Central Nervous System Tuberculomas – Pathological Correlation with Imaging

Submitted for MCh Neurosurgery

By

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CERTIFICATE

This is to certify that the thesis entitled "**Central Nervous system tuberculomas – pathological correlation with imaging**" is a bonafide work of **Dr. Varun Aggarwal and** was conducted in the Department of Neurosurgery, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram (SCTIMST), under my guidance and supervision.

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5/10/18

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DECLARATION

This thesis titled "**Central Nervous system tuberculomas – pathological correlation with imaging** ", is a consolidated report based on a bonafide study of the period from January 1999 to December 2011, done by me under the Department of Neurosurgery, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram. This thesis is submitted to SCTIMST in partial fulfillment of rules and regulations of MCh Neurosurgery examination.

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Introduction

Tuberculosis is a prevalent disease in developing countries. CNS tuberculosis is very rare. CNS tuberculosis involves the 10% of extra pulmonary TB cases. The spine tuberculosis include 2% of central nervous system TB. Intracranial tuberculosis usually presents as tuberculous meningoencephalitis, whereas spinal tuberculosis usually presents as tuberculous spondylitis or arachnoiditis.

CNS tuberculosis rarely presents as tuberculoma. Although central nervous system tuberculoma is treatable, delayed diagnosis is associated with severe morbidity. Widespread use of MRI has allowed more accurate and frequent detection of central nervous system tuberculoma. Drugs are indicated in patients with diffuse brain tissue involvement; however, tuberculoma is a well-defined mass lesion, allowing relatively straightforward surgical resection. Surgical resection has therapeutic use and diagnostic use. To date, the lack of large patient cohorts has precluded a proper analysis of this disease.

This study outline the patho-radiological correlation of Central nervous system tuberculomas and aims to provide some strategies for early diagnosis and treatment.

Review of literature

Tuberculosis is an annoying chronic infectious diseases. Approximately 90 lacs cases occur with 15 lacs lives claimed each year. Tubercle bacillus has infected 30% of world's population and 10% of that population will have active disease[1]. There is paucity of cases in developed country because of better socioeconomic life. Contributed factors for persistent of TB are AIDS, MDR and XDR TB, and inability to complete treatment[2]. In 2010, TB is the major health problem in India and other developing countries[1]. Central nervous system (CNS) TB relates to around 10% of all extra pulmonary TB[3]. But CNS TB represents severe type of extra pulmonary TB and has highest percentage of morbidity and mortality. Outcome of TB was mostly grave till antitubercular drugs were discovered[4]. CNS Tuberculomas accounts for 30% of all intracranial masses in endemic areas[5]. Thirty percent of intra cranial masses are tuberculomas[6]. CNS tuberculomas are formed by interaction of the host's body and the mycobacterial pathogen. Tuberculomas may cause compression symptoms like hydrocephalus and focal symptoms.

Etiology

Tuberculosis is caused by numerous species of mycobacteria depends on zoonotic vectors, geographic region and drug resistance. Total 7 types of bacteria have shown its presence in TB cases, together they called as the Mycobacterium tuberculosis (MTB) complex[7]. Any infliction of disease by bacteria apart from MTB is termed as mycobacteriosis eg M bovis. It shows growth only in aerobic medium and it is acid fast bacillus. Infected person spread infection by coughing. 9[8] The macrophages in lung are infecting by inhaled MTB.

Pathophysiology

Tuberculoma pathophysiology is complicated to understand[9]. First infected macrophages produce inflammatory mediators which leads to recruitment of peripheral macrophages and monocytes. These infected macrophages migrates to lymph nodes and disseminate to in other part of body. Enzymes in lysosome kill mycobacteria then present their antigens via major histocompatibility complex (MHC) class II to CD4+ T cells; CD4+ T-cells secrete interferon-gamma (IFN-c), which induce macrophage recruitment and enhance lysis of mycobacteria[10.] The patient body react by forming granuloma around the infected and necrosed immune cells to contain it, but viable bacteria has been demonstrated in these granulomas. This granuloma formation is though protective sometime may turn against the patient recovery. This inflammatory process may get exaggerated and produce unwanted effects. One of such effect is erosion of granuloma into the bronchus leading to its air dessimination[9.] Furthermore, the bacillus itself has many ways to protect itself from immune cells and antibiotics. One of the main mechanism is the inherent resistant nature of mycolic acid present in the cell wall. A recent study found that TB causes up regulation the fibrinolytic system, which peak during the chronic phase of disease[11] and these factors may contribute to the chronic inflammation observed in TB infection. In addition to higher risk of CNS TB in the young and patients with HIV, genetic makeup of the host and also bacterium leads to selective neurotropism. According to study of Hernandez pado et al in 2011 modelling of cerebral tuberculosis: hope for continuous research in solving the enigma of the Bottom Billion's Disease. Author reported that mutations of IL1 and IL2 receptor and variation in glycolipid gene is responsible for extra pulmonary dissemination.[12] a strong T helper cell response in confers neuroprotection, and inhibition of the response is controversial, it is believed to be a risk factor for disease. TB vaccine Bacille-Calmette-Guérin

(BCG), regulates migration and infiltration of monocytes, memory T-cells, and natural killer cells to the infection focus in order to contain it. The mechanism is not known.[13, 14]

The objective of Mendez et al. study was to find relation between body response (cytokine) and location of TB infection. They found similar levels of IL4 in all patients, lower level of IL12 and elevated levels CCL2 and decreased chemokine in patients with meningeal TB compared to renal or pulmonary TB. Recent work is mainly focused on TB genotype on different pathogenesis. Hernández-Pando et al. examined TB strains isolated from CSF of mices[12]. Virulent strains induced lower levels of IFN-c and high levels IL 4. Virulence is directly proportional to high expression of TNF and low expression of nitric oxide synthase. Epidemiologic studies demonstrated that certain TB genotypes are endemic in certain areas, probably because of evolutionary properties and host-pathogen compatibility[13, 14]. Literature is mainly available on Beijing subtype, which has been responsible for majority of outbreaks worldwide. The virulence of the Beijing strain is attributable to its capability to inhibit T helper cell and its IL production, this is mainly because of A-Crystalline antigen[15-20]. A study done by Aguilar et al demonstrated the variable virulence of Beijing strain.[21] He showed few strains were accompanied with diffuse tissue damage with decreased level of IFN and iNOS compared to few others. These data suggest variability in Beijing phenotype with respect to virulence and its transmission.[21].

TB primarily affects the lungs and disseminates via blood or lymphatics. Miliary TB is a disseminated form with numerous 1-3 mm tubercles. Miliary TB and tuberculous meningitis may be present without CNS tuberculoma formation[22]. Rich's focus, the historical name for a CNS tuberculoma, named after pathologist Arnold Rice Rich. Rich suggested that TBM is preceded by metastatic seeding of MTB within the parenchyma or meninges, followed by growth and

eventual maturation of the caseous tuberculoma into a fluid-filled tuberculous abscess and rupture of this abscess into the subarachnoid space causes TBM[23].

History and examination

Clinical features of CNS tuberculoma patients are headache, vomiting, hemiparesis or seizures, drowsiness, papilledema and mimicking other lesions of CNS. TBM may present as neck pain, back pain, projectile vomiting, fever and drowiness[7]. 5-30% of space occupying lesion in children are tuberculoma[5]. CNS tuberculomas are presents as CNS tumor in absence of active TBM and TB. There is no correlation with TBM staging with tuberculoma[24]. Tuberculoma may present in orbit, sella and suprasellar areas.

Tuberculoma causes encasement of bacteria thus making CSF sample sterile. This localized formation of granulomas leads to difficulty in getting easy culture based diagnosis. Over the time improvement in diagnostic technique has led to availability of different methods:

I) AFB staining and Culture

TBM can diagnosed by smear examination of CSF. Staining methods for AFB are Ziehl-Neelsen, Kinyoun, or auramine-rhodamine. These method can detect approximately 100 AFB/ml of CSF. CSF in ventricle has highest chance to detect bacteria. Minimum 15 ml of CSF should be taken for examination. In immunocompromised patient, bacteria can be detected even in lesser amount of CSF[25]. ATT reduce the sensitivity of AFB smear and culture. The deposit should be stained and cultured on solid or in liquid media. Solid culture media is not suitable for bacteria comparison to liquid media[26]. A tissue sample is better diagnostic than CSF. Gastric aspirates

and bone marrow aspirates may assist in detecting extra-neural tuberculosis in children. Stereotactic brain biopsy can be used for diagnosis of tubercular abscesses and atypical tuberculomas. In TBM, there is increase count of leukocytes and lymphocytes in CSF. In biochemistry, there is reduce sugar and increase protein in CSF.

II) Molecular and Biochemical Analysis

A) Tuberculin skin test (TST)

The sensitivity of TST is 10 to 50%[27, 28]. The factor effecting the TST are age of person, immune status, previous vaccination with BCG, method of administration and nutrition[29]. Positive TST does not indicate active disease[30].

B) ADA

ADA is indicative of cell-mediated immunity of the body[31]. The range of sensitivity and specificity ADA is 44 to 100% and 71 to 100%, respectively[32]. According to some studies ADA is not useful in diagnosing TBM in immunocompromised patients[33]. CSF ADA is an indicator of grave prognosis in children with TBM[34]. Differential of high value of ADA are bacterial meningitis, fungal meningitis, parasitic infection like brucellosis and lymphoid tumors[35, 36].

C) Tuberculostearic acid

Tuberculostearic acid is present in the cell wall AFB [37]. This test is costlier, not to be used routinely.

D) PCR technique

The sensitivity and specificity of nucleic acid amplification (NAA) is 56% and 98% respectivly[38]. NAA has same sensitivity as that of microscopy in TBM[39]. It is useful in detecting bacterial DNA even after the start of ATT. NAA can be positive in other varieties CNS tuberculosis[40, 41].

E) Interferon- y **release assays (IGRAs):**

IGRAs can detect the interferon-y (IFN- y). Interferon y is formed by lymphocyte against antigens, early secreted antigenic target 6 and culture filtrate protein 10. The results of IGRAs doesn't depends upon the BCG vaccination[42-44]. In IGFA, ELISA is used to find IFN-y. IFN-y is specific for MTB so IGRAs cannot differentiate between previous infection and new infection. In vitro, IFN-y had been detected in 50 percent of cases. In prognosis of the disease, IGRAs is still controversial[45]. But the use of this test is increasing in developing [46].

III) Imaging

Tuberculomas may present as single or multiple SOL in brain. Infratentorial tuberculomas are more common in children than adults[47].The factors affecting the imaging are caseous necrosis in granuloma.

A) CT scan

CNS Tuberculomas may present as hypodense (necrosis), isodense and hyperdense (protein and calcium) SOL on Computed tomographic scans. Target sign is also pathognomonic to CNS tuberculoma. Tuberculomas can show ring enhancement or homogeneous enhancement on contrast CT scan.

B) **MRI scan**

Non-caseous tuberculoma are hypointense and hyperintense on T1 and T2 weighted MRI respectively. The core of caseating granulomas may be solid (T1/T2 iso to hypointense) or liquid (T1 hypo and T2 hyper). On Contrast T1 images, tuberculoma may show both rim and homogenous enhancement. Presently appearance of target sign is non pathognomonic for tuberculoma[48].

C) **MR spectroscopy**

Tuberculomas show lipid lactate peak on spectroscopy (0.9, 1.3, 2.0 and 2.8 ppm). Lipid lactate peak depends upon the quantity of lipid in caseous necrosis[49]. Pyogenic brain abscess can be differentiated for CNS tuberculoma by presence of additional peak of valine, leucine and isoleucine[50]. Sometimes tuberculoma may show increase in choline which create suspicion of malignancy [49].

D) **Magnetization transfer imaging (MTI)**

T1 magnetization transfer images help to diagnose multiple CNS tuberculomas. Tuberculoma has lower magnetization transfer to adjacent brain parenchyma. The presence of lipid within tuberculoma is probably responsible for lowering magnetization transfer. The cysticercus granulomas has higher MT ratio to tuberculoma.[51].

E) Diffusion weighted imaging (DWI)

It is useful to diagnose abscess (infective pathology). Caseating tuberculoma with liquid center shows diffusion restriction.

F) **Dynamic contrast enhanced (DCE) MRI**

It quantify the vascularity of lesion. A study done by Gupta et al., reported that relative cerebral blood volume of cellular portion significantly correlated with cellular fraction volume microvascular density and VEGF[52].

G) **Diffusion tensor imaging (DTI)**

It can detect arrangement of tract in brain parenchyma. A study of Gupta et al. on DTI derived indices correlate with immunohistochemistry obtained matrix metalloproteinase (MMP-9) expression in cellular fraction of brain tuberculoma, has reported that DTI indices show strong

correlation with MMP-9 and these may be used as a surrogate marker of MMP-9 expression in CNS tuberculoma[53].

Management

1) Medical

Initial treatment of choice in CNS tuberculoma is anti-tuberculosis therapy. A study was done by Idris et al. on Tuberculoma of the brain 87% response rate in his cohort of 16 patients with ATT[54]. There are 5 drugs as first line defenses for tuberculosis infection namely isoniazid, rifampicin, pyrazinamide, streptomycin and ethambutol. Second line defense are ethionamide, kanamycin, cycloserine, and para-amino salicylic acid[55]. Corticosteroids are mainly indicated in modifying the immune reaction of the body favorably by decreasing adhesion and incidence of hydrocephalus. Thalidomide[47] has been tried in patient who don't respond adequately to ATT. Because of the chronicity of the tuberculous infection, ATT is generally recommended for long durations ranging from 6 moths to 18 months. ATT is continued despite patient being symptomatically better or showing radiological resolution. The incidence of CNS tuberculoma has reduced after the introduction of BCG vaccination [56].

2) Surgical

Surgical intervention is warranted if there is no response to medical management or development of complications related to mass effect or raised ICP. Raised ICP is managed with therapeutic Lumbar puncture, lumboperitoneal shunt for communication hydrocephalus and ventriculoperitoneal shunting for noncommunicating hydrocephalus[48]. Risk of infection is reduced with Endoscopic third ventriculostomy (ETV) compare to ventriculoperitoneal shunt in HIV patients. But there is risk of injury to basilar artery because basal exudates[57].

Those tuberculomas causing mass effect and not responding to medical management required surgical resection. Surgical decision is also influenced by the site of lesion, surrounding anatomy and regional practice[58]. Extent of resection is also variable. Although complete excision is desirable but seldom achieved because of attendant risk of untoward complications. Subtotal excision followed ATT is another alternative. Biopsy under neuronavigation is better and preferred over open surgical biopsy. Recent advances in endoscopic surgery has made possible to do biopsy or excision of skull base tuberculomas through endonasal transphenoidal route[59]. A study was done by Ersahin et al. demonstrated successful excision of cranial tuberculoma using stereotaxy.[60]. Tuberculomas because of its caseating nature and abscess formation with minimal blood supply, amenable to endoscopic aspiration. Personalized assessment of all patients should be done taking into account patient factors, virulence, aggression of disease, its response to ATT, location of tuberculoma before subjecting the patient to any of the surgical procedure. The risk of postoperative tuberculous meningitis also needs to be addressed which can occur after surgical intervention.

Treatment and management considerations

Up to 1/4th of medically managed TB patients may show paradoxical enlargement of tuberculoma or appearance of new lesions despite on optimal ATT. Such an unwelcomed response is mainly seen more in extra-pulmonary patients than pulmonary group. It is unlikely for a tuberculoma which has not shown any response to ATT for 2 or more years to show any response to continued ATT.[61]. The American Thoracic Society and CDC does not recommend rifampicin-pyrazinamide regimen for LTBI due to hepatic side effects and increased mortality; instead an isoniazid with rifapentine therapy under direct supervision for 12 weeks duration for patients more than 12 years.[62] Hepatitis is well established side effect of ATT which sometimes

precludes use of ATT. But few authors have advocated use of ATT after remission of hepatitis as shown by Thwaites et al[24]. Management of tuberculomas in AIDS cases is challenging with respect to diagnosis and treatment. Corticosteroids, which improves outcome in TBM may cause further immunosuppression in already compromised immune system. In AIDS cases with TB antiretroviral therapy may improve immune response of the body which may respond in an exaggerated way and cause paradoxical worsening of TB called as Immune reconstitution inflammatory syndrome[61].

Future investigation and treatment of CNS tuberculosis

Despite the basic understanding of TB, it is still most prevalent disease especially in developing countries. Most of the research is now focus on pathogenesis, drug resistance and vaccine development. Hernández-Pando et al. have successfully modelled cerebral TB infection in mice to better understand the immune mechanism, bacterial pathogenesis and to study the potential efficacy of MDR TB to ATT and oral vaccine[63.] Vaccines derived from recombinant M. smegmatis[64], and Mycobacterial antigens 85A and heat shock protein X50 has produced some immunity in mice and may one day be an alternative to BCG. Newer anti- TB drugs are being tested for efficacy and CSF penetration, and clinical vaccine and ATT trials for MDR and CNS TB are in progress.[65] Though most of the past studies shows that the immune system which comes into play when TB infection occurs is cell mediated immunity some report otherwise. Authors have shown that human gamma globulins if administered prior to TB exposure in a mouse model conferred some immunity to contraction of infection demonstrating a role for humoral immunity in protection against TB, but further studies are needed[66].

Aims and objective

To compare histopathological features of central nervous system tuberculomas with their radiological findings

Material and methods

Patient selection

A retrospective study of patient related data, who underwent surgery for CNS tuberculomas, at Sree Chitra Tirunal Institute for Medical Sciences and Technology, from 1999 to 2011 forms the basis of this study.

Imaging parameters included in the study were

1. Computerised Tomogram (contrast enhanced)
2. Magnetic Resonance Imaging: The sequences of MRI performed include - T1-weighted, T2-weighted, diffusion, perfusion, MRS and post gadolinium (0.2 mg/kg).

MRI and CT were studied by neuroradiologist and neurosurgeon in combined meets.

Histopathology specimen: All slides were reviewed by the pathologist who was blinded from the patient imaging and clinical data. Tuberculomas were diagnosed by presence of granuloma with or without caseating necrosis. Following stains were used: - haematoxylin and eosin, Ziehl-Neelson and periodic acid Schiff.

Inclusion criteria- All the patient with either CT or MRI and histogically proven cases of tuberculoma were included.

Exclusion criteria- Patients who were medically managed

Factor studied were

- Number –a) solitary, b) multiple
- Location i.e. supratentorial or infratentorial or spinal cord
- Dimensions

- Margins
- Perilesional edema
- Non contrast and contrast CT imaging features
- MRI features (T1,T2, Contrast, DWI, Perfusion and MRS)

Study was divided in 2 groups as following

Granulomas

a. Granuloma with caseating necrosis
b. Granuloma with no caseation

Statistical analysis

Microsoft word Excel 2010 and SPSS software were used for statistical analysis. A *p* value of less than 0.05 was considered significant.

Results

Patient characteristics- Demographic Profile

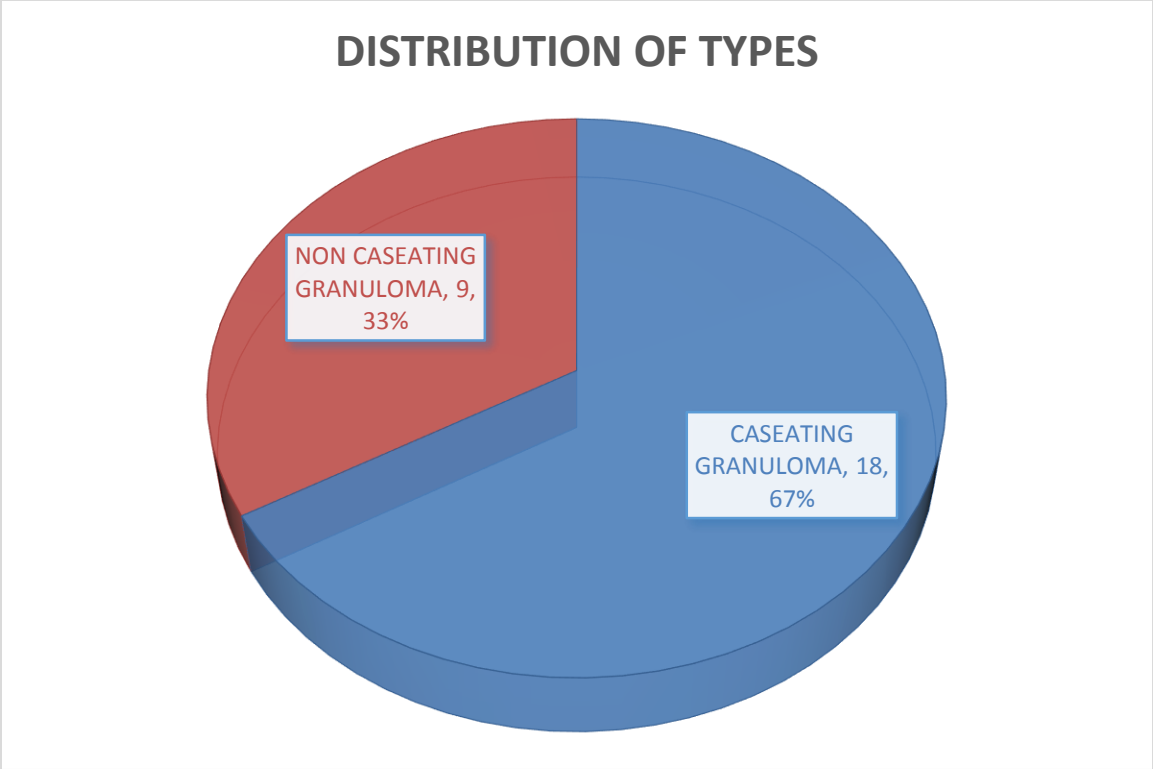
A total of 27 consecutive patients who underwent surgery (partial excision or total excision) from 1999 until 2011. Of the 27 patients in the study sample, 12 were male and 15 were female. The mean age of 30.8 years with range of age from 7-68 years. Patients presented with seizures (n-10), headache, vomiting, gait unsteadiness, motor and sensory weakness. On examination they had papilledema, cranial nerve paresis, motor and sensory weakness and cerebellar sign. The patients were negative for pulmonary TB and HIV in our study

Pathology

18 out of 27(66.7%) patient were having caseating granuloma and 9(33.3%) patients were having non caseating granuloma (Table-1 and Graph-1)

		NUMBER(N)	Percentage
1.	CASEATING GARNULOMA	18	66.7
2.	NO CASEATING GRANULOMA	9	33.3

Table 1: Showing number of cases with caseating and non caseating granuloma

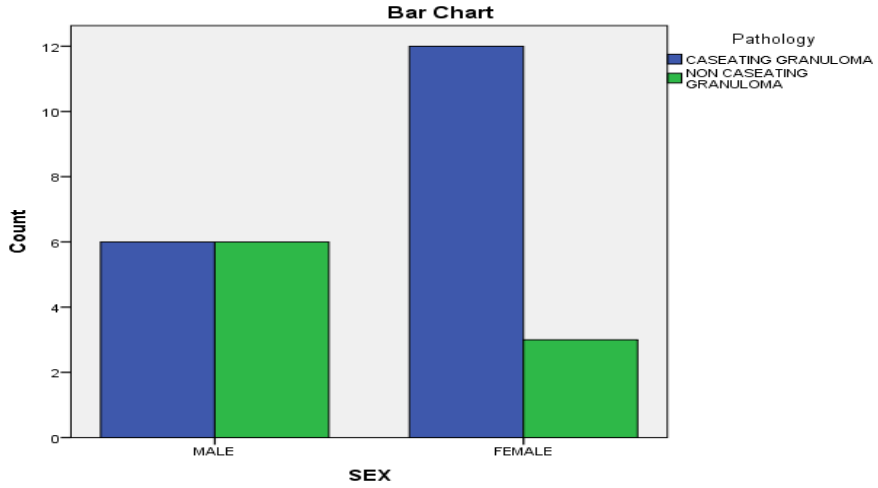


Graph 1: Showing distribution of caseating and non caseating granulomas

In our study 15 patients were female and 12 patients were male. In female patients, 12 (80%) patient had caseating granuloma and 3(20%) had non caseating granuloma. In male patients 6(50%) patient had caseating granuloma and 6(50%) patient had non caseating granuloma. P-value is 0.029(Table-2).

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
SEX	MALE	Count	6	6	12
		% within SEX	50.0%	50.0%	100.0%
		% within Pathology	33.3%	66.7%	44.4%
		% of Total	22.2%	22.2%	44.4%
	FEMALE	Count	12	3	15
		% within SEX	80.0%	20.0%	100.0%
		% within Pathology	66.7%	33.3%	55.6%
		% of Total	44.4%	11.1%	55.6%
	Total	Count	18	9	27
		% within SEX	66.7%	33.3%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	66.7%	33.3%	100.0%

Table 2- showing the percentages of the patients with different types of granulomas in male and female patients

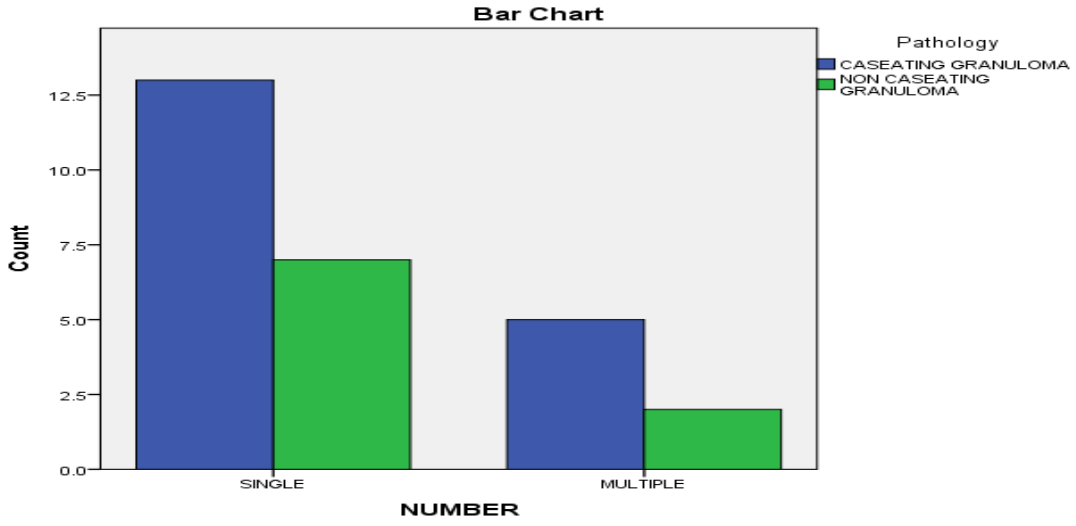


Graph-2 Showing no of cases in each category with further distribution in male and female sex

In this study 20 patients had single granuloma and 7 patient had multiple granulomas. Most of the caseating granulomas (72.2%) were single and only 27.8% were multiple. In non caseating granulomas 77.8% were single and 22.2% were multiple. P value calculation was .139 (Table-3) (Graph-3).

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
NUMBER	SINGLE	Count	13	7	20
		% within NUMBER	65.0%	35.0%	100.0%
		% within Pathology	72.2%	77.8%	74.1%
		% of Total	48.1%	25.9%	74.1%
	MULTIPLE	Count	5	2	7
		% within NUMBER	71.4%	28.6%	100.0%
		% within Pathology	27.8%	22.2%	25.9%
		% of Total	18.5%	7.4%	25.9%
Total		Count	18	9	27
		% within NUMBER	66.7%	33.3%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	66.7%	33.3%	100.0%

Table-3- Showing distribution of granulomas as single or multiple



Graph-3 Showing distribution of granulomas as single or multiple

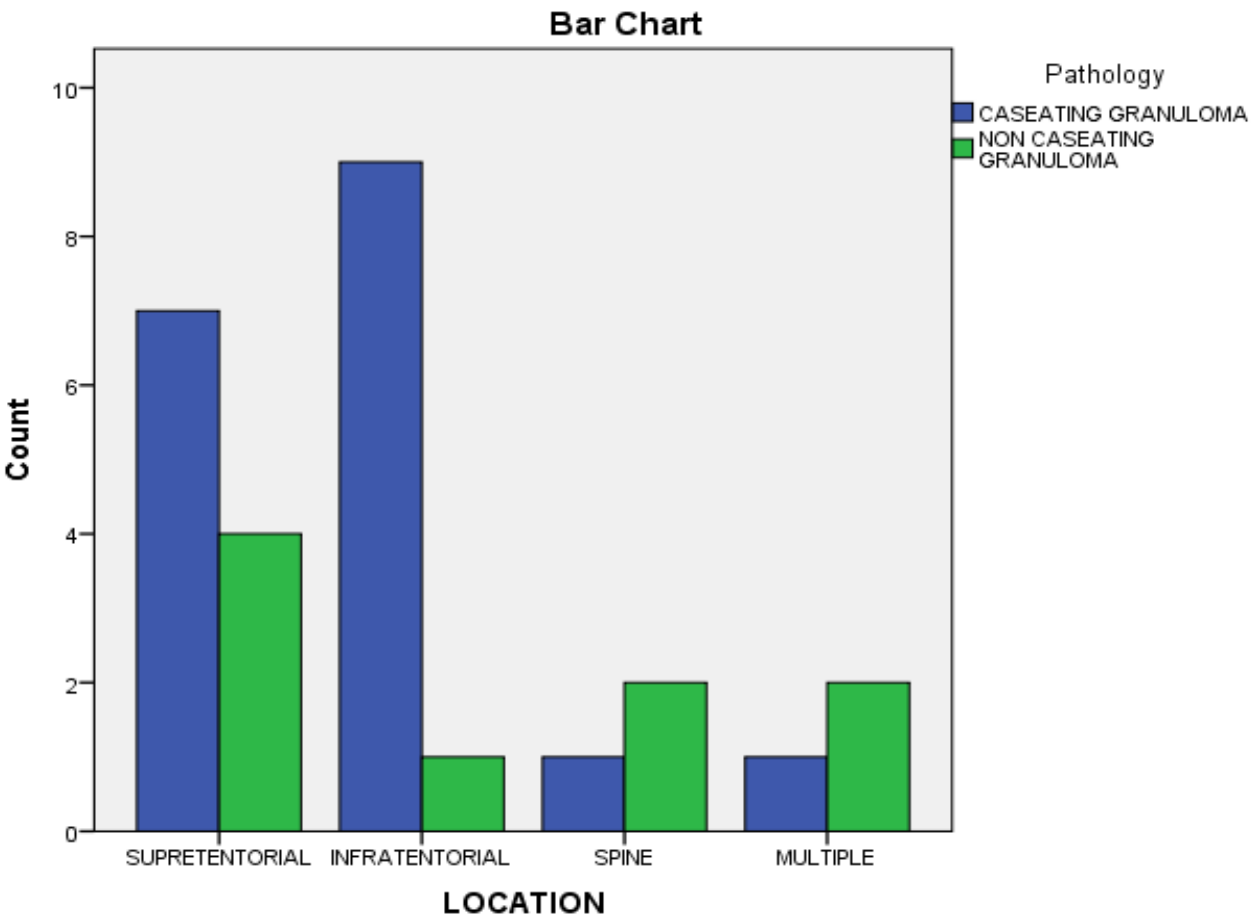
77.8% (n=21) cases of tuberculomas were >2.5cm. This study 83.3% of caseating granulomas were >2.5cm in size. 66.7% of non caseating granulomas were >2.5cm**.**

In the study, caseating granulomas were supratentorial, infratentorial (cerebellum and vermis), spine and at multiple location in 38.9%, 50%, 5.6%, 5.6% respectively. In non caseating

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
LOCATION	SUPRETENTORIAL	Count	7	4	11
		% within LOCATION	63.6%	36.4%	100.0%
		% within Pathology	38.9%	44.4%	40.7%
		% of Total	25.9%	14.8%	40.7%
	INFRATENTORIAL	Count	9	1	10
		% within LOCATION	90.0%	10.0%	100.0%
		% within Pathology	50.0%	11.1%	37.0%
		% of Total	33.3%	3.7%	37.0%
	SPINE	Count	1	2	3
		% within LOCATION	33.3%	66.7%	100.0%
		% within Pathology	5.6%	22.2%	11.1%
		% of Total	3.7%	7.4%	11.1%
	MULTIPLE	Count	1	2	3
		% within LOCATION	33.3%	66.7%	100.0%
		% within Pathology	5.6%	22.2%	11.1%
		% of Total	3.7%	7.4%	11.1%
	Total	Count	18	9	27
		% within LOCATION	66.7%	33.3%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	66.7%	33.3%	100.0%

Table-4: Showing distribution of granulomas in different parts of CNS

Granuloma were supratentorial (44.4%), infratentorial (11.1%) (Cerebellum and vermis), spine (22.2%) and at multiple location (11.1%). P value is .756 (Table-4) (Graph-4).

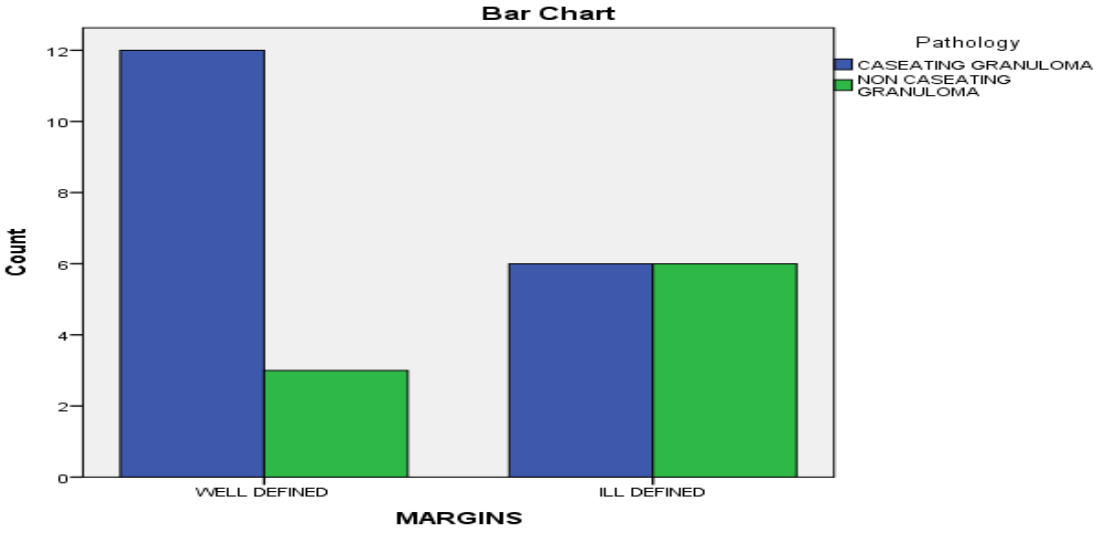


Graph 4- Showing distribution of granulomas in different parts of CNS

In our study, 12 (66.7%) patient of caseating tuberculomas had well defined margin compare to 3(33.3%) patient of noncaseating tuberculoma. Calculated P value is 0.1 which is non-significant (Table-5) (Graph-5)

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
MARGINs	WELL DEFINED	Count	12	3	15
		% within MARGINS	80.0%	20.0%	100.0%
		% within Pathology	66.7%	33.3%	55.6%
		% of Total	44.4%	11.1%	55.6%
	ILL DEFINED	Count	6	6	12
		% within MARGINS	50.0%	50.0%	100.0%
		% within Pathology	33.3%	66.7%	44.4%
		% of Total	22.2%	22.2%	44.4%
	Total	Count	18	9	27
		% within MARGINS	66.7%	33.3%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	66.7%	33.3%	100.0%

Table 5- Showing distribution of granuloma according to margins



Graph 5- Distribution of granulomas with different margins

In this study, 13(72.2%) patients of caseating granuloma and 6(66.7%) patients of non caseating granuloma were associated with perilesional edema. P value is .766(Table-6)

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
EDEMA	PRESENT	Count	13	6	19
		% within EDEMA	68.4%	31.6%	100.0%
		% within Pathology	72.2%	66.7%	70.4%
		% of Total	48.1%	22.2%	70.4%
	ABSCENT	Count	5	3	8
		% within EDEMA	62.5%	37.5%	100.0%
		% within Pathology	27.8%	33.3%	29.6%
		% of Total	18.5%	11.1%	29.6%
	Total	Count	18	9	27
		% within EDEMA	66.7%	33.3%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	66.7%	33.3%	100.0%

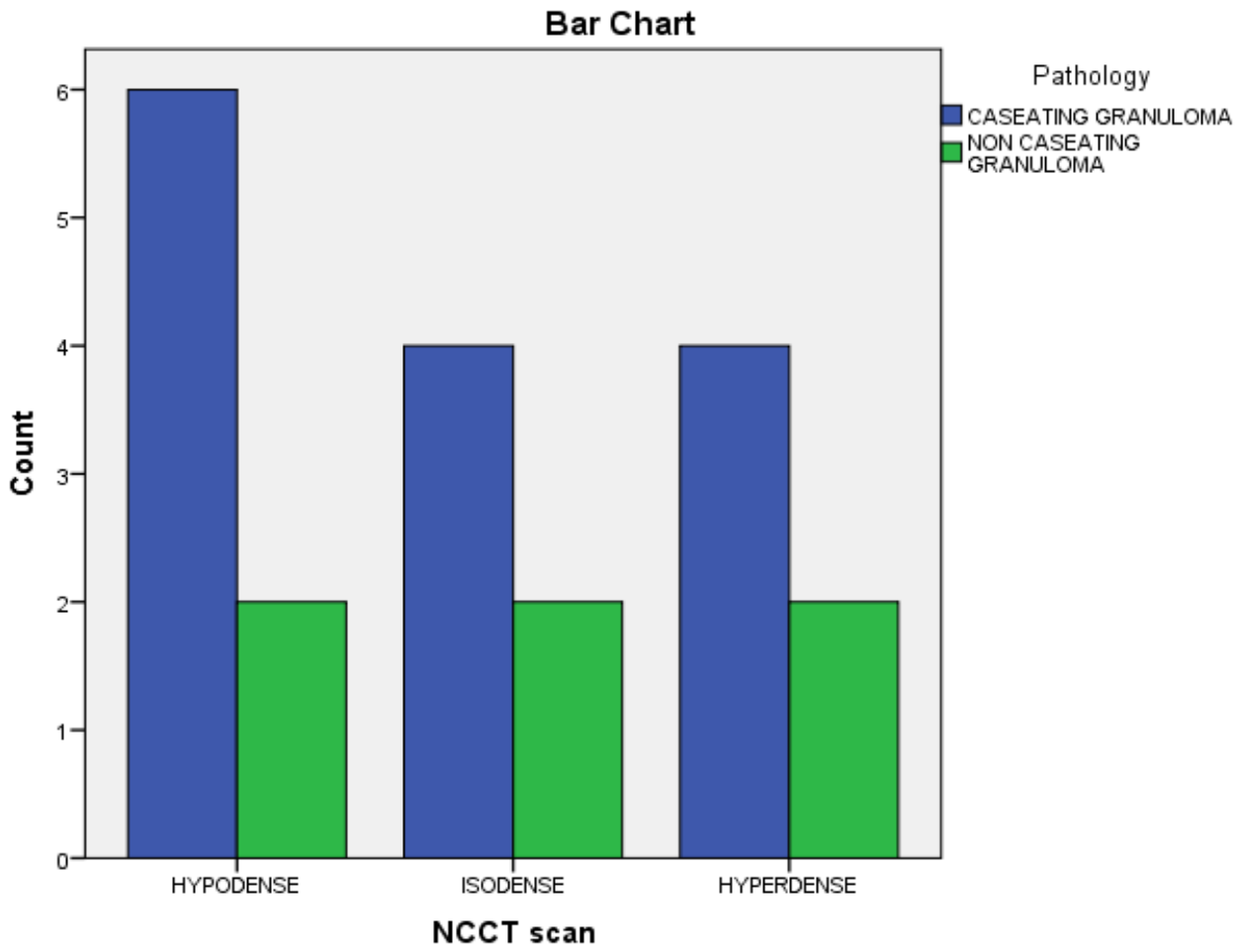
Table 6- Percentage of patients with perilesional edema in two groups

NCCT HEAD

In our study 20 patients underwent CT scan of brain. On non-contrast CT scan, caseating granuloma were hypodense (42.8%), isodense (28.5%) and hyper dense (28.5%). Non caseating granuloma were hypodense (33.3%), isodense (33.3%), hyperdense (33.3%) on non- contrast CT scan. P value is .924

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
NCCT scan	HYPODENSE	Count	6	2	8
		% within NCCT scan	75.0%	25.0%	100.0%
		% within Pathology	42.9%	33.3%	40.0%
		% of Total	30.0%	10.0%	40.0%
	ISODENSE	Count	4	2	6
		% within NCCT scan	66.7%	33.3%	100.0%
		% within Pathology	28.6%	33.3%	30.0%
		% of Total	20.0%	10.0%	30.0%
	HYPERDENSE	Count	4	2	6
		% within NCCT scan	66.7%	33.3%	100.0%
		% within Pathology	28.6%	33.3%	30.0%
		% of Total	20.0%	10.0%	30.0%
Total		Count	14	6	20
		% within NCCT scan	70.0%	30.0%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	70.0%	30.0%	100.0%

Table 7- Showing imaging finding of CT scan for different types of granulomas

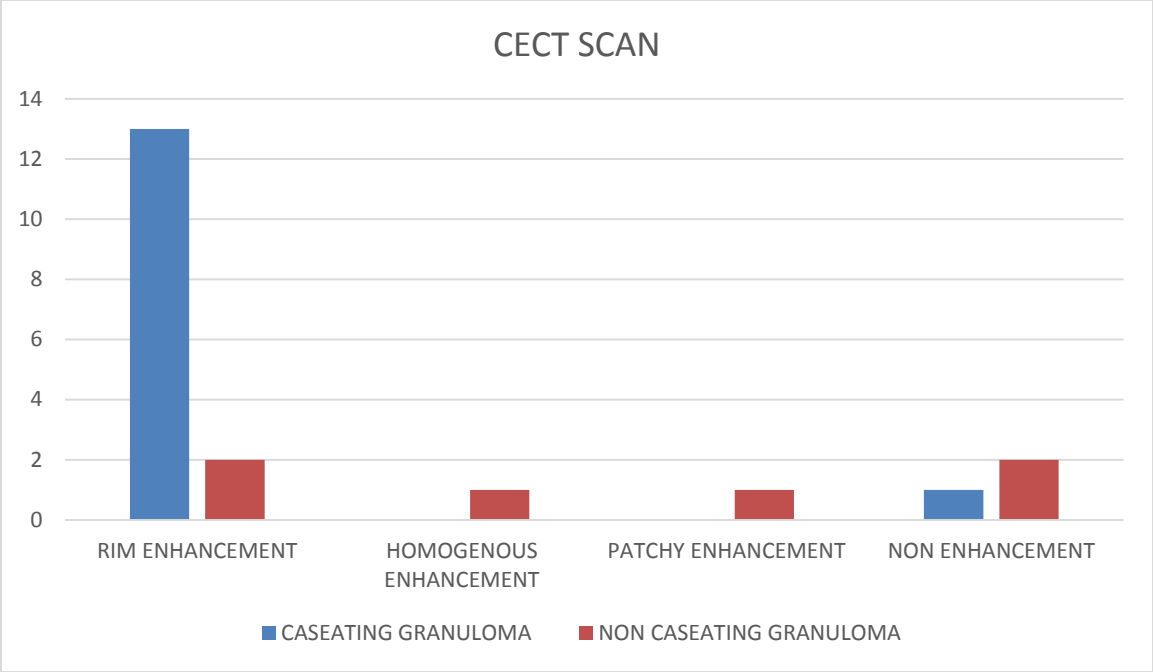


Graph-7 Bar diagram showing number of patients with hypodense, isodense and hyperdense lesions on NCCT scan

In this study, 13(92.9%) patient with caseating granuloma were showing rim enhancement with significant *p*-value. Non caseating granuloma were showing either rim enhancement, homogenous enhancement, patchy enhancement or non-enhancement. P value is .036

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
CECT SCAN	RIM ENHANCEMENT	Count	13	2	15
		% within CECT SCAN	86.7%	13.3%	100.0%
		% within Pathology	92.9%	33.3%	75.0%
	HOMOGENOUS ENHANCEMENT	Count	0	1	1
		% within CECT SCAN	0.0%	100.0%	100.0%
		% within Pathology	0.0%	16.7%	5.0%
	PATCHY ENHANCEMENT	Count	0	1	1
		% within CECT SCAN	0.0%	100.0%	100.0%
		% within Pathology	0.0%	16.7%	5.0%
	NON ENHANCEMENT	Count	1	2	3
		% within CECT SCAN	33.3%	66.7%	100.0%
		% within Pathology	7.1%	33.3%	15.0%
		% of Total	5.0%	10.0%	15.0%
Total		Count	14	6	20
		Expected Count	14.0	6.0	20.0
		% within CECT SCAN	70.0%	30.0%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	70.0%	30.0%	100.0%

Table 8: Different findings of granulomas noticed on Contrast enhanced CT scan



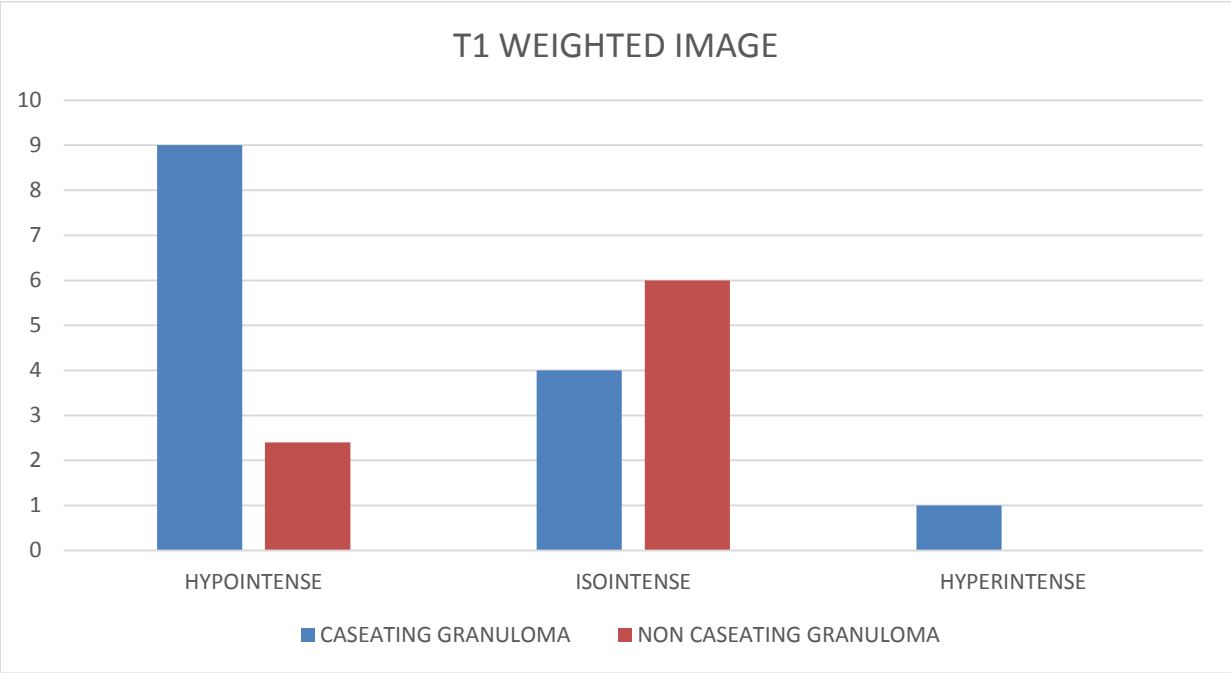
Graph 8: Bar diagram showing Contrast enhanced CT finds of different granulomas

MRI T1 WEIGHTED IMAGE

MRI was available for 23 patients. Caseating granuloma cases were hypointense, isointense and hyperintense in 64.2%, 28.5%, 7.1% respectively. Non caseating granulomas were hypointense and isointense in 33.3% and 66.7% respectively. P value found on calculation was .176 (Table 9 and Graph-9)

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
T1	HYPOINTENSE	Count	9	3	12
		% within T1	75.0%	25.0%	100.0%
		% within Pathology	64.3%	33.3%	52.2%
		% of Total	39.1%	13.0%	52.2%
	ISOINTENSE	Count	4	6	10
		% within T1	40.0%	60.0%	100.0%
		% within Pathology	28.6%	66.7%	43.5%
		% of Total	17.4%	26.1%	43.5%
	HYPERINTENSE	Count	1	0	1
		% within T1	100.0%	0.0%	100.0%
		% within Pathology	7.1%	0.0%	4.3%
		% of Total	4.3%	0.0%	4.3%
Total		Count	14	9	23
		% within T1	60.9%	39.1%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	60.9%	39.1%	100.0%

Table 9: MR imaging findings in T1 weighted sequence of different granulomas



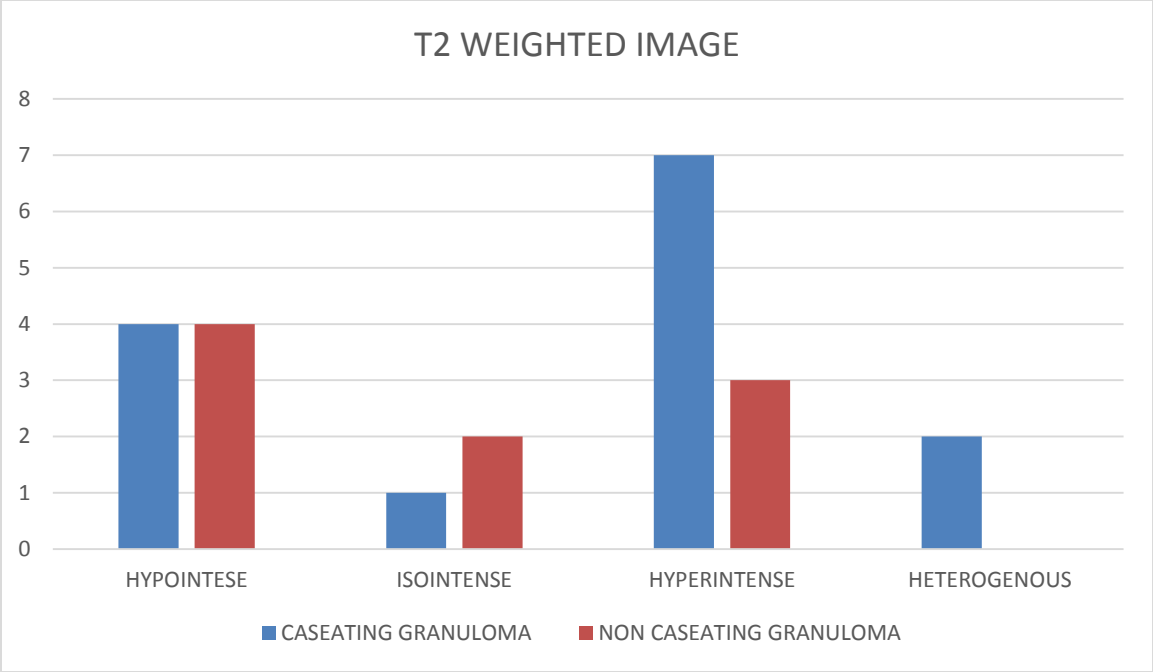
Graph 9: Bar diagram showing MRI T1 Weighted scan findings in both groups

MRI T2 WEIGHTED IMAGE

In our study, caseating granuloma were hypointense (28.6%), isointense (7.1%), hyperintense (50%) and heterogenous (14.3%) on T2 weighted sequence. Whereas non caseating granulomas were hypointense (44.4%), isointense (22.2%), hyperintense (33.3%) and heterogenous (0%) on T2 weighted sequence. P value 0.394 (Table 10 and Graph 10).

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
T2	HYPOINTENSE	Count	4	4	8
		% within T2	50.0%	50.0%	100.0%
		% within Pathology	28.6%	44.4%	34.8%
		% of Total	17.4%	17.4%	34.8%
	ISOINTENSE	Count	1	2	3
		% within T2	33.3%	66.7%	100.0%
		% within Pathology	7.1%	22.2%	13.0%
		% of Total	4.3%	8.7%	13.0%
	HYPERINTENSE	Count	7	3	10
		% within T2	70.0%	30.0%	100.0%
		% within Pathology	50.0%	33.3%	43.5%
		% of Total	30.4%	13.0%	43.5%
	HETEROGENOUS	Count	2	0	2
		% within T2	100.0%	0.0%	100.0%
		% within Pathology	14.3%	0.0%	8.7%
		% of Total	8.7%	0.0%	8.7%
Total		Count	14	9	23
		% within T2	60.9%	39.1%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	60.9%	39.1%	100.0%

Table 10: T2 weighted MR imaging sequence of caseating and non caseating granulomas



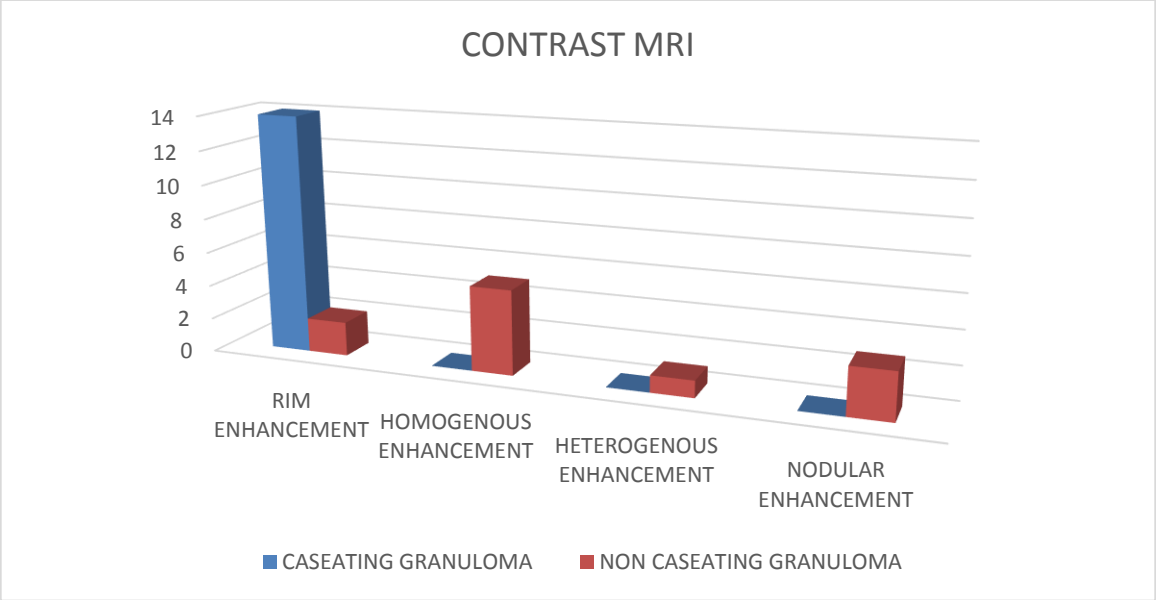
Graph 10: Bar diagram of T2 Weighted MR sequence of caseating and non caseating granulomas

CONTRAST MRI

All the cases of caseating granuloma (100%) were showing rim enhancement on T1 contrast image. Non caseating granuloma patient were showing rim enhancement (22.2%), homogenous enhancement (55.6%), heterogenous enhancement (11.1%) and nodular enhancement (11.1%). P value was highly significant 0.001(Table 11 and Graph 11)

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
CONTRAST	RIM ENHANCEMENT	Count	14	2	16
		% within CONTRAST	87.5%	12.5%	100.0%
		% within Pathology	100.0%	22.2%	69.6%
	HOMOGENOUS ENHANCEMENT	Count	0	5	5
		% within CONTRAST	0.0%	100.0%	100.0%
		% within Pathology	0.0%	55.6%	21.7%
	HETEROGENOUS ENHANCEMENT	Count	0	1	1
		% within CONTRAST	0.0%	100.0%	100.0%
		% within Pathology	0.0%	11.1%	4.3%
	Nodular Enhancement	Count	0	1	1
		% within CONTRAST	0.0%	100.0%	100.0%
		% within Pathology	0.0%	11.1%	4.3%
		% of Total	0.0%	4.3%	4.3%
Total		Count	14	9	23
		% within CONTRAST	60.9%	39.1%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	60.9%	39.1%	100.0%

Table 11: Contrast MR imaging showing different enhancement features of granuloma



Graph 11: Bar diagram showing of MR contrast enhancement features of tuberculomas

MR spectroscopy

MR Spectroscopy was available for 17 patients. 75% (n=9) of caseating granuloma patient had lipid lactate peak. 60% (n=3) of non caseating granuloma patient had lipid lactate peak. P value is .536 (Table 12)

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
MRS	Lactate Present	Count	9	3	12
		Expected Count	8.5	3.5	12.0
		% within MRS	75.0%	25.0%	100.0%
		% within Pathology	75.0%	60.0%	70.6%
		% of Total	52.9%	17.6%	70.6%
	Lactate Absent	Count	3	2	5
		Expected Count	3.5	1.5	5.0
		% within MRS	60.0%	40.0%	100.0%
		% within Pathology	25.0%	40.0%	29.4%
		% of Total	17.6%	11.8%	29.4%
Total		Count	12	5	17
		Expected Count	12.0	5.0	17.0
		% within MRS	70.6%	29.4%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	70.6%	29.4%	100.0%

Table 12: Showing MR spectroscopy different features of tuberculomas

PERFUSION

MR Perfusion was available for 12 patients. 90% (n=9) of caseating granuloma patients were showing low perfusion. In non caseating granuloma cases were both high (50%) and low perfusion (50%). P value is .166 (Table 13).

PERFUSION * Pathology Cross tabulation

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
PERFUSION	1.	Count	1	1	2
		Expected Count	1.7	.3	2.0
		% within PERFUSION	50.0%	50.0%	100.0%
		% within Pathology	10.0%	50.0%	16.7%
		% of Total	8.3%	8.3%	16.7%
	2.	Count	9	1	10
		Expected Count	8.3	1.7	10.0
		% within PERFUSION	90.0%	10.0%	100.0%
		% within Pathology	90.0%	50.0%	83.3%
		% of Total	75.0%	8.3%	83.3%
Total		Count	10	2	12
		Expected Count	10.0	2.0	12.0
		% within PERFUSION	83.3%	16.7%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	83.3%	16.7%	100.0%

Table 13- Perfusion characteristics of tuberculoma lesions

DIFFUSION WEIGHTED MR

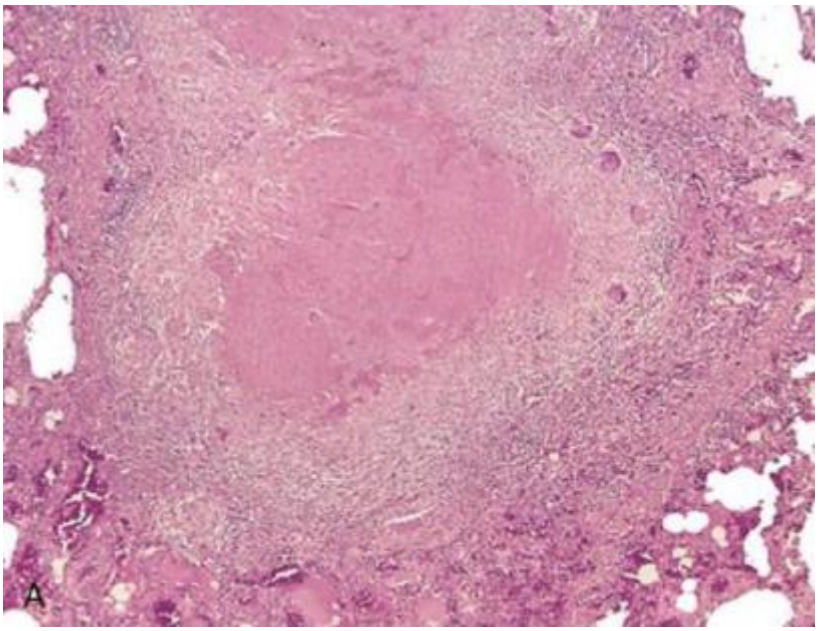
DW MRI was available for 16 patients. Caseating granuloma cases were showing no diffusion restriction in 90% of cases. Whereas all of the non caseating granuloma patients were showing no restriction. P value is .424 (Table 14).

DIFFUSION * Pathology Cross tabulation

			Pathology		Total
			CASEATING GRANULOMA	NON CASEATING GRANULOMA	
DIFFUSION	Restriction present.	Count	1	0	1
		% within DIFFUSION	100.0%	0.0%	100.0%
		% within Pathology	10.0%	0.0%	6.2%
	Restriction absent.	Count	9	6	15
		% within DIFFUSION	60.0%	40.0%	100.0%
		% within Pathology	90.0%	100.0%	93.8%
Total		Count	10	6	16
		% within DIFFUSION	62.5%	37.5%	100.0%
		% within Pathology	100.0%	100.0%	100.0%
		% of Total	62.5%	37.5%	100.0%

Table 14- Diffusion characteristics of CNS tuberculoma

Tuberculoma showing central caseous necrosis, macrophages and giant cell



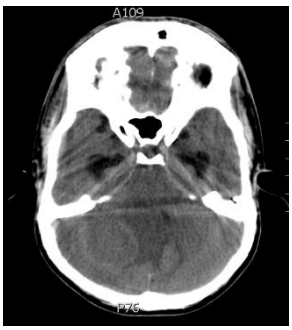


Figure 1

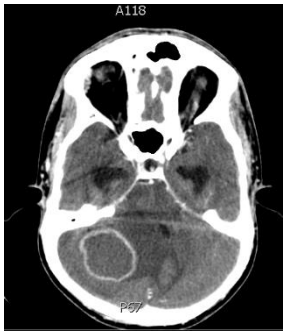


Figure 2

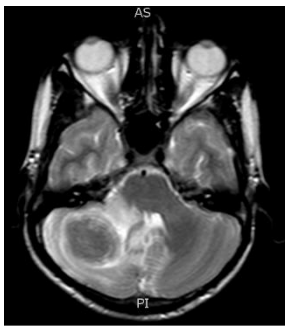


Figure 3

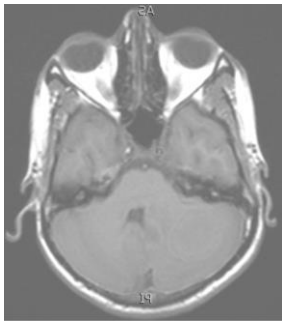


Figure 4

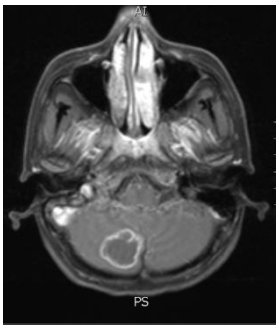


Figure 5

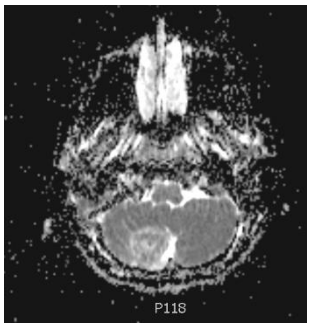


Figure 6

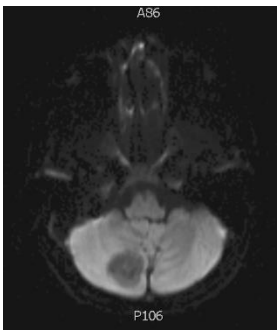


Figure 7

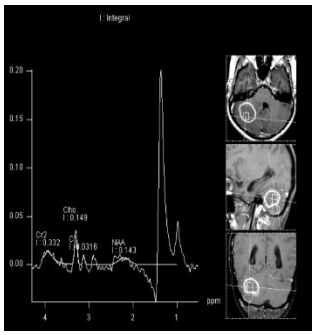


Figure 8

Figure 1:- Non-contrast CT- Tuberculoma is isodense

Figure 2:- Contrast CT scan -Tuberculoma is showing rim enhancement

Figure 3:- T2 weighted MRI -Tuberculoma is hypointense

Figure 4:- T1 weighted MRI -Tuberculoma is isointense

Figure 5:- Gadolinium T1 weighted MRI- Tuberculoma is showing rim enhancement

Figure 6/7:- Diffusion and ADC MRI -Tuberculoma is showing no restriction

Figure 8:- MR Spectroscopy showing high lipid peak

Discussion

The incidence of extra pulmonary TB is 20% of total cases diagnosed of TB. 10% cases of extra pulmonary tuberculosis are CNS tuberculosis[3]. In developing countries, the incidence of intracranial tuberculoma is 0.15%-0.18%. Intracranial tuberculoma are difficult to diagnosis because CSF and excised material are often negative for AFB.

In our study we observed that major component of tuberculomas were caseating granuloma (66.7%). A study was done by Sonmez et al in 2008, reported caseating granuloma in 85.9% of cases CNS tuberculomas[67].

Intracranial tuberculoma may be solitary or multiple. Multiple tuberculomas are more common[68]. In a study of Sonmez et al in 2008, tuberculomas were multiple in 89% of cases[67]. Kilani et al. had reported, multiple tuberculomas in 23 cases (80%) in a 122-patient study[68]. Results of the present study shows comparable results to the published literature.

Studies published by different authors showed the variable sizes of tuberculomas. Gupta et al. reported that tuberculoma with size <2.5 cm are more common[69]. Tuberculomas smaller than 2.5 cm were observed at a level of 90.5% in a study by Sonmez et al in 2008[67]. Most of the patients in present study presented with mass effects of large size tuberculoma which on evaluation found to be > 2.5cm

Sonmez et al in 2008 reported, the tuberculomas in cerebellar hemispheres and brainstem were 26.5% and 9.5% respectively. In present study also prevalence of supratenorial tuberculomas (40.7%) were more than infratentorial tuberculomas (37%).

No study has described regarding characteristics margins features of tuberculomas of caseating and non caseating type in imaging. We have found that the margins of tuberculoma are well

defined in caseating granuloma (66.7%) compare to non caseating granulomas (33.3%) but not found to be statistically significant which may be due to small sample size.

In this study, 70.4% of tuberculoma lesions were associated with perilesional edema. Number of studies has reported feature of perilesional edema, associated with tuberculomas but very few has quoted the high percentage association.

The "target sign" (central nidus of calcification surrounded by a ring of enhancement) was once considered to be pathognomonic for tuberculoma[71], but this has recently been called into question [72]. In this study no patient was found to have target lesion sign. About 10% of the lesions were hyperdense or hypodense on CT scans in the study of Wasay M et al in 2003 on 100 case of CNS tuberculomas[73]. Arthur R.D et al in 2013, has reported that caseating granuloma are hypodense to hyper dense and non caseating granuloma are hypodense to isodense[48]. In our study caseating granulomas were hypodense (42.8%), isodense (28.5%) and hyper dense (28.5%). Non caseating granuloma were hypodense (33.3%), isodense (33.3%), hyperdense (33.3%) on non- contrast CT scan but this feature was not found to be significant on P value calculation 0.934. In contrast CT scan, Arthur R.D et al has reported that caseating and noncaseating granulomas are showing rim enhancement and homogenous enhancement respectively[48]. In the present study 92.9% of caseating granuloma patient had rim enhancement. But non caseating granuloma were showing rim enhancement, homogenous enhancement, patchy enhancement and non-enhancement. This was found to be significant (p value is .036)

Noncaseating tuberculoma are hypointense and hyperintense on T1 and T2 weighted MRI respectively. The core of caseating granulomas may be solid (T1/T2 iso to hypointense) or liquid (T1 hypo and T2 hyper). On Contrast T1 images, tuberculoma may show both rim and homogenous enhancement. Presently appearance of target sign is non pathognomonic for

tuberculoma[48]. Sonmez et al in 2008 reported tuberculomas including caseating granulomas with cystic centre were observed in 85.9%, and noncaseating granulomas were 14.1%, none of the tuberculomas with caseating granulomas with solid centre were identified. In present study caseating granuloma cases were hypointense, isointense and hyperintense in 64.2%, 28.5%, 7.1% respectively on T1 weighted images. Non caseating granulomas were hypointense and isointense in 33.3% and 66.7% respectively on T1-weighted images. In T2-weighted MRI, caseating granuloma were hypointense (28.6%), isointense (7.1%), hyperintense (50%) and heterogenous (14.3%) on T2 weighted sequence. Whereas non caseating granulomas were hypointense (44.4%), isointense (22.2%) and hyperintense (33.3%) on T2 weighted sequence. Similarly in our study caseating granuloma with liquid centre is common and comparable to the literature.

In Gd T1 weighted images, Arthur R.D et al, has reported that caseating and non caseating granulomas are showing rim enhancement and homogenous enhancement respectively[48]. In the present study, caseating granuloma (100%) were showing rim enhancement on T1 contrast image. Non caseating granuloma patient were showing homogenous enhancement (55.6%). Results of present study is similar to data published in previous studies.

Tuberculomas show lipid lactate peak on spectroscopy (0.9, 1.3, 2.0 and 2.8 ppm). Lipid lactate peak depends upon the quantity of lipid in caseous necrosis[49]. 75% (n=9) of caseating granuloma patient in present study had lipid lactate peak.

DWI imaging is useful to diagnose abscess (infective pathology). Caseating tuberculoma with liquid center shows diffusion restriction[49]. In this study, Caseating granuloma cases were showing no diffusion restriction in 90% of cases. Whereas all of the non caseating granuloma patients were showing no restriction.

Conclusion

Intracranial tuberculoma albeit a benign entity but always represent a “diagnostic dilemma” in the differential diagnosis of intracranial space-occupying lesion. Central nervous system tuberculomas are mostly well defined caseating cystic granulomas with characteristic imaging findings on contrast MR and CT scan, which are predominantly solitary with significant diagnostic feature being rim enhancement. Of late advance MRI sequences like spectroscopy, diffusion, perfusion and magnetization can further aid in diagnosis and management of CNS tuberculoma. The clinical suspicion of tuberculomas should always be borne in mind when such lesions are being evaluated in patients who present with intracranial lesions.

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Name	Age	Sex	CT scan		MRI											Location	Pathology	POST OP IMAGING
			NON CONTRAST	CONTRAST	NO.	SIZE(CM)	MARGIN	PERILESIONAL	T1	T2	Contrast	MRS	PERFUSION	MT	DW1			
ANIL CR	28	M	ISO	RIM ENHANCEMENT	MULTIPLE (2)	3.7x3.3x2.3	WELL DEFINED	PRESENT	ISO	HYPO	RIM ENHANCEMENT	INCREASED LIPID,NORMAL CHOLINE CREATINE	NOT INCREASED	NOT DONE	NO RESTRICTION	RIGHT CEREBELLUM	CASEATING GRANULOMA	NO RESIDUAL LESION AND HYDROCEPHALUS REDUCED
SUDHA T	30	F	NOT DONE	NOT DONE	SINGLE	2.2X1.3X1.4	ILLDEFINED	NOT	HYPO	ISO	RIM ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NO AVAILABLE	D7	CASEATING GRANULOMA	NOT AVAILABLE
SUSEELA S	55	F	HYPODENSE	NO ENHANCEMENT	MULTIPLE	SMALL	IRREGULAR	PRESENT	HYPO	HYPER	RIM ENHANCEMENT	NO LIPID LACTATE PEAK	HYPOPERFUSION	NOT DONE	NO RESTRICTION	MULTIPLE	NON CASEATING RANULOMA	expired
SYED RABIA	15	F	HYPODENSE	RIM ENHANCEMENT	SINGLE	2.5x2.7x2.4	WELL DEFINED	NO	HYPO WITH PERIPHERAL MILD HYPER	HYPO WITH PERIPHERAL ISO	PERIPHERAL RIM ENHANCEMENT	NO LIPID LACTATE PEAK	HYPOPERFUSION	REDUCED	NO RESTRICTION	RIGHT CEREBELLUM	CASEATING GRANULOMA	NO RESIDUAL LESION AND HYDROCEPHALUS REDUCED
NAJIMA M	22	F	HYPODENSE	NO	SINGLE	5x4.4x4.2	IRREGULAR	PRESENT	ISO	HETEROGENOUS	RIM ENHANCEMENT	MILD INCREASE CHOLINE AND LACTATE	HYPOPERFUSION	INCREASED	NO RESTRICTION	LEFT MEDIAL FRONTAL LOBE	CASEATING GRANULOMA	NO RESIDUAL LESION
PREMALATHA P	34	F	HYPODENSE	RIM ENHANCEMENT	SINGLE	3x1.5x1.5	ILLDEFINED	PRESENT	LESION HYPO, RIM	HYPO	RIM ENHANCEMENT	LIPID LACTATE PEAK PRESENT	NOT INCREASED	INCREASED	NO RESTRICTION	LEFT CEREBELLUM	CASEATING GRANULOMA	NO RESIDUE
BINCY C	10	M	HYPERDENSE	RIM ENHANCEMENT	MULTIPLE	3.2x1.6x1	WELL DEFINED	NO	NO	NO	NO	NO	NO	NOT DONE	NO	RIGHT CEREBELLUM	CASEATING GRANULOMA	HYDROCEPHALUS PRESENT
SUBBA LAKSHMI M	16	F	HYPODENSE	RIM ENHANCEMENT	SINGLE	2.12x1.83	WELL DEFINED	PRESENT	HYPO	HYPER	RIM ENHANCEMENT	NO LIPID LACTATE PEAK	NOT INCREASED	NOT DONE	NOT DONE	VERMIS	CASEATING GRANULOMA	NO GROSS RESIDUE AND NO HYDROCEPHALUS
SRUTHI K	9	F	NOT AVAILABLE	NOT DONE	SINGLE	6.1x4.1x6.4	ILLDEFINED	PRESENT	HYPO CENTRAL AND HYPER	HYPER CERNTRAL AND PERIPHERAL ISO	RIM ENHANCEMENT	INCREASED LIPID, INCREASED CHOLINE , DECREASED NAA	INCREASED	NOT DONE	NO RESTRICTION	RIGHT PARIETAL LOBE	CASEATING GRANULOMA	NO GROSS RESIDUE
VAHITHA F	25	F	ISODENSE	RIM ENHANCEMENT	SIGNLE	3x2.9x2.5	ILLDEFINED	PRESENT	ISO	HYPO	HETEROGENOUSENHANCEMENT	INCREASED LIPID LACTATE, NAA AND CREATININE NORMAL	INCREASED	NOT DONE	NO RESTRICTION	LEFT BASAL GANGLIA AND SUPRASELLAR AREA	NON CASEATING GRANULOMA	RESIDUE PRESENT WITH SERIAL SCAN SHOWED REDUCTION IN SIZE AND PERFUSION
ANIL KUMAR VS	41	M	NOT AVAILABLE	NOT DONE	SINGLE	3.2x2.6X1.8	WELL DEFINED	PRESENT	CENTRAL HYPO AND PERIPHERAL HYPER	CENTRAL HYPERINTENCE AND PERIPHERAL HYPO	RIM ENHANCEMENT	INCREASED LIPID LACTATE, NAA DECREASED AND CHOLINE INCREASED	HYPOPERFUSION	NOT DONE	RESTRICTION	RIGHT CEREBELLUM	CASEATING GRANULOMA	NO RESIDUE
JACKSON ALBERT	18	M	NOT AVAILABLE	NOT DONE	SINGLE	3.1x3.6x2.7	WELL DEFINED	PRESENT	CENTRAL HYPER AND PERIPHERAL ISO-	CENTRAL HYPER AND PERIPHERAL HYPO	RIM ENHANCEMENT	INCREASED LIPID LACTATE, NAA DECREASED AND ELEVATED CHO/CR RATIO	HYPOPERFUSION	INCREASED	NO RESTRICTION	VERMIS	CASEATING GRANULOMA	NOT AVAILABLE
ELDHO JOBI JOHN	37	M	NOT AVAILABLE	NOT DONE	SINGLE	2x2x1	ILL DEFINED	NO	ISO	HYPO	HOMOGENOUS ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	D6-8 INTRADURAL	NON CASEATING GRANULLOMA	NOT AVAILABLE
GANESH PANDI P	29	M	HYPERDENSE	PACHY ENHANCEMENT	SINGLE	6x3.3x2	WELL DEFINED	NO	ISO	ISO	UNIFORM ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	LEFT MECKEL'S CAVE	NON CASEATING GRANULOMA	RESIDUAL LESION PRESENT
ULAGANATHAN M	22	M	HYPER	RIM ENHANCEMENT	MULTIPLE	SMALL LESIONS	MULTIPLE CONGLOMERATED WELL DEFINED	PRESENT	CENTRAL HYPO AND PERIPHERAL ISO	CENTRAL HYPERINTENCE AND PERIPHERAL HYPO WITH VASOGENIC EDEMA	PERIPHERAL RIM ENHANCEMENT	INCREASED LIPID LACTATE, NAA DECREASED AND CHOLINE	HYPOPERFUSION	NOT DONE	NO RESTRICTION	MULTIPLE RIGHT CEREBRAL AND LEFT CEREBELLAR	CASEATING GRANULOMA	RESIDUAL LESION PRESENT
ANNAMMA THOMAS	56	F	NOT AVAILABLE		SINGLE	2x1	ILLDEFINED	NO	ISO	HYPO	HOMOGENOUS ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	D5-5 INTRADURAL EXTRAMEDULLARY	NON CASEATING GRANULOMA	NOT AVAILABLE
KALIRAJ S	29	M	HYPODENSE	RIM ENHANCEMENT	SINGLE	3.2x2.8x2.9	WELL DEFINED	PRESENT	ISO	ISO WITH PERIPHERAL HYPO	RIM ENHANCEMENT	LIPID LACTATE PEAK PRESENT	NOT AVAILABLE	NOT DONE	NO RESTRICTION	VRMIS WITH EXTENSION TO RIGHT CEREBELLUM	NON CASEATING GRANULOMA	RESIDUAL LESION PRESENT
LUXY JOSY	29	F	ISO	RIM ENHANCEMENT	SINGLE	2.7X1.3X3	WELL DEFINED	PRESENT	ISO	HYPO	RIM ENHANCEMENT	LIPID LACTATE PEAK PRESENT	NOT AVAILABLE	NOT DONE	NOT DONE	RIGHT CEREBELLUM	CASEATING GRANULOMA	NO RESIDUE
VALSALA M	31	F	ISO	RIM ENHANCEMENT	SINGLE	3x2x1	WELL DEFINED	PRESENT	NOT AVAILABLE	NOT AVAILABLE	NOT AVIALBLE	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	LEFT PARIETAL LOBE	CASEATING GRANULOMA	NO RESIDUE
SUBHA K.S	7	F	HYPO	RIM ENHANCEMENT	MULTIPLE	4.1x3.2x1(LARGEST)	WELL DEFINED	PRESENT	NOT AVAILABLE	NOT AVAILABLE	NOT AVIALBLE	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	LEFT PARIETAL LOBE AND RIGHT CEREBELLUM	CASEATING GRANULOMA	NO PARIETAL LOBE RESIDUE
SAROJA M	45	F	HYPO	RIM ENHANCEMENT	SIGNLE	4.2x3.2x2	ILLDEFINED	NO	HYPO	HYPER	RIM ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	LEFT CEREBELLUM	CASEATING GRANULOMA	NOT AVAILABLE
ANAN KUTT	68	M	HYPERDENS	HOMOGENOUS ENHANCE	SINGLE	3x2X2	ILLDEFINED	PRESENT	HYPO	HYPER	UNIFORM ENHANCEMENT	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	RIGHT FRONTOPARIETAL	NON CASEATING GRANULOMA	NOT AVAILABLE
THANKAPPAN MG	52	M	ISO	RIM ENHANCEMENT	SINGLE	3.5x2.3x2	ILLDEFINED	PRESENT	NOT AVAILABLE	NOT AVAILABLE	NOT AVIALBLE	NOT AVAILABLE	NOT AVAILABLE	NOT DONE	NOT DONE	LEFT MEDIAL FRONTAL LOBE	CASEATING GRANULOMA	NO RESIDUE
SOMRAJAN	44	M	ISO	NO ENHANCEMENT	SINGLE	2.5x6	ILLDEFINED	PRESENT	ISO	HYPO	NODULAR ENHANCEMENT WITH DURAL TAIL	NO LIPID LACTATE PEAK	NOT AVAILABLE	NOT DONE	NO RESTRICTION	LEFT TEMPORAL	NON CASEATING GRANULOMA	NO RESIDUE
NARAYANA PILLAI	36	M	NOT AVAILABLE	NOT DONE	MULTIPLE	4.5x3.5x2.5(LARGEST)	WELL DEFINED	PRESENT	HYPO	HYPER	HOMOGENOUS ENHANCEMENT	LIPID LACTATE PEAK PRESENT INCREASE CHOLINE AND REDUCE NAA	NOT AVAILABLE	NOT DONE	NO RESTRICTION	MULTIPLE LESIONS(RIGHT FRONTAL/UNCAL/PARIETAL AND OCCIPITAL)	NON CASEATING GRANULOMA	NAD
AMRITHA PV	11	F	HYPERDENSE	RIM ENHANCEMENT	1	2.7x18x19	WELL DEFINED	NO	ISO	HETEROGENOUS	RIM ENHANCEMENT	NO LIPID LACTATE PEAK	NOT AVAILABLE	NOT DONE	NO RESTRICTION	SEPTUM PLLEUCIDUM	CASEATING GRANULOMA	NO RESIDUE
RADHA K	35	F	HYPERDENSE	RIM ENHANCEMENT	MULTIPLE	SMALL	WELL DEFINED	PRESENT	HYPO	HYPER WITH CENTRE HYPO	RIM ENHANCEMENT	LIPID LACTATE PEAK	NOT INCREASED	NOT DONE	NO RESTRICTION	PARIETAL/TEMPORAL/FRONTAL	CASEATING GRANULOMA	LOST

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SIMILARITY INDEX

PRIMARY SOURCES

1	DeLance, Arthur R., Michael Safaee, Michael C. Oh, Aaron J. Clark, Gurvinder Kaur, Matthew Z. Sun, Andrew W. Bollen, Joanna J. Phillips, and Andrew T. Parsa. "Tuberculoma of the central nervous system", Journal of Clinical Neuroscience, 2013. CrossCheck	349 words — 7%
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