DISCUSSION

This study was conducted on forty patients with bilateral basal ganglia lesions aiming to study the role of MRI in the diagnosis and evaluation of the bilateral basal ganglia lesions. We used in this study the conventional MRI and in some cases we used advanced MRI techniques when needed.

In this study, we had 22 male patients and 18 female patients, with their age ranging between 7 days and 69 years.

Bekiesinska-Figatowska M et al[4] stated that basal ganglia injury may be unilateral as: in Rasmussen encephalitis, diabetes with hemi chorea/hemi ballism and infarction, or – more frequently – bilateral in many other pathologic conditions such as HIE, Leigh disease, hepatic disease, extra pontine osmotic myelinolysis, toxic, toxoplasma infection and neoplastic like lymphoma. The findings are not specific in most of the cases and must be considered in conjunction with the clinical status and laboratory findings but knowledge of the diseases that affect basal ganglia can limit the spectrum of differential diagnosis.

The studied patients classified according to the clinical presentation into acute or chronic presentation; acute presentation was the commonest and presented by 65 %(ischemia, HIE, venous thrombosis, global ischemia, Leigh disease, extra pontine osmotic myelinolysis, toxoplasma infection, toxic and icteric encephalopathy.) Chronic presentation presented by 35% (hepatic failure, endocrine abnormality, Hallervorden-Spatz syndrome, Huntington disease, Virchow robin spaces, glutaric acidemia type I, lymphoma and metastasis.).

These findings were consistent with the findings stated by HoVB et al[19] that a simplified approach to the differential diagnosis is based on acute versus chronic conditions and radiologic manifestations. Acute conditions include hypoxia, hypoglycemia, carbon monoxide poisoning, hemolytic-uremic syndrome, osmotic myelinolysis, and encephalitis. Chronic conditions include inherited "inborn errors of metabolism" Huntington disease, and dysmyelinating diseases or acquired (squeal of acute disorders).

These findings were consistent with the findings stated by Beltz EE et al[77]that a focused approach to limit the differential diagnosis is based on patient presentation with acute or chronic symptoms.

The studied patients classified into two categories pediatric group and adult group .the pediatric group accounted for 60 % of the examined patients while the adult group presented by 40% of the examined patients.

These classification was consistent with that findings stated by Lim CC et al [61]that several publications have classified various conditions causing bilateral basal ganglia lesions according to broad groups including acquired and congenital, pediatric and adult, acute and chronic diseases.

All the presenting patients have neurological manifestations (epilepsy, movement disorder, psychomotor regression, mental deterioration and disturbed level of

consciousness.) some of them have non neurological manifestations as respiratory distress, high pitched cry, delayed puberty, ascites, lymph adenopathy and fever.

These findings were consistent with that finding stated by Lim CC et al[61]that diseases of the basal ganglia are characterized by movement disorder however, not all basal ganglia lesions associated with movement abnormalities, some acute conditions such as hypoxia or hypoglycemia may be overshadowed by coma or systemic manifestations.

The studied patients were classified according to the symmetry of the affected nuclei whether bilateral symmetrical or bilateral asymmetrical affection of the basal ganglia. The most common was the symmetrical affection of the basal ganglia representing 85% with predominance of the metabolic category which represents 70% .while the asymmetric affection of the basal ganglia representing only 15% in the form of lacunar ischemia, toxoplasmosis, Virchow Robin spaces, lymphoma and metastasis.

These findings were consistent with that finding stated by Hegde AN et al [1]that bilaterally symmetric abnormalities in the lentiform and caudate nuclei typically suggest systemic or metabolic causes, whereas asymmetric, focal, or discrete lesions affecting the basal ganglia tend to indicate involvement by infections or neoplasms.

These findings were consistent with that finding stated by Lim CC et al[61] that although systemic or metabolic diseases typically result in bilaterally symmetrical lesions, asymmetrical involvement or unilateral lesions may rarely be seen.

These findings were consistent with that finding stated by Anderson JC et al [3]that lacunar infarcts are usually asymmetric.

The final suggested diagnosis reached by conventional MRI and MR spectroscopy is seven patients of Leigh disease , six patients of HIE, five patients of (icteric encephalopathy and hepatic failure.),three patients of (toxic encephalopathy), two patients of (Virchow Robin spaces and glutaric acidemia type I) and one patient of the following (Huntington disease, Hallervorden-spatz syndrome, endocrine abnormality, extra pontine osmotic myelinolysis, global ischemia, toxoplasma infection, ischemia, venous thrombosis, metastasis and lymphoma.)

Metabolic causes diagnosed in twenty eight patients constituting 70% of the present study (Leigh disease, hepatic failure, icteric encephalopathy, hypoxia, Glutaric acidemia type I, extra pontine osmotic myelinolysis, endocrine abnormality namely hypopituitarism). Toxic causes diagnosed in three patients constituting 7.5 % of the present study (CO,calmipam ,unknown substance.).Neoplastic causes diagnosed in two patients constituting 5 % of the preset study (lymphoma, metastasis.). Degenerative causes was diagnosed in two patients constituting 5% of the present study(Huntington disease, Hallervorden-Spatz syndrome.) inflammatory causes in one patient constituting 2.5% of the present study (toxoplasmosis).Vascular causes diagnosed in two patients constituting 5% of the present study (ischemia, venous thrombosis.),and dilated perivascular spaces diagnosed in two patients constituting 5% of the present study (Virchow Robin spaces.).

These findings were consistent with the findings stated by Lim CC et al[61] who identified the causes of bilateral basal ganglia abnormalities on MRI as acute toxic insult (carbon monoxide ,cyanide, Methanol, Hydrogen sulphide), while chronic toxic insult were

manganese and methyl benzene.Acute metabolic causes were hypoxic-ischemic encephalopathy, hypoglycemia, hyperglycemia ,osmotic myelinolysis, haemolytic uremic syndrome. While chronic metabolic causes were Leigh disease, Kearns-Sayre syndrome, Wilson's disease, hypoparathyroidism, liver disease, Tay-Sachs disease, Glutaric acidemia, methylmalonic acidemia. Acute vascular causes were deep cerebral vein thrombosis, hypertensive crisis, and cerebral thromboembolism.Acute infection as toxoplasma infection, while chronic infections as Creutzfeldt-Jakob disease and congenital infections. Hereditary/Degenerative causes were Hallervorden-Spatz disease, Huntington's disease and neurofibromatosis type I.

Clinical and laboratory correlation was done to confirm the MRI diagnosis in thirty eight patients (95%); while in two patients (5%)the diagnosis was confirmed by the histo-pathological examination (lymphoma and metastasis.)

These findings were consistent with the findings stated by Hegde AN et al [(1)]that the diagnosis is not straight forward, and the correlation of typical imaging with clinical and laboratory data can help to make the correct diagnosis.

The present study includes seven patients with Leigh disease constituting about 17.5% of the total patients with an age averaging 3years. All presented with acute illness following an attack of fever in the form of psychomotor regression seizures, nystagmus and dystonia. The MRI findings in five patients were bilateral symmetrical T2, FLAIR hyperintense signal in the caudate and lentioform nuclei (three of them showed involvement of the cerebral peduncle bilaterally with two of them showing involvement of the periaqueductal region.), with restricted diffusion and detectable lactate peak in MRS. One of the remaining two cases showed T1hypointense signal and T2 hyperintense signal within the globus pallidus with no diffusion restricted but the MRS showed detectable lactate peak. The last case showed T1 hypointense signal, T2 hyperintense and FLAIR heterogeneous signal within the caudate and putamen nucleus with restricted diffusion and detectable lactate peak in MRS. Laboratory correlation of the seven cases revealed lactic acidosis, elevated lactate /pyruvate ratio and elevated lactic acid in CSF.

These findings were consistent with the findings reported in a study by Kartikasalwah et al[(78)] where the diagnostic criteria of Leigh disease were (a) progressive neurological disease with motor and intellectual developmental delay (b)signs and symptoms of brainstem and/or basal ganglia disease(c) raised lactate levels in blood and/or(CSF) and (d) characteristic symmetric necrotic lesions in the basal ganglia and/or brainstem .The most characteristic findings are bilateral, symmetric hyper intensities in the basal ganglia, thalamus, substantia nigra, and brainstem nuclei on T2-weighted MRI.

These findings were consistent with the findings reported in a study by Bekiesinska-Figatowska et al[(4)]where Leigh disease affects infants and children and involve the basal ganglia; putamen nucleus in particular and the sub thalamic nuclei and brain stem. The lesions are T2-/FLAIR and often DWI hyperintense. MR spectroscopy (MRS) shows typically lactate elevation as the children have lactic acidosis.

The present study included two patients of glutaric acidemia type I constituting about 5 % of the total patients presented with chronic illness in the form of macrocephaly, seizures, dystonia and chorea.MRI examination showed bilateral symmetrical T2, FLAIR hyperintense signal within the basal ganglia (lentiform and caudate nuclei) associated with

the characteristic wide operculae "bat wing" dilatation of Sylvian fissures and temporal hypoplasia, one of the two patients shows increased T2 signal of the periventricular white matter and atrophic basal ganglia with widened lateral ventricle .

Brismar J et al [79]found that the finding of very widely open operculae suggests glutaric acidemia type I, and if combined with basal ganglia lesions are almost pathognomonic, especially in a child with macrocephaly ,and also found that white matter described as of diffusely low density on CT or high T2 intensity on MR.

Singh Pet al[80] found that features of glutaric acidemia are widening of the Sylvain fissures, mesencephalic cistern and enlarged pre-temporal subarachnoid spaces as well as hyperintense basal ganglia , deep and subcortical white matter.GA-1 should be considered in infant or young child with acute encephalopathy in the presence of macrocephaly and extrapyramidal manifestations.

The present study included five patients with chronic hepatic failure constituting about 12.5% of the total patients of with an age above 45 years. All presented with chronic liver illness with super added neurological symptoms in the form of ataxia, tremors and dysarthria. The MRI findings in five cases were bilateral symmetrical T1 hyperintense signal in the globus pallidus, two of them showed affection of the deep and periventricular white matter in the form of T2 and FLAIR hyperintense signals as well as pituitary gland affection in the form of T1 hyperintense signal.

Rovira A et al[33] stated that classic MR imaging abnormalities include high signal intensity in the globus pallidum on T1 likely due to manganese deposition, and an elevated glutamine/glutamate peak coupled with decreased myo-inositol and choline signals on MR spectroscopy, recent data have shown that white matter abnormalities, also related to increased CNS ammonia concentration.

The present study included six patients with hypoxic ischemic encephalopathy constituting about 15 % of the total patients ,one patient of profound preterm HIE ,one acute profound term HIE, one sub-acute term profound HIE, one prolonged profound term HIE,one pediatric age HIE and one adult HIE. HIE have different pattern due to different age groups and severity of hypoxia; first the profound preterm HIE shows basal ganglia and thalamic affection in the form of T1 iso intense signal, T2 and FLAIR hyperintense signal with restricted diffusion associated with encephalomalacic changes of the PVWM and DWM, sub cortical gliosis and atrophic changes.

Hegde AN et al[1] stated that neuroimaging findings depend on the severity of the insult, time of the study and patient age, with neonatal asphyxia being a special situation with a characteristic distribution of lesions in the immature brain .

These findings were consistent with the findings of Chao CP et al[81] that due to the difference in the brain maturity at time of insult, severity of hypotension, and duration of insult, there are four patterns of brain injury. In preterm neonates, mild hypotension causes periventricular injury; severe hypotension results in infarction of the deep gray matter, brainstem, and cerebellum. In term neonates, mild hypotension causes parasagittal cortical and subcortical injury; severe hypotension causes characteristic injury of the lateral thalami, posterior putamina, hippocampi, cortico-spinal tracts, and sensorimotor cortex.

One patient (2.5%) of acute profound term HIE, shows involvement of the basal ganglia, perirolandic cortex and posterior limb of the internal capsule, ventro lateral aspect of the thalamus, with T1,T2 and FLAIR hyperintense signal with restricted diffusion. Another patient (2.5%) of sub-acute profound term HIE, shows involvement of the basal ganglia, perirolandic cortex and posterior limb of the internal capsule with T1, FLAIR hyperintense signal and T2 hypointense signal with non-restricted diffusion (pseudo normalization).

These findings were consistent with that reported by Ghei SK et al [82] that in profound hypotension shunting to vital structures is inadequate and parenchymal injury seen in the ventro-lateral thalami, posterior putamina, cortico-spinal tracts, and hippocampi. Diffusion-weighted (DW) imaging useful in the early days depicts restricted diffusion that gradually increases over the next several days; DW imaging may be falsely negative during the first hours, 1 week after the hypoxic event, a phenomenon known as pseudo normalization. On T1-weighted images during days 3–7, abnormal areas of hyperintensity are seen in the postero-lateral putamina, ventro-lateral thalami, and cortico-spinal tracts and the normal hyperintense focus seen in the posterior limb of the internal capsule may be lost, known as the absent posterior limb sign. On T2-weighted images the normal hypointense foci seen in the postero-lateral putamina, posterior limb of the internal capsule, and ventro-lateral thalamus become indistinct or abnormally iso- or hyperintense relative to adjacent gray matter. If the injury is more pronounced, the caudate head, globi pallidi and a larger area of the putamina and thalami may be involved. After the first few days, the areas of hyperintensity gradually become hypointense as edema resolves and mineral deposition occurs, with atrophy of the affected structures.

One patient (2.5%) of prolonged profound HIE presented with basal ganglia and thalamic affection in the form of T1 hyperintense signal, T2 and FLAIR hypointense signal with restricted diffusion, associated with encephalomalacic changes of the cortex, sub cortical and periventricular white matter.

These findings were consistent with that reported by Ghei SK et al [82]that in prolonged HII episode with a severe reduction in cerebral perfusion or hypoxia, the clinical and imaging manifestations are catastrophic. There is widespread injury to the basal ganglia and thalami and diffuse involvement of the cortex and white matter, a severe pattern known as total brain injury that encompasses both the basal ganglia–thalamus and peripheral patterns of injury.

The other two patient (one pediatric age and other adult age) presented also with acute presentation of asphyxia, D.L.C. and epilepsy. The MRI reveals bilateral symmetrical T2 and FLAIR hyperintense signal with restricted diffusion, one of them shows T1 hypointense signal within the swollen basal ganglia and the other shows T1 isointense signal with normal sized basal ganglia, with no cortical, thalamic, brain stem or cerebellar affection…features of hypoxic ischemic encephalopathy.

Hegde AN et al [1]stated that in adults severe HIE characteristically affects the gray matter structures, including the cerebral cortex, basal ganglia, and hippocampi. The thalamus and cerebellum may also be affected, the earliest finding at MR imaging is restricted diffusion. T2-weighted images demonstrate subtle increased signal intensity and swelling of the affected areas.

Bekiesinska-Figatowska M et al [4]stated that hypoxic–ischemic brain injury in term newborns presents as symmetrical involvement of the basal ganglia (and/or thalami) with signal hyperintensity in all the sequences (T1, T2, FLAIR and DWI) and swollen appearance of the affected structures . In adult, it may cause similar brain injury, involvement of the basal ganglia and cerebral cortex and the increased volume of basal ganglia are not observed in the adults.

The present study included one patient with Global ischemia (2.5%) of 27 years old male presented with acute DLC and coma with history of drug abuse; MRI revealed diffuse gyral swelling, edema of both cerebral hemispheres, hippocampal regions and bilateral symmetrical affection of the caudate and putamen nuclei in the form of T1 hypointense signal, T2 and FLAIR hyperintense signal with restricted diffusion.

These findings were consistent with the findings stated by Hegde AN et al[1] that in adults severe HIE characteristically affects the gray matter structures, including the cerebral cortex, basal ganglia, and hippocampi. The thalamus and cerebellum may be affected, but the brainstem and cerebral white matter are spared the earliest finding at MR imaging is increased signal intensity of the affected areas on diffusion-weighted images. Conventional T2-weighted images demonstrate increased signal intensity and swelling of the affected areas.

These findings were consistent with the findings stated by Bekiesinska-Figatowska M et al [4] that HIE in an adult may causes involvement of the basal ganglia and cerebral cortex that is typical due to high metabolic activity of the gray matter in adults. The increased volume of basal ganglia is not observed in the adults. MRI has a prognostic value: extensive lesions, cortical affection and restricted diffusion indicate poor prognosis.

The present study included one patient with old ischemia /lacunar infarct (2.5%) of 68 years old presented with right hemiplegia.MRI was done and reveals bilateral asymmetrical affection of the basal ganglia (left body of caudate and right putamen.) in the form of T1 and FLAIR hypointense signal and T2 hyperintense signal. This lacunar infarct surrounded by hyperintense signal in the FLAIR sequence that differentiates it from the Virchow-Robin spaces. This is associated with right sub-acute thalamic hematoma and chronic amyloid angiopathy.

These findings were consistent with the findings stated by Bekiesinska-Figatowska M et al [4]that ischemia of the caudate and lentiform nuclei, are usually unilaterally. The signal characteristic is typical of an ischemic focus, with early diffusion restriction followed by T2 and FLAIR hyperintensity and T1 hypointensity.

These findings were consistent with the findings stated by Anderson JC et al[3] that the vessels supplying the basal ganglia are largely end vessels and thus are susceptible to infarction. Lacunar infarcts are usually asymmetric, have typical imaging characteristics of infarcts on MRI.An acute infarct will have high signal on diffusion; develop hyper intensity on T2-weighted images and Hypointensity on T1-weighted images

The present study included one patient (2.5%) of child age with extra pontine osmotic myelinolysis constituting about 2.5% of the total cases; MRI was done and revealed bilateral symmetrical T 2, FLAIR hyperintense signal in the basal ganglia and

restricted diffusion as well as FLAIR hyperintense cortical areas along the right parieto-occipital region.

These findings were consistent with that founded by Karakaş HM et al[83] that osmotic demyelination syndrome (ODS) is rare acute process involves the pons (central pontine myelinolysis) and other locations at the center of both thalami and at the posterior parts of lentiform nucleus (extra pontine myelinolysis) follow osmotic stress .on T2-weighted MR images confluent hyper intense areas in the center of the pons whereas its periphery was spared. In the acute phase MRI revealed pontine and extra pontine hyperintensities on T2- and hypo intensities on T1-weighted images. Diffusion restriction is observed in the presented case the thalamus had a decreased level of NAA and an increased level of Cho. These were consistent with neuronal loss or dysfunction and membrane synthesis that develop after the demyelinating process.

The present study included two case of Virchow Robin spaces (5%) of old age patient presented with neurological symptoms.MRI was done and revealed involutional brain changes and bilateral asymmetrical fluid signal within the basal ganglia (caudate and putamen.), With no surrounding hyperintensity signal seen in the FLAIR differentiating it from the lacunar infarct.

These finding were consistent with that stated by Bekiesinska-Figatowska M et al[4]that dilated Virchow−Robin spaces frequently seen in the lentiform nuclei around the lenticulo-striate arteries. This finding often observed unilaterally. The signal intensity follows the cerebrospinal fluid on all the MR sequences. Virchow−Robin spaces are a normal finding.

These finding were consistent with that stated by Hegde AN et al[1] that the lentiform nucleus exhibit dilated Virchow-Robin perivascular spaces and bilaterally symmetric age related calcification both are normal findings and should not be confused with pathologic changes.

The current study shows three patients of intoxication (7.5%) of the total patients; the first one is 22 years male with history of CO poisoning was in a comatose state, due to possible CO poisoning. His mother stated that he could not wake for work in the morning, and had a charcoal stove in his bedroom. MRI revealed bilateral symmetrical hyperintense signal T1, T2, FLAIR with restricted diffusion within the basal ganglia notably at the globus pallidus associated with involvement of the periventricular and deep white matter. the second case was 31 years female with history of psychic trouble and suicidal attempt by intake of over dose of calmepam tablet presented to the emergency with coma ,respiratory distress and metabolic acidosis first CT was done revealed bilateral hemorrhagic lesion within the basal ganglia as well as the corona radiate and frontal lobe follow up MRI was done it shows bilateral symmetrical T1 hypointense ,T2 and FLAIR hyperintense signals in the caudate and putamen ,with hemorrhagic component in the frontal lobe .The third case was 19 years female with history of intoxication with unknown material presented with DLC, MRI was done it shows bilateral symmetrical T1 hypointense, T2, FLAIR hyperintense signal in the caudate and putamen, with no diffusion restriction.

This findings were consistent with that finding reported with Velioglu M et al [84]that MRI demonstrates symmetrical globus pallidus and white matter changes in most patients

with CO poisoning . The temporal, parietal, and occipital lobes are usually affected, with asymmetrical cortical and subcortical lesions.Damage to only the cerebellum is rare unless multiple areas of the cerebellum are involved.

This finding was consistent with the finding stated by Hegde AN et al[1] that Patients usually present with acute cognitive impairment or coma after accidental exposure or attempted suicide. Carbon monoxide has a propensity to affect the globus pallidus T2 prolongation in the basal ganglia is typical in acute poisoning, often with restricted diffusion on diffusion-weighted MR images. In carbon monoxide poisoning T1 shortening may be encountered.

The present study include one patient of toxoplasmosis (2.5%) of 52 years old HIV patient presented with ataxia seizures fever and lymphadenopathy, MRI was done and revealed multiple small ring enhancing focal lesion in the basal ganglia (right putamen and left caudate), cerebellum, thalamus, crus cerebri and the fronto-parital lobes sparing the periventricular region and the corpus callosum. The lesions show T1hypointense signal and T2 hyperintense signal surrounded by edema showing no diffusion restriction. Laboratory investigation was done and reveals positive anti-toxoplasma Ig G antibodies.

This finding consistent with that finding stated with Hegde AN et al[1] that Cerebral toxoplasmosis is an opportunistic infection in immune compromised patients, such as (HIV) infection, CNS involvement leads to fever, headache, and confusion, coma, focal neurologic deficits, and seizures. Imaging reveals multiple focal lesions in the basal ganglia and lobar gray matter–white matter junctions on T2 lesions are typically hypo to iso intense, usually with prominent mass effect and vasogenic edema. Hemorrhagic lesions hyper intense on T1-weighted images and are hypo intense on gradient-recalled echo images.After contrast injection, nodular or ring enhancement is typically noted .MR spectroscopy typically demonstrates lipid breakdown products without elevated choline levels.

This finding consistent with that finding found by Dietrich U et al [85]that MRI usually reveals multiple lesions in the basal ganglia and the cortico-medullary junction of the cerebral and cerebellar hemispheres. The centre of the lesions is coagulation necrosis and gives low or iso intense signal on T1- and iso intense or high signal onT2-weighted images. The periphery of the lesions is an accumulation of encysted organisms. Inflammatory and vascular changes lead to the formation of iso intense or high-signal rings which may show contrast enhancement. Nodular enhancement is seen in smaller lesions and in lesions without necrosis. In patients with AIDS cerebral toxoplasmosis is usually seen as enhancing lesions, but non enhancing or hemorrhagic foci may be present.

The present study reveals one patient of Hallervorden-Spatz disease (2.5%) of 5 years female presented with dystonia with oro-mandibular symptoms dysarthria and ataxia MRI was done and revealed bilateral symmetrical globus pallidus T2 and FLAIR hypo intensity sparing antero-medial hyper intense foci…." eye of the tiger sign". More manifest on Diffusion weighted images due to T2 black out effect….features sequel to focal iron deposition while serum and CSF iron are normal.

This finding was consistent with the finding stated by Asumal KB et al [86]that Hallervorden-Spatz syndrome (PKAN) an autosomal recessive disorder and it is a subset of neuro-degeneration with brain iron accumulation (NBIA).Clinical presentation is rigidity,

dysarthria, dystonia, retinitis pigmentosa, progressive mental retardation, cognitive impairment, spasticity, tremors. T2 images demonstrates hyperintense changes in anterio medial part of globus pallidus and hypointense lateral part of globus pallidus and pars reticulata of sub stantia nigra; associated T2 hypointensity can manifest as hyperintensity if there is inflammation and demyelination. SWI/T2* - shows low signal in corresponding areas from iron deposition.MR spectroscopy shows decreased NAA peak due to neuronal loss and may show increased myoinositol.

This finding was consistent with the finding reported by Kamate M, et al[87] that (MRI) of brain revealed bilateral symmetrical hypointensity in the globus-pallidus with central hyperintensity, giving an 'eye of tiger' appearance on T2 weighted and FLAIR images. Retinitis pigmentosa in eyes and characteristic MRI findings gave the diagnosis of pantothenate kinase associated neuro degeneration (PKAN).

This finding is consistent with that finding stated by Hegde AN et al [1]that In NBIA, the diagnostic MR imaging feature is bilateral hypointensity in the globus pallidus at T2-weighted imaging, which correlates with iron accumulation observed at pathologic examination. Patients with pantothenate kinase–associated neuro degeneration with the PANK2 mutation demonstrate the "eye-of tiger sign," with a high-signal-intensity center surrounded by the more typical hypo intensity in the globus pallidus. The eye of tiger sign is not seen in PANK2 mutation–negative patients.

The present study include one patient of Huntington disease (2.5%) of 3.7 years female presented with psychomotor regression ,ataxia ,rigidity, chorea ,dystonia and epilepsy. MRI was done and revealed bilateral symmetrical T1 and FLAIR hypo, and T2 hyper intense signal involving the heads of caudate nuclei along with the putamina on both sides which show reduced volume with consequent characteristic isolated enlargement of the frontal horns of the lateral ventricles…..features matching with inherited neurodegenerative disease, likely juvenile Huntington disease.

This findings are consistent with that stated by Ho VB et al [60]that the main radiologic feature of HD is caudate atrophy, can be quantified by the use of ratios that compare the inter caudate distance (CC) to other internal standards most commonly, frontal horn (FH) width and calvarial width (inner table [IT]). In HD, there is an increase in CC out of proportion to FH or IT, which results in a decrease in the FH/CC ratio and an increase in the CC/IT ratio (bi caudate ratio).

The present study include five patients of kernicterus(12.5%) presented in the neonatal period with elevated bilirubin level, jaundice and abnormal movement MRI revealed bilateral symmetrical affection of the globus pallidus and the sub thalamic nuclie in the form of T1 increased signal.

These findings are matching with the findings stated by causkun A et al [52]that kernicterus is a neurological syndrome resulting from the deposition of unconjugated bilirubin in brain cells. Resulting from preferential deposition of bilirubin in the globus pallidus (GP), sub thalamic nucleus, hippocampus, putamen, thalamus, and cranial nerve nuclei (III, IV, and VI) .MRI revealed symmetric, high intensity signal in the bilateral globus pallidus on T1and T2 weighted images is the most characteristic.

The present study includes one patient (2.5%) of metastasis with a known primary with confirmatory diagnosis by the stereotactic biopsy. Presented with headache .MRI revealed multiple scattered focal brain lesions within the cerebral hemispheres as well as the basal ganglia showing T2, FLAIR hyperintense signal, T1 hypointense signal and heterogeneous post contrast enhancement, with minor foci of marginal restrictions on diffusion.

These findings are matching with the findings stated by Oprişan A et al[76] that intra cerebral mass in patients with known malignant tumor strongly suggests brain metastases. In 70% imaging demonstrates more than one lesion. Cerebral metastases mostly occurs at the junction of cortex and underlying white matter, roughly 80% being located in the arterial distribution zones of the cerebral hemispheres, 3% in the basal ganglia, and 15% in the cerebellum. MRI revealed diminished signal on T1and increased signal on T2-weighted images. T1-weighted with contrast reveals ring, punctuate or solid enhancement. Some of them, especially with large size, have central necrosis, and hemorrhage.

The present study includes one patient (2.5%) of primary CNS lymphoma with confirmatory diagnosis by the stereotactic biopsy. Presented with headache and epilepsy.MRI revealed multiple hypointense signal in T1, T2 and FLAIR sequences with homogenous post contrast enhancement and restricted diffusion involving the periventricular region and body of caudate.

These findings are matching with that findings stated by Dina TS et al [69-71]That lymphoma in MRI presented as multiple lesions involving the deep, periventricular white matter, corpus callosum, and basal ganglia. Periventricular location and sub ependymal spread distinguish it from toxoplasmosis. MR spectroscopy shows elevated choline levels. The high attenuation of primary CNS lymphoma at CT and its iso- or hypo intensity at T2-weighted imaging attributed to high tumor cellularity. Solid enhancement typically occurs in immune-competent patients, whereas lesions with ring enhancement and central necrosis occur in AIDS patients.

The studied patients were classified according to the associated radiological abnormality with the basal ganglia lesions into association related to the same pathology or non-related and others have no association at all.

This finding is consistent with that finding stated by Hegde AN et al [1]that in systemic and metabolic abnormality of the basal ganglia bilaterally ,careful assessment of the brain abnormalities that occur simultaneously outside the basal ganglia is important.

SUMMARY

This study aimed to study the role of Magnetic Resonance Imaging in the evaluation of bilateral basal ganglia lesions. It was conducted upon forty patients having definite or suspicious basal ganglia lesions.

All the patients were subjected to full history taking and thorough clinical examination. Magnetic resonance imaging was performed on 1.5 T MR System using a standard head coil. Imaging included conventional MRI and advanced MRI if needed.

Many diseases present as basal ganglia abnormalities.MRI allows the detection of basal ganglia injury.

Knowledge of patient age, onset and clinical course of the disease with MRI is a powerful tool to establish the diagnosis.

The basal ganglia lesions were identified on the T1WI, T2WI and FLAIR. Then determine being unilateral or bilateral. Unilateral as: in Rasmussen encephalitis, diabetes with hemi chorea/hemi ballism and infarction, or more frequently bilateral in many pathologic conditions.

Bilateral lesions then classified according to the onset of the clinical presentations into acute and chronic, acute onset as in icteric encephalopathy, toxic (CO- calmepam, unknown), toxoplasma infection, Leigh disease, extra pontine osmotic myelinolysis, venous thrombosis, global ischemia, HIE and ischemia. Chronic onset in hepatic failure, endocrine abnormality hypopituitarism, Hallervorden -Spatz syndrome, Huntington disease, Virchow Robin spaces, glutaric acidemia type I, lymphoma and metastasis.

Basal ganglia lesions also classified according to age into pediatric age group and adult group. The pediatric age group are HIE, venous thrombosis, Leigh disease, extra pontine osmotic demyelination, glutaric acidemia type I, endocrine abnormality (hypo pituitarism.), icteric encephalopathy, Huntington disease and Hallervorden -spatz syndrome. The adult group are HIE, global ischemia, ischemia, hepatic failure, Vichow Robin spaces, toxoplasma infection, toxic causes and neoplastic (lymphoma and metastasis.).

Laboratory and histo-pathological correlation confirm the diagnosis. These include serologic studies or immunoassays for toxoplasmosis; and determination of serum sugar levels for hypoglycemia and hyperglycemia; serum sodium levels and osmolality for osmotic myelinolysis; lactate levels in the serum and CSF ,serum lactate pyruvate ratio for Leigh disease, metabolic acidosis for global ischemia, HIE and venous thrombosis and toxic causes ,elevated carboxy haemoglobin level and metabolic acidosis for CO poisoning, elevated serum ammonia and liver enzymes for hepatic failure and histo-pathological correlation for confirmation of lymphoma and metastasis.

Bilateral lesions then classified according to the symmetry of the basal ganglia nuclei affection. Asymmetrical affection noted in limited lesions as in lacunar ischemic infarct, toxoplasma infection, dilated Virchow Robin spaces and neoplastic lesions (metastasis and lymphoma.).Symmetric lesions frequently noted with predominance of the metabolic causes (HIE, global ischemia, Leigh disease, icteric encephalopathy, chronic hepatic disease, glutaric acidemia type I, extra pontine osmotic myelinolysis and endocrine abnormality).Toxic causes (CO poisoning, calmepam toxicity, unknown substance) and degenerative causes in (Hallervorden-Spatz syndrome and Huntington disease.).

The basal ganglia associated with thalamus involved in HIE (profound preterm HIE, profound term HIE), ischemia, deep cerebral venous thrombosis and toxoplasma infection.

Basal ganglia affection without thalamic affection caused by toxic poisoning, liver disease, Huntington disease, glutaric acidemia type I and Hallervorden-Spatz syndrome which characterized by bilateral symmetrical globus pallidus T2 and FLAIR hypointensity sparing antero-medial hyper intense foci" eye of tiger sign", sequel to focal iron deposition, (with normal level of serum and CSF iron.)

Detection of associated abnormalities in the brain other than the basal ganglia and thalamus is helpful in narrowing the differential diagnosis. These include cortical involvement in hypoxia, and white matter abnormalities in poisoning (CO, calmepam toxicity). The brain stem may be involved in Leigh disease, osmotic myelinolysis.

Leigh disease characterized by basal ganglia affection and associated with cerebral peduncle and periaqueductal affection with detectable lactate peak in MRS.

Huntington disease characterized by basal ganglia affection associated with characteristic widening of the frontal horn of the lateral ventricle bilaterally due to atrophy of the caudate nuclei.

Hepatic disease characterized by the globus pallidus affection (hyperintense signal in T1 sequence) and associated with DWM, PVWM affection, as well as hyper intense signal of the pituitary gland.

CO poisoning characterized by the globus pallidus affection associated with affection of DWM, PVWM with history of CO poisoning.

Preterm HIE characterized by affection of the DW, PVWM in the form of encephalomalacic changes, subcortical gliosis, thalamic affection and basal ganglia affection.

Profound term HIE characterized by affection of the posterior limb of the internal capsule (absent posterior limb sign) ventro-lateral aspect of the thalamus, perirolandic cortex and basal ganglia affection, in prolonged profound is wide spread affection of the basal ganglia,thalamus,cortex and white matter.

Glutaric acidemia type I characterized by bilateral temporal lobe hypoplasia, dilated sylvian fissures bilaterally as well as the CSF spaces and hyperintense signal of the DWM in patient with abnormal movement and macrocephaly.

In extra pontine osmotic myelinolysis, the symmetrical basal ganglia affection associated with cortical affection along the parieto-occipital region.

Toxoplasma infection characterized by multiple ring enhancing lesions at the basal ganglia, cerebellum, thalamus, crus cerebri and the fronto-parietal lobes sparing the periventricular region and the corpus callosum in HIV patients. Laboratory investigation reveals positive anti-toxoplasma Ig G antibodies.

In hypopituitarism decreased height of the pituitary gland.

In dilated Virchow Robin spaces, there is bilateral asymmetrical fluid signal with in the basal ganglia (caudate and putamen.), with no surrounding FLAIR hyperintensity differentiating it from the lacunar infarct.