond 5 years. This finding and the site of the tumor affect selection of therapy. [1]

- Surgery

At present, surgery no longer has a role in the diagnosis or treatment of diffuse pontine glioma. The MRI findings at presentation are highly specific, and histologic findings do not influence treatment. [24] Meaningful resection is not possible, as the diffuse tumor is interwoven within white matter tracts traversing the brainstem and resection does not confer a survival advantage. [25,26]

In contrast, some nondiffuse tumors, especially those that are focal or cystic, are surgically accessible with modern neurosurgical techniques. [27] Many patients may require no additional postoperative treatment, even after subtotal resection. [25] Extensive resection of a dorsal exophytic tumor, which leaves a layer of tumor on the surface of the brainstem, may prolong progression-free survival without additional treatment. [8,25,28] Whether these surgical results are an improvement over those achieved with conventional local-field irradiation is unclear. A lesion confined to the tectum does not require resection or biopsy, simply shunt placement or third ventriculostomy on development of obstructive hydrocephalus, followed by close observation. [29] Such a patient may remain stable for years or decades, and seldom requires radiotherapy.

- Radiotherapy

Radiotherapy had been the recommended treatment for all brainstem gliomas, but now is used more selectively. For a patient with a diffusely infiltrative brainstem glioma, standard treatment remains conventional external beam, local field radiotherapy to a dose of 54 to 60 Gy in 6 weeks. Without radiation, median survival is approximately 20 weeks. [30] Radiotherapy results in a worthwhile, albeit temporary, improvement in neurologic function, though the overall prognosis for such a patient remains dismal. Increasing the radiation dosage beyond 60 Gy has not proved effective. With hyperfractionation, using total doses of 64 Gy or higher, delivered in twice daily, smaller dose fractions over 6 weeks, failed to yield additional survival benefit.

For a patient whose focal brainstem glioma is of favorable low-grade pathology, radiotherapy may be delivered postoperatively to residual disease or deferred until there is disease progression. [8,25] No controlled or prospective trials have been performed in these patients. For a patient whose tumor recurs after surgical resection, or for a patient with a WHO grade 3 or 4 astrocytoma, radiotherapy should be considered. Radiotherapy should also be considered for a patient with a symptomatic or inoperable tumor.

- Chemotherapy

Currently, there is little, if any, evidence to suggest that chemotherapy has affected the outcome of children with diffuse brainstem gliomas. Response rates to various single-agent and multiple-agent regimens have been disappointingly low, even when chemotherapy was given as initial treatment. Cytotoxic, biologic, or radiosensitizing agents have yet to improve outcome. For a focal brainstem glioma, weekly carboplatin with vincristine has shown activity in a limited number of young patients. [31]

Clinical trials in diffuse brain stem gliomas

For many years clinicians believed that brainstem tumors in children were so rare, and had such a poor prognosis, that it would be impossible to perform a national clinical trial. However in 1983, radiation oncologists from 13 institutions within the Pediatric Oncology Group (POG) participated in a questionnaire survey examining the results of radiation treatment over the 10-year period January 1972 through December 1981. While the outcome of these 62 patients was disappointingly low, with 3-year survival of only 23%, several conclusions were apparent. [32] The role of surgery was limited to confirmation of diagnosis, or volume reduction in selected patients, while radiotherapy was the mainstay of treatment. Failure of local control was the major obstacle to cure, with no evidence to support whole-brain or whole-craniospinal axis irradiation. Investigators soon realized that a cooperative group effort was needed to address serial questions regarding biology and treatment for children afflicted with these kinds of tumors. [32] Thereafter a series of phase I/II trials were conducted in search for ways to improve results of treatment, including altered radiotherapy fractionation schedules, and other approaches such as the use of systemic chemotherapy in combination with radiotherapy and radiation sensitizers. Table 1 summarizes some of these trials. A series of radiation dose escalation studies used hyperfractionated radiotherapy (HRT) with doses from 64.8 Gy to 78 Gy, in a twice daily schedule with an interfractional interval of no less than 6 hours. [33-45] At the highest radiation dose levels, corticosteroid dependency, vascular events, white matter changes, hearing loss, hormone deficiencies, and seizures were observed. These important studies demonstrated that lower-radiation doses would need to be used for future studies.

Table 1. Clinical Trials in Brainstem Gliomas
A trial of concurrent carboplatin and etoposide with 70.2 Gy HRT did not improve outcome over radiation alone, with only 11% of patients with 2-year survival. This experience plus an earlier study of adjuvant chemotherapy of lomustine, vincristine, and prednisone compared with conventional radiotherapy alone showed no improvement in survival with the addition of chemotherapy. An important phase III randomized trial, POG 9239, tested radiation at the standard dose of 54 Gy in 1.8 Gy daily fractions versus 70.2 Gy in 1.17 Gy twice daily fractions, with all children receiving infusional cisplatin as a radiation sensitizer. There was no improvement in event-free survival or survival using HRT, thus leaving conventional radiotherapy as the radiotherapeutic regimen of choice for children with newly-diagnosed brainstem glioma. A comparison of children treated with 70.2 Gy HRT with or without cisplatin, demonstrated that patients receiving cisplatin plus HRT actually fared worse than those receiving HRT alone. The addition of escalating doses of interferon following HRT produced dose-limiting toxicity, but survival of less than 10%. Groups have also tried approaches such as blood-brain barrier disruption (eg, the bradykinin RMP-7) and inhibition of drug resistance with cyclosporine to overwhelm P-glycoprotein mediated xenotoxin efflux. Novel approaches are worthy of continual clinical trials in children with diffusely infiltrative pontine tumors.

**Future perspectives**

Effective experimental models and strategies that will improve survival for patients with diffuse brainstem gliomas are needed. At least three reasons account for our present lack of success. First, diffuse brainstem gliomas share some similarities in biology and behavior as the adult high-grade gliomas, where therapeutic advances have likewise been minimal. The median survival for an adult with cerebral glioblastoma is approximately 41 weeks. Secondly, the generation of biologic studies of these tumors is now hampered by the current practice of not obtaining tissue for diagnosis. We cannot assume that biologic properties of brainstem tumors in children are completely identical to cerebral high-grade gliomas of adults. Limited prior studies using archival tissue from diffuse brainstem gliomas have demonstrated overexpression of the erbB1 receptor and presence of PTEN mutations. However, our future potential to more fully characterize the genetic or molecular nature of diffuse brainstem gliomas via conventional molecular or genetic methods is now thwarted by a lack of specimens. Finally, the location of these tumors, where the vital architecture of white matter tracts and gray matter nuclei are compactly juxtaposed with brain matter, has made resection or the placement of therapy directly into the tumor essentially impossible.

Currently clinical trials employ chemotherapeutic and other biologic agents investigated in trials in adult gliomas, which unfortunately, are not sufficiently similar to the diffuse brainstem glioma of children. Nevertheless, immediate and future attention has turned to epidermal growth factor receptor signal inhibitors, antiangiogenesis agents (thalidomide), farnesyl...
transferase inhibitors, radiation sensitizers (gadolinium texaphyrin, arsenic trioxide), inhibitors of chaperone proteins (e.g., 17-AAG), and histone deacetylase inhibitors (valproic acid). The Children’s Oncology Group is studying the oral alkylating agent temozolomide concurrent with and following conventional radiotherapy, in an attempt to build on the small success demonstrated in adults with glioblastoma. [54] Phase I/II studies are being conducted in the Pediatric Brain Tumor Consortium with the EGFR inhibitor erlotinib and the farnesyl transferase inhibitor R115777. Future advances in diffuse brainstem gliomas will likely require entirely new technological approaches. Advanced MRI techniques may begin to elucidate the biology of these tumors, especially over their clinical course. Local delivery of drugs by an osmotic pump is one example of an early effort to invoke novel engineering and an entirely different paradigm. Whether bioengineering or nanotechnology can provide therapeutic success in this tumor remains to be determined.

**SUMMARY**

In comparison to children, brain stem gliomas in adults is a less understood disease and present a much lower incidence (<2% of gliomas) [56]. The survival rate however can be very much longer with the peak incidence in the third and fourth decades [57]. The duration of symptoms is generally shorter in children while in adults it tends to be longer with tectal tumors, fourth ventricle dorsally exophytic tumors and cervicomedullary gliomas having a more favorable prognosis when compared to diffuse lesions.

Tectal plate gliomas have been reported as a particulary indolent lesion often remaining stable in size for many years. The majority of these lesions were described as low grade gliomas, presented with late-onset aqueductal stenosis often without associated brain stem signs [63]. The average age at the time of diagnosis is about 10 years in children, with the majority of cases presenting with raised intracranial pressure secondary to obstruction of the Sylvian aqueduct [69], with some other cases presented with Parinaud syndrome [70] like the patients observed in our illustrative cases, although this is also an uncommon feature. Even before the development of computed tomography, tectal tumors were an unrecognized because of late onset hydrocephalus, once said that this can be the smallest lesion that can lead to death of the patient.

The radiologic investigation of these tumors have been up graded in the CT scan era with better visualization of the tectal region; but the majority of lesions continue to appear as noncommunicating hydrocephalus alone, although calcification or a hypodense lesion on the tectal plate can be observed. MRI of these tumors reveals tectal distortion or thickening caused by a localized mass, leading to aqueductal compression and hydrocephalus [71]; characteristic T1 hypointensity and T2 hyperintensity [72-76]. MRI is an accurate and noninvasive method of diagnosis that can be indicated in all cases of late onset hydrocephalus and aqueductal obstruction, especially in adults. Even when present, contrast enhancement after gadolinium injection was an independent factor of tumor grading [72]. The characteristics of the lesion seen on the MRI is of course dependent on its pathologic basis.

The prognosis of tectal plate lesion is a much debated issue in neurosurgical publications. It is reasonable to expect that a malignant lesion, brain stem metastasis and rapidly infiltrating lesions present a poor prognosis. But even in cases of tectal gliomas some controversies had arise.

Initially the pathology of intrinsic tectal lesions were considered to be similar to other brainstem tumors. In fact this was later discovered as not true. Many papers have oriented the presence of this tumor as a special site of low grade gliomas [59-61,64,65,73]. These tumors appear to present a better prognosis given to a slow growing rate and boundaries displacement instead of an infiltrative behaviour. However, the management of the patients remains controversial. The majority of authors oriented the management including a mandatory histopathological analysis to ascertain the low grade mark, and just afterwards the adequate treatment is traced.

Surgical treatment consists on tumor resection or open biopsy; generally the surgical approach is that described for pineal region lesions, like the suboccipital-transtentorial or supracerebellar-infratentorial approach, prefered by this paper’s senior author and routinely performed in this institution adopting the semi-sitting position.

Ultimately, with the advance of neuroendoscopy, a new perspective in dealing with these lesions had arisen. When the objective is to treat the hydrocephalus, a rigid endoscope can be utilized to perform a third ventriculostomy with no major technical complications; the visibility of the inner surface of the third ventricle, with all structures involved is very good [77].

Tectal tumors may extrude from the tectum into the lumen of the cerebral aqueduct and subsequently protrude to the third ventricle, pushing away the posterior commissure and enlarging the orifice. Some authors consider this lesion as intraventricular ones, treating each lesion individually [74].
With the advent of the flexible neuroendoscope other types of therapy could be performed. Potential treatment options include shunt placement [75] and aqueductal plasty [74,76]. The third ventriculostomy success and failure rate is similar to those cerebral-spinal fluid (CSF) shunts – about 30% [24].

In conclusion, tectal tumors represent a differentiated category in neuro-oncology. Despite a variety of lesion encountered in this region, most publications indicate that low grade glioma is most prevalent. However, the major difficulty is regarding the adequate treatment of these lesions. A number of articles orient an invasive approach consisting of surgical treatment, given the need for mass reduction or just to obtain histopathological sample [60,82,83]; other authors decline to conservative treatment, justified by the benign behavior and slow growth of this tumors [59,61,63,74,84,85].

**Addendum**

- A new version of this PDF file (with a new case) is uploaded in my web site every week (every Saturday and remains available till Friday.)
- To download the current version follow the link "http://pdf.vassermetwally.com/case.pdf".
- You can also download the current version from my web site at "http://vassermetwally.com".
- To download the software version of the publication (crow.exe) follow the link: http://neurology.vassermetwally.com/crow.zip
- The case is also presented as a short case in PDF format, to download the short case follow the link: http://pdf.vassermetwally.com/short.pdf
- At the end of each year, all the publications are compiled on a single CD-ROM, please contact the author to know more details.
- Screen resolution is better set at 1024*768 pixel screen area for optimum display.
- Also to view a list of the previously published case records follow the following link (http://wordpress.com/tag/case-record/) or click on it if it appears as a link in your PDF reader
- To inspect the patient's full radiological study, click on the attachment icon (The paper clip icon in the left pane) of the acrobat reader then double click on the attached file.
- Click here to download the short case version of this case record in PDF format

**REFERENCES**

References


88. Case of the week...Brain stem glioma. [Click to download in PDF format]

89. Case of the week...Focal midbrain tumor. [Click to download in PDF format]