Prediction And Correlation Of Computed Tomography Findings With First Clinical Presentation in patients diagnosed Primary Brain Tumour.

By:

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Abstract

English

Topic: Prediction and correlation of computed tomography findings with first clinical presentations in patients diagnosed primary brain tumour.

Overview: The brain tumour is increasing in incidence nowadays probably with the availability of new high technology facilities such as computed tomography, magnetic resonance imaging and angiogarphy. There is a wide spectrum of presentation of patients in term age and clinical presentations. An early detection of the tumours will generally give a better outcome with the availability of neurosurgeons and neuroimaging facilities. Neuroimaging play an important role in detecting the lesion ,hence to reduce the morbidity and mortality.

Objective and method: The aim of this study is to establish the predictive and correlative value between clinical and CT scan findings. All patients histologically proved to be primary brain tumours were included in this study. The findings on computed tomography findings and on first clinical presentations were recorded and analysed.

Results: A total of 47 patients were included in the study. All variables for the imagings had shown no significant predictive and correlative value on univariate and multivariate analysis. The most likely reason was due to small sample size. Posterior fossa was the most common tumour site. The most common clinical presentation was headache (85%) and vomiting(60%). Astrocytoma was the most common brain tumor constituting 32% of the cases.

Conclusion: There was no correlation between computed tomography findings and clinical presentation. However in large sample size study it will give a significant value as be done in other study.

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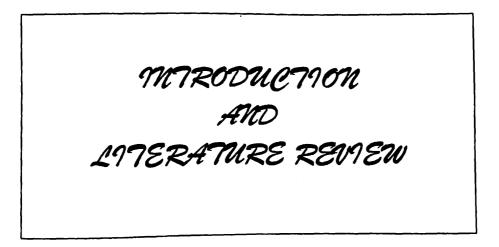
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Conclusion: This study was unable to predict and correlate the computed tomography findings with the first clinical presentations in patients diagnosed primary brain tumour.

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SECTION ONE



1.1 INTRODUCTION

The brain tumor for most individuals is the most dramatic form of human illness. They are among the most rapidly fatal of all cancers. Only about half of patients are still alive 1 year after diagnosis. They occur as the most common solid tumor of childhood and the second most common malignancy of childhood. Among adults, primary brain tumor rank from 6th to 8th in frequency of all neoplasms.

The management of brain tumor was made easy by the technology advancement in the diagnosis and localization of brain tumor using Magnetic Resonance Imaging(MRI) and Computed Tomography (CT) scan of the brain.

Most of brain tumor needs to be operated for complete resection. This may be followed by other modalities of treatment such as radiotherapy, chemotherapy or immunotherapy.

The clinical features of brain tumors are due to both the local effects and the generalized effect of an intracranial mass. They can present in several ways. By their growth, they can cause an increase in the intracranial pressure, either directly by the mass of the tumor or indirectly by obstructing the circulation of the cerebrospinal fluid (CSF) and producing hydrocephalus. In addition, bleeding may occur into the tumor, with a sudden increase in its mass effect. The symptoms that may be produced by a generalized increase in intracranial pressure are headache, nausea, vomiting and a reduction in the level of consciousness. Headache is a common in patients with tumor and abscess, but may be absent. It is usually generalized, dull and aching,

exacerbated by straining, coughing or defecation, and classically worse in the morning, where it may be accompanied by vomiting. The patients may exhibit papilloedema and unilateral or bilateral abducens paresis. The central nervous system is enclosed in a rigid bony framework, and any increase in mass content produced by abscess, oedema, tumor, haemorrhage or failure to absorb cerebrospinal fluid results in an increase in pressure within the cranium. A generalized increase in intracranial pressure may be tolerated for a period of time; but with further tumor growth, brain herniation may occur, with a rapid decline in the patient's neurologic function.

A second way in which an intracranial pressure may present is by the loss of function of the portion of the nervous system involved by the tumor. In contrast to the symptoms and signs caused by an increase in intracranial pressure, those caused by the loss of the nervous system function often permit an accurate presumptive diagnosis based on the neurologic history and physical examination.

Finally, an intracranial tumor may present with hyperactive function. The tumor itself can be the cause of this hyperfunction, such as a pituitary adenoma that overproduces one or more hormones

The presence, location and type of intracranial tumor can sometimes be suspected from the patient's symptoms and signs. In this case, the diagnostic studies are tailored to confirm the suspicion and to facilitate treatment. Often however, the symptoms are vague and the signs are nonspecific, especially early in the course of the disease. In this circumstances, the initial diagnostic studies are of a general or screening type.

<u>1.2 LITERATURE REVIEW</u>

A few study had been carried out in correlating with sign and symptoms to the site of brain tumors supratentorial vs infratentorial. Alexander et al (1995)

Headaches are the most common symptom. They may indicate a growing tumour increasing pressure on normal tissue in and around the brain. Any new or persistent severe headache should be reported to a physician. Headache often occurs in form of attacks, which may be severe, and sometimes unbearable, for the patient. Often the patient reports that the headaches are most violent in the early hours of the morning and subsequently decrease gradually. A more specific location of headache, however severe, rarely yields any information as to the site of the tumor. For instance, a tumour in the back part of the skull may cause pain at the back of the head, but it may also give rise to pain exclusively in the forehead. With other tumors in other regions, the headache may also be felt some distance away from the site of the tumor. In the case of some tumors located near the surface of the skull, a constant, strictly localised headache may give an indication as to the site, but these are exceptions.

Finally, it should be noted that headache may also be completely absent in some cases of brain tumor. Furthermore, it is often noted that a headache that is present at first may disappear completely, for some inexplicable reason, during the subsequent course of the disease. These have been identified by Forsyth and Posner (1993) in a prospective study of patients with either primary or metastatic tumors. Headaches were present in almost half of the patients with each of these tumor types. In only 8 of

These have been identified by Forsyth and Posner (1993) in a prospective study of patients with either primary or metastatic tumors. Headaches were present in almost half of the patients with each of these tumor types. In only 8 of the 53 patients with headaches were neurologic symptoms or signs absent (these would include hemiparesis, seizures, papilledema).

The most-common headache (in 41 of the 53 patients) resembled tension-type headache. Some descriptions of it were dull ache, pressure, like a sinus headache. Its location was usually bifrontal. When it was unilateral (25% of patients had this), it was always on the side of the tumor. Posterior head pain occurred mostly with posterior-fossa tumors. nausea or vomiting occurred in 48% of patients. Such symptoms are, of course, most atypical for tension-type headache. Another feature distinguishing this tumor-related headache from tension-type was its intensification when the patient bent over or strained to lift an object or to move the bowels.

The headache was intermittent in 62% and continuous in 36% of the sufferers (unknown in 2%). The intensity of the headache had no consistent pattern from morning to night. In only 5 patients did the headaches resemble migraines.

All of them had abnormal neurologic symptoms or signs inconsistent with migraine. Seven patients had headaches which did not fit the features of any standard headache type. 18 of the 53 patients with headache had increased intracranial pressure. Most of them had headaches resembling tension-type headaches. Their headaches were, in general, more severe, more nauseating, and less responsive to common analgesics than the headaches in patients without increased pressure. Cartmill M et al (1998) Relation of headaches to tumor size, in patients with headaches, the mean twodimensional tumor size (18.3 square cm) and the mean midline shift of the brain (6.1

mm) were greater than in those without headaches (9.3 square cm and 2.7 mm). These relationships are consistent with the standard hypothesis of headache generated by tumors, namely that the headache is caused by traction on pain-sensitive structures such as large blood vessels, the dura, and cranial nerves. Forsyth PA et al (1993)

In paediatric age group in 315 patients presented with headache and no known neurological deficit underwent MR and another 69 underwent CT brain study showed only 4% (13) had surgical space occupying lesions. Seven independent multivariate predictors of surgical lesion were identified. The strongest predictors were sleep related headache and no family history of migraine .other predictors were vomiting ,absence of visual disturbance , headache less than 6 months duration and confusion . They also found that no difference in detection of surgical space occupying lesion between MR and CT. So for the conclusion at high risk on the basis of the criteria usually need neuroimaging, while children at low risk may be safely followed up clinically without neuro imaging.

In children seizures were the most common presentation and CT scan was suggested for all cases with newly diagnosed epilepsy.(Sjors K, dept. paed. University Hospital Lund,, Sweden 1980-87). However in one study showed only 12% of 203 paediatric patients presented with seizure to emergency department revealed abnormal finding where the majority was due to hemorrhage(32%). So the conclusion of this study the absence of defined high risk factors predicted normal head CT findings and the deferred of emergency CT in this population to be considered. Medina LS et al (1997)

The occurrence of epileptic attacks is very frequent, and usually an early symptom of a brain tumor. It is very useful for physicians if they can supplement the date of the person's medical history by personal observation of an attack.

Brain tumors give rise to epilepsy in the majority of cases. If the epileptic attacks repeatedly exhibit certain constant focal phenomena, and this applies particularly to

Jacksonian attacks, it is possible to determine the site of the tumor with a greater or lesser degree of certainty on the basis of these findings. Warden CR et al (1997) Mierzejewska et al (1976) study in Poland reported the brain tumor cases over 50 years old, psychic changes and neurological deficit (hemiparesis) were the most frequent manifestations

Sayama I et al (1986) in his study, reported patient presented with uncommunicative and difficult to walk in three months prior to admission and also noted nocturnal urinary incontinence just before admission, computed tomography scan revealed tumor in the posterior wall of the third ventricle with subsequent hydrocephalus where later histologically confirmed to be pineocytoma.

Global amnesia one of the initial clinical manifestation in brain tumor (Erokhina LG et al (1997). Affectives and schizophrenia-like psychoses are related to dysfunctions of the right and left hemisphere, respectively and lesion of the temporal lobes commonly cause depression. There is no clinical method of localizing or excluding a brain tumor by its psychiatric manifestatio.n Uribe VM et al (1990)

Bradypsychism is a general slowing-down of the psychological process -is a symptom that is frequently encountered, particularly in the case of large tumors in the cerebral hemispheres. Occasionally, the patient exhibits more extensive psychological disorders, sometimes even genuine psychoses, and these will cause major problems in daily living.

Tumour associated aphasia in left hemisphere primary brain tumor on 32 patients found significant relationship between greater patient age, tumor size and tumor grade

but tumor location within the left hemisphere did not correlate with aphasia (Recht LD et al (1989)

Sala F et al (1996) in his study on clinical and neurologic finding children (40 patient <3 years old) with intracranial brain tumor showed vomiting(70%), ataxia(53%), headache(28%), lethargic (28%), increased head circumference (23%) and irritability(23%). Infratentorial (88%) and average size 4.3 ± 1.4 cm x 4.2 ± 1.7 cm x 4.1 ± 1.8 cm.

In 71 cases of glioma consisted of 35 girls and 36 boys. In this study tumor involved the left hemisphere in 42 children and the right in 29. 23 children were younger than 5 years of age at the time of diagnosis, 18 were between 5 and 10 years and 30 were between 10 and 18 years of age. The most common presenting symptoms were seizures in 41 patients, headache in 29, vomiting in 22 and hemiparesis in 16. Less common symptoms include visual loss in 11 children, lethargy in 10, personality changes in 8, aphasia in 6 and hemisensory complaints in four. Papilloedema is the most common finding on physical examination was detected in 19 children. various focal findings were detected in 32 children, in 26, these neurological deficit were mild and 6 had severe neurological deficit. On imaging findings of computed tomography found that the tumour involved parietal lobe in 27 children, the frontal lobe in 25 children, the temporal lobe in 21 and in occipital lobe in 12. In 14 children more than one lobe was involved. Ian F. Pollack et al (1995)

Hydrocephalus is present in 85% (Comi AM et al (1998). Most common tumour produced hydrocephalus was choroid plexus. In review study of 38 cases of choroid plexus revealed all cases showed hydrocephalus. In 27 cases the tumor arose from the

lateral ventricle (15 in left and 12 in right), 3 cases from third ventricle, 5 cases from the fourth ventricle and 3 cases were located extraventricularly at the cerebellopontine angle. Most common symptoms were intracranial hypertension symptoms and signs (headache (17) nausea/vomiting (15), macrocephaly (12), drowsinees (16), papiloedema (6), sixth cranial nerve palsy (4), exopthalmos (1). In motor sign, hemiparesis/hemiplegia (5), gait ataxia (4), hypotonicity (4) and cerebellar sign in 1. Seizures developed in 5 patient whereas psychic disorder in 4 and mental retardation in 5 patient. (Philipe P et al (1998).

Review of 54 cases of cerebellar astrocytoma, revealed that vermis was the commonest site in 17 patients, left hemisphere in 14, right hemisphere in 11, midline in 12 and fourth ventricle in 2 patients. The most common presenting symptoms were headache in 32 (82%), vomiting in 24 (62%), nausea in 19(49%), gait ataxia in 20(51%), arm ataxia in 19(49%), weakness in 8(21%) and diplopia in 8(21%). Hydrocephalus was present in 32 cases. There was no predilection value for a particular portion of the cerebellum on the part of the any particular tumor. Histological subtype revealed pilocytic astrocytoma was the most common represent 72% of cases. M Morreale et al (1997)

A.N. Konovalov et al (1996) in his review of 10 cases of meningioma in pineal region found that the symptoms and signs mainly related to the increased intracranial pressure, disturbances of coordination and ocular abnormalities. The size of the tumors calculated from the diagnostic images and verified at surgery varied from 3 x 2.8 cm to 6 x 4.2 cm in size. The clinical history is usually insidious and a long interval often passes between the start of symptom and diagnosis. The most common clinical presentation was headache and gait disturbances in 6 cases, mental changes and cerebellar sign in 5 cases, hearing impairment, pyramidal weakness, pupillary abnormalities and diplopia in 3 cases and upward gaze paresis in only 1 case. Upward gaze paresis was the typical for the pineal region tumor where the tumor compressing the mesencephalic tectum.

Hearing impairment was due to local tumour interference with central auditory pathways. Most of the patient 's symptoms and signs return to normal post operatively indicate that were due to mass effect of the tumors.

Clinical and radiological correlation was performed in 9 cases of intracranial schwanoma at Mayo clinic by Auer RN et al (1982). Preoperative CT scan in 8 patient and MRI in 3 patient were reviewed. All the tumours were well circumscribed and intraaxial in location. The size ranged from 1.2 cm to 6 cm in size the average of 3.4cm. Presence of mild perifocal edema in all cases except in 1 case. 5 of the 9 lesions involved at least a portion of the temporal lobe, 3 were periventricular in location (adjacent to the trigone of the lateral ventricle) and 1 large multiloculated mass involved the temporal lobe, insula and basal ganglia. 3 tumours in the posterior fossa region including the cerebellar hemisphere in 2 cases and the inferior vermis. In another 1 tumor located at the parietal lobe near the vertex. Degree of enhancement was depend to the cystic and solid component whereby the solid component revealed homogenous moderate enhancement and cystic type revealed intense enhancement of the wall. When compared to the preoperative angiography demonstrated that 2 of the tumors were hypovascular, while the rest showed mild vascularity and tumour staining that persisted into the venous phase of the angiogram. For the clinical aspect, the duration of the illness prior to diagnosis varied greatly, ranging from 10 days to 9 years. Patient in the younger group tended to show no symptoms or to have longer duration of the illness. In 1 patient, the tumour was an incidental finding on CT scan when evaluating for minor head injury. Another patient had exprienced positional headache. CT scan showed a tumor lying adjacent to the trigone of the lateral ventricle. No neurological deficit accounted. 2 young patients presented with focal seizures, but exhibited no functional deficits. On the other hand, patient 49 years old or older tended to exprience a more rapid clinical course and to exhibit neurological signs. 3 of the five had a duration of symptoms of 3 weeks or less, 3 presented with neurological deficits and 3 had severe headaches.

In other similar study of intracranial Schwanoma of 23 patients, age less than 29 yrs (80%). old, the most common symptoms were chronic seizure and headache with a duration varying from 1 year to 40 years. The majority of the tumour were located in the frontal and temporal regions and six were in infratentorial.

G.P. Casadei et al (1993)

Case	Age (yr)	Sex	Symptoms	Duration of symptoms	Tumor location
1	16	М	none	0	rt.temporal
2	17	м	seizures	6/12	lt.temporal
3	21	м	seizures	9yrs	rt.parietal
4	23	F	positional headaches	10 days	lt.temporal (periventricular)
5	49	F	headache	2 mo	lt.temporal (periventricular)
6	52	F	headache/ hemiparesis	3 wks	rt. cerebellum

Table: correlation of s	ite and symptoms
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7	55	М	headache	3 wks	inferior vermis
8	79	F	ataxia	2 mos	lt.cerebellum
9	84	F	mental change/ hemiparesis	3 wks	rt. medial temporal

There was reported cases of intractable seizure in glioma. The study population included 45 patients, ranging in age between 4 and 73 years (mean 30 yr) with about equal number of male and female ratio. The most common presenting symptom was intractable seizure activity and very few had focal motor or sensory sign which was present very subtle. No patient had a fixed language deficit although several individuals demonstrated occasional interictal or temporary postictal expressive aphasia. DF Smith et al (1991).

All patient had preoperative CT scan with intravenous contrast. Subtle contrast enhancement was documented in only two of the 45 patients. The CT scans showed the typical appearance of a homogenously hypodense lesion in every instance. There were 12 right sided tumours and in most cases (33) the mass was located in the dominant (left) hemisphere. The location of all seizure foci predominantly temporal lobe tumours, the vast majority of the epileptiform discharge originate from mesial temporal lobe structures, such as amygdala, uncus, anterior hippocampus and parahippocampal gyrus. From this study, found that temporal lobe tumor in 29 patients, frontal and parietal lobe tumor in 8 patients respectively. Histological type of the tumor were oligodendroglioma in 14 patients, astrocytoma in 13, gangloglioma in 9 and oligoastrocytoma in 9 patients. (Spencer DD et al (1987) Bullit et al (1995) found that in facial pain correlating with location of tumor found peripherally placed tumors tend to cause atypical facial pain associated with sensory loss, middle fossa –trigeminal neuralgia and progressive neurological deficit and posterior fossa tumors are most likely to cause trigeminal neuralgia and subtle neurological deficit.

Vomiting is seen as a reliable symptom in patient with brain tumour, but it rarely leads to diagnosis in the absence of a recognized neurologic deficit. (Squires RH et al (1997). Vomiting due to a disorder of the function of the brain may often be recognized because of its sudden explosive character and the almost complete absence of either preceding or subsequent nausea. The attacks of vomiting and headache may often coincide. The frequency of the vomiting is highly variable. If vomiting is the initial symptom and is a frequent daily occurrence, it constitutes an argument in favor of the tumor being located in the back part of the brain.

An awake patient presented dilation of the pupil of the eye. This is in principle an objective sign. It may be present for a long time before any disorder of vision occurs. In more than 95 percent of the cases of dilation of the pupil of the eye, there exists a space-occupying tumor in the brain. If the dilation of the pupil is much more pronounced in one eye than in the other, it generally constitutes an argument in favor of the location of the tumor in the cerebral hemisphere on the same side as the eye with the greater amount of congestion. Hockley AD et al (1991)

There is a case where patient presented with central neurogenic hyperventilation, which induced, by cerebral tumour, cytologically proved to be B-cell Lymphoma. After treatment with steroid and brain irradiation it showed significantly improved, however after 6 months it recurred again and presented with central neurogenic hyperventilation. PauznerR et al (1989)

Focal tumours have an excellent prognosis regardless of the site of tumor origin. Diffuse tumour of the mesencephalon and pons have a significantly poorer prognosis than focal tumors, with diffuse pontine tumours having the worst prognosis. The presence or absence of enhancement after administration of contrast has no significant relation with outcome, overall or within specific tumor subgroups. Fiscbein NJ et al (1989)

P. C. Burger et al (1989) had conducted a study of topographic anatomy and CT correlation of 15 patients histologically proved to be Glioblastoma Multiforme. The age range between 61 to 86 year old of 9 male and 6 female. The interval of first symptom and radiological diagnosis were documented and found that the earliest was 2 days in which tumour located at the left temporoparietal lobe in 61 year old female and patient died on the next day. The longest interval was 4 years in which tumor located at the corpus callosal in 72 year old female and patient died after 10 weeks of diagnosis.

In review of correlation of clinical, radiological and pathological of 193 intracranial meningioma was done in Canada by M Rohringer et al (1989). They had divided in two groups of meningioma consisted of 179 benign meningioma and 14 malignant meningioma. Results showed a greater proportion of patients with malignant than benign meningiomas demonstrated limb paresis on initial examination, although the malignant tumour sample size was small. In both group, headache was the most common presentation in 75 patients, personality change in 46 patients, seizures in 37,

visual impairment in 34 patients, focal seizures in 31, ataxia in 31, aphasia in 21, decreasing in level of consciousness in 15 and decreasing hearing in 2 patient. On physical examination, normal finding in 53 patients, paresis in 64 patient, memory impairment in 32 patients, cranial deficit in 21, visual field deficit in 22, aphasia in 18, papilledema in 17, altered level of consciousness in 11 and clinically reduced hearing in 4.

Regarding tumour location, in 67 patients(35%) the tumors were located over the cerebral convexities, in 43 in parasagital and in 33 they were located along the sphenoid ridge. The remaining tumors were distributed throughout the cranial compartment with 10 (5%) located within the lateral ventricle. Of the 43 patients with parasagital tumours, the majority (49%) were located over the anterior 1/3 of the falx cerebri, 29% lay within the middle third, and the remaining 22% were located along the posterior third of the falx. The majority of malignant tumors (50%) were located over the cerebral convexities.

On computerized tomography findings, significant midline shift seen in 152 patient, homogenous markedly enhancement in 134 patients and non homogenous enhancement in 50 patients, no perifocal edema in 86 patients (all in benign type), mild perifocal edema in 57 patients (55 in benign and 2 in malignant), moderate perifocal edema in 20 patients (10 in benign and malignant respectively), severe perifocal edema in 30 patients (28 in benign and 2 in malignant) and bony hyperostosis in 33 patients (32 in benign and 1 in malignant). M.Rohringer et al (1989)

D.M. Garcia et al had reviewed 84 cases of cerebellar astrocytoma with the patient's age ranged from 1 to 19 years with mean age of 6 years with the clinical presentation. The most common presentation was papilledema (86%), headache (82%), ataxia (82%), nausea/vomiting (81%), cranial nerve deficit (37%), visual impairment (21%), altered mentation (8%), hemiparesis (5%), seizures (2%) and speech impairment (2%). Most of the patient presented with more than one sign and symptoms.

Bradycardia also one of the sign of brain tumor especially when it involves at the medulla oblongata and it were possibly due to vagus stimulation caused by the tumor i.e glioma in the medulla oblongata.

Correlation study between peritumoral edema with CT scan and angiographic showed that presence of peritumoral edema when vascular portion is provided by leptomeningeal branches of the internal carotid whereas in the absence of edema when the whole vascular supply of the tumor is provided by the meningeal branches of the external or internal carotid (Cascao Mani J, French). Other study had shown that severity of edema positively related to the size of tumor, also influenced by histology subtypes however no correlation to the site of tumor. In other study on 74 meningiomas found 49 cases has peritumoral edema which are 46 supplied by pia vessels, 3 were supplied exclusively by dural vessels(Edema Index >3) and for those without peritumoral edema showed no pial blush (EI<1.1) Bitzer et al Germany. Degree of edema also close relationship to the aggressiveness of tumor. Ide M et al (1994)

In general, local symptoms and signs can be predicted according to the location of the tumor.

Site	Left frontal area	Left temporal area	right temporal area	Cerebellum	Midbrain	Brain stem
Clinical presentation	Epileptic attacks Psychological disorders Motor aphasia Hemiparesis	Epileptic attacks Sensory Hemiparesis Hemianopia Psychological disorders	Epileptic attacks Hemiparesis Hemianopia Psychological disorders	Ataxia Corneal areflexia Nystagmus	Diminished reactions of the pupils of the eyes Vertical disorder of gaze Ataxia Increased tendon reflexes	Horizontal disorders of gaze Alternating hemiplegia Absence of dilation of pupil of the eye

Imagings Modalities

Plain radiography.

This examination can reveal effects that a tumor might have on the bone

of the skull. It can also show tiny calcifications (deposits of calcium) in the

brain that may indicate the presence of a tumor.

The role of plain radiograph is for screening presence of indirect sign to suggest high index of suspicious of brain tumor and further guide for further imagings. In meningioma plain skull radiograph will show evidence of hyperostotic changes . Widening and erosion of pituitary fossa to suggest presence of pituitary tumor or indicate long standing increase of intracranial pressure. Displacement of normal physiologic calcification such as pineal gland will suspicious of presence of space occupying lesions. Sometimes destruction of skull also indicate presence of brain tumor. Abnormal calcification will alert presence of calcified brain tumor such as in oligodendroglioma.

Computed Tomography (CT)

At present, the most common screening examination is computed tomography. Most brain tumors are demonstrated by such scanning, especially if scans made after the intravenous injection of an iodinated contrast agent (contrast-enhanced scans) are compared with analogous unenhanced scans. The sensitivity of CT for detecting intracranial neoplasms is sufficiently high to detect incidental lesions in neurologically asymptomatic patients. CT has the ability to differentiate a wide range of tissue types including air, fat, soft tissue and bone with superior spatial resolution. CT is highly sensitive to blood within the brain and is the technique of choice for evaluation of the presence of acute intraparenchymal, subarachnoid, subdural or epidural hemorrhage (acute blood may be indistinguishable from brain tissue on MR imaging). Bony structures are optimally demonstrated and CT is the imaging modality of choice to evaluate skull metastases and other bony pathology.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI), which involves a high-powered magnet, became available in the mid 1980's. MRI images are quite distinct, allowing a more detailed examination than is possible with CT. Because MRI gives not only an axial view but also coronal and sagittal views, three-dimensional examination is possible. Due to the risks involved in standard angiography (an invasive procedure carried out routinely before CT became available), MRA (magnetic resonance angiography) has replaced angiography in most situations. MRA uses a special software package along with MRI, which can provide relatively good answers about the vascularity of the tumor. MRS (magnetic resonance spectroscopy) is also relatively new. This diagnostic tool can show a chemical make-up of the tumor and differentiate tumor growth from other lesions. Recent technological advances have made it possible to locate the primary center of the brain (motor cortex, speech center, etc.) by functional MRI (fMRI).

MR imaging has become the primary technique for probing the brain and will continue to have that role in the future. Molecular imaging with various MR, SPECT, or PET probes may further our understanding of brain metabolism and function. There is no question that we will have a more profound understanding of how the brain works as functional imaging becomes more refined.

Positron Emission Tomography

Positron emission tomography (PET) has provided valuable biophysiological information on various central nervous system disorders. In brain tumors, various radiotracers have been applied with PET to evaluate tumor blood flow and metabolism, as well as to detect tumors and to assess the degree of malignancy . Among the various radiotracers, fludeoxyglu-cose F 18 (FDG) has been the most frequently used for the evaluation of glucose metabolism in brain tumors. In addition, the assessment of tumor protein synthesis has been attempted with various amino acid tracers, and the most experience has been obtained with L-methyl-11 C-methionine (11 C-methionine). In a few studies both FDG and 11 C-methionine have been applied

to tumors (8-10). In the present study, we applied both tracers to patients with glioma or meningioma to evaluate the differ-ences of FDG and 11 C-methionine as radiotracers for brain tumors. T Ogawa et al (1996)

Aetiology and predisposing factors

There is little information about the relation of environmental factors to primary brain tumours. The cause of the majority of nervous system tumours remains unknown. Predisposing factors include the cranial irradiation and exposure to some chemicals, which are associated with an increased incidence of both astrocytomas and meningiomas. Current views on neoplastic transformation in the nervous system follow the general hypothesis of the importance of oncogenes and tumour suppressor genes. These factors operate as final common mechanisms irrespective of the initiating oncogenic process. e.g. chemical carcinogens, oncogenic viruses, irradiation, inherited mutant genes or cytogenetic defect (64). The oncogenes, which potentiate or initiate cell mitosis, may be expressed inappropriately in neoplastic cells. Oncogenes expressed in astrocytomas include c-*cis* (35,73).

Classification.

Classification of nervous system tumours is based on cell of origin, histology, and biological behaviour. When these tumours are grouped by cell of origin, it is possible to reduce the very wide variety of types found, some common, some very rare, to a relatively small number of categories. A histogenic approach to classification of brain tumours has developed over the years. Bailey and Cushing in 1926 attempted to incorporate the histological characteristics and preferential sites of origin and this was extended by Kernohan, who added histological grading based on cellular

differentiation, the presence of mitoses, endothelial proliferation and necrosis (5.45). Currently in use is the classification based primarily on the microscopic characteristics of tumours determined by an international panel of the World Health Organisation One of the problems with a histogenic classification is that the clinical pattern of central nervous system tumours may not correlate with the histological malignancy. Expanding space-occupying tumours of whatever histological type can be rapidly fatal. A histologically benign slow growing tumour may produce devastating effects in a short time because of its critical location, such as within the brain stem.

HISTOLOGICAL TYPING OF TUMOURS OF THE CNS

(According to the WHO)

Ι.	Tumours of Neuroepithelial Tissue
	A. Astrocytic tumours
	1. Astrocytoma
	Variants:
	a. Fibrillary
	b. Protosplastic
	c. Gemistocytic
	2. Anaplastic (malignant) astrocytoma
	3. Glioblastoma
	Variants:
	a. Giant cell glioblastoma
	b. Gliosarcoma
	4. Pilocytic astrocytoma

- 5. Pleomorphic xanthoastrocytoma
- 6. Subependymal giant cell astrocytoma (Tuberous sclerosis)

B. Oligodendroglial tumours

- 1. Oligodendroglioma
- 2. Anaplastic (malignant) oligodendroglioma

C. Ependymal tumours

1. Ependymoma

Variants:

- a. Cellular
- b. Papillary
- c. Clear cell
- 2. Anaplastic (malignant) ependymoma
- 3. Myxopapillary ependymoma
- 4. Subependymoma

D. Mixed gliomas

- 1. Oligo-astrocytoma
- 2. Anaplastic(malignant) oligo-astrocytoma
- 3. Others.

E. Choroid plexus tumours

- 1. Choroid plexus papilloma
- 2. Choroid plexus carcinoma

F. Neuroepithelial tumours of uncertain origin

- 1. Astroblastoma
- 2. Polar spongioblastoma
- 3. Gliomatosis cerebri

G. Neuronal and mixed neuronal-glial tumours

- 1. Gangliocytoma
- 2. Dysplastic gangliocytoma of cerebellum (Lhermitte-Duclos)
- 3. Desmoplastic infantile ganglioglioma
- 4. Dysembryoplastic neuroepithelial tumour
- 5. Ganglioglioma
- 6. Anaplastic (malignant) ganglioglioma
- 7. Central neurocytoma
- 8. Paraganglioma of the filum terminale
- 9. Olfactory neuroblastoma (Esthestoneublastoma)

Variant: Olfactory neuroepithelioma

H. Pineal parenchymal tumours

- 1. Pineocytoma
- 2. Pineoblastoma
- 3. Mixed/transitional pineal tumours

I. Embryonal tumours

1. Medulloepithelioma

- 2. Neuroblastoma
- Variant: Ganglioneublastoma
- 3. Ependymoblastoma
- 4. Primitive neuroectodermal tumours (PNETs)
- 5. Medulloblastoma

Variants:

- a. Desmoplastic medulloblastoma
- b. Medullomyoblastoma
- c. Melanocytic medulloblastoma

II. Tumours of Cranial and Spinal Nerve Sheath

A. Schwannoma (Neurilemoma, Neurinoma)

Variants:

- a. Cellular
- b. Plexiform
- c. Melanotic

B. Neurofibroma

- 1. Circumscribed (solitary)
- 2. Plexiform

C. Malignat peripheral nerve sheath tumour (Neurogenic

sarcoma, Anaplastic neurofibroma, Malignant schwannoma)

Variants:

a. Epithelioid

b. MPNST with divergent mesenchymal and/or epithelial

differentiation

c. Melanotic

III. Tumours of the Meninges

A. Tumours of meningothelial cells

1. Meningioma

Variants:

- a. Meningiothelial
- b. Fibrous (fibroblastic)
- c. Transitional (mixed)
- e. Angiomatous
- f. Microcystic
- g. Secretory
- h. Clear cell
- i. Chordoid
- j. Lymphoplasmacyte-rich
- k. Metaplastic
- 2. Atypical meningioma
- 3. Papillary meningioma
- 4. Anaplastic (malignant) meningioma

B. Mesenchymal, nonmeningothelial tumours

- 1. Benign neoplasms
 - a. Osteocartilagious tumours