

INTRACRANIAL PLASMA CELL GRANULOMA

**Dr. Talla Vinay Bhushanam*, M.S. M.Ch., Dr. Alugolu Rajesh, M.S., M.Ch., Dr. Megha Shantveer Uppin M.D.,
Dr. Suryaprabha M.D., D.M.**

Department of Neurosurgery and Neurology, Department of Pathology, Nizam's Institute of Medical Sciences,
Hyderabad, A.P., India.

***Corresponding Author: Dr. Talla Vinay Bhushanam**

Department of Neurosurgery and Neurology, Department of Pathology, Nizam's Institute of Medical Sciences, Hyderabad, A.P., India.

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ABSTRACT

Plasma cell granuloma (PCG)/ inflammatory pseudo- tumour or inflammatory myofibroblastic tumour is relatively rare entity arising within numerous organs. PCGs have been found in many organ systems however, involvement of brain that too primary is very rare with upto 28 cases reported till date in English literature. A 19 year male patient suffering from secondary generalised seizures, diagnosed imageologically as tuberculoma failed to show clinical and radiological improvement despite adequate anti-tubercular treatment, was subjected to surgical excision with intraoperative corticography. We herein describe our case along with the review of literature regarding primary CNS plasma cell granulomas. Radiological diagnosis of granulomas, treated empirically as tuberculosis needs to be followed cautiously and the diagnosis reviewed in case if there is no response to chemotherapy and be subjected to biopsy/excision and histopathology/culture for there are many radiological mimics with intracranial plasma cell granuloma being one among them.

KEYWORDS: Central nervous system, inflammatory, intracranial, myofibroblastic tumour, plasma cell granuloma, pseudo-tumour.

INTRODUCTION

Inflammatory pseudo tumour is a pathological term used to describe a reactive, inflammatory, non-neoplastic phenomenon that occurs in various organ systems and most of them have been found in lungs.^[1-9] The term "plasma cell granuloma is preferred to other synonyms such as inflammatory pseudo-tumour, xanthogranuloma, fibroxanthoma, or histiocytoma because of the predominance of plasma cells common to these lesions.^[1] Involvement of extrapulmonary sites is considered innocuous, however, recurrences and sarcomatous changes requiring redo surgery and chemo-radiotherapy have also been described.

CASE REPORT

A 19 year male presented with generalised clonic tonic seizures and left sided headache for over 2 years with a recent onset of behavioural and memory disturbances. There were no motor or sensory deficits. CT scan brain plain and contrast done 2 years back showed a hyperdense lesion in the left frontal region, enhancing well on contrast (Figure 1). MRI Brain at that time showed a lesion in the left middle frontal gyrus, of size 14 X 20 X 26 mm, isointense on T1W and hyperintense on T2W with heterogenous enhancement on contrast (Figure 2). Blood investigations were normal with an ESR of 8mm/hr and Mantoux tuberculin skin test of

12mm skin induration. A provisional diagnosis of cerebral tuberculoma was made at a peripheral centre and the patient was started on antitubercular treatment with isoniazid, rifampicin, pyrazinamide, and ethambutol for 2 months followed by isoniazid and rifampicin for another 16 months. Antiepileptic phenytoin sodium was added according to his weight for seizure control. However, even after completion of 18 months, neither a change in the seizure control nor radiological change was observed (Figure 3). Hence he was referred to our center for further evaluation and management.

Neurological examination is within normal limits except for minimal blunting of higher mental functions. Blood investigations were normal. SEP was not done preoperatively. Left fronto-parietal craniotomy and electro-corticography guided gross total excision of the tumour was done. Tumor was fleshy, firm, and mildly vascular with a clear gliotic plane from the surrounding parenchyma (Video attached). The corticography showed bursts of spikes with frequency of 2 Hz lasting for 3 seconds noted mostly superior and anterior parts of the tumour. Post excision corticography showed no spikes in and around the tumour cavity.

Post operative course was uneventful and patient is doing well after 4 months with no further seizure episodes and

gradual tapering of anti-epileptics. Serum electrophoresis done at one month post-op was normal.

Gross appearance of the resected specimen showing tumour (fig A) of 2 X 2 X 3 cms size, lobulated, partially encapsulated, reddish grey, highly vascular, rubbery consistency with dural adhesion at one surface. Histopathological examination (Fig B) showing the tumour composed of fibrocollagenous dural tissue infiltrated by aggregates of dense plasma cells, eosinophils and lymphocytes with predominant plasma cells. Focally there are nodules of plasma cells with central areas showing apoptotic cells and nuclear debris. Blood vessels are congested and Smooth muscle actin is positive in them. Cells are negative for EMA and vimentin.

DISCUSSION

PCG was first described in 1973 for a lung lesion by Bahadori and Liebow.^[1] Intracranial plasma cell granuloma was first reported in 1980 by West et al.^[10] Primary intracranial plasma cell granuloma is a rare entity with only 28 cases reported till date in English literature (Pubmed search with key words as - Plasma cell granulomas, intracranial). They are described it as inflammatory pseudotumors characterised histologically by benign proliferation of plasma cells. Mature polyclonal plasma cells are the major cellular component beside histiocytes, fibroblasts, xanthomatous cells, lymphocytes, mast cells and eosinophils. Other lesions of major consideration in the differential diagnosis include plasmacytoma, granulomatous inflammations, histiocytosis X and Wegner's granulomatosis.^[11]

In cerebral CT scan PCG's are usually hyperdense and are surrounded by variable oedema with a slight to intense enhancement on contrast.^[31] Correspondingly, T1- weighted MRI demonstrates iso- to hyperintense lesions with distinct enhancement after application of Gadolinium. T2- weighted MRI sometimes reveal interdigitation with the adjacent cortex which is in line with observations of pathological changes in the neighbouring cerebral tissue. Although PCG's are well circumscribed lesions, lymphocytoplasmocytic inflammation, neuronal loss, and reactive gliosis can be found within the adjacent cortex.

Its diagnosis should be considered in a child with microcytic hypochromic anemia, hypergammaglobulinemia, and high ESR. Because of its rapid growth, local invasiveness, and recurrence, this lesion may be misdiagnosed as a sarcoma even by experienced pathologists.^[15] The elevated serum immunoglobulin reflects a result rather than cause of inflammatory pseudo-tumour.^[9] Preoperative differential diagnoses in the cases of CNS plasma cell granuloma include meningioma, hematoma, pituitary adenoma, ganglioglioma, astrocytoma, and metastasis.^[17]

Table 1: Review of cases of intracranial Plasma cell granuloma.

Author Year	Age Sex	Presenting symptoms	Blood invest.	SPEP Igs	Location of tumour / consideration	Extracranial involve	Light micro and Electron micro – general and extra features		Treatment	Out come- 2yrs
West et al 1980 ^[10]	17/M	Headache	Microcytic hypochromic anemia Elevated ESR	Elevated	Post. Fossa infra and supra tent. Meningioma	No	Plasma cells, russel bodies,lymphocytes in hyaline	Intranuclear inclusions, arachnoid villi	Total excis	No recurr.
Meada et al 1984 ^[26]	36/F	Gait disturbances Dysarthria, left VI n. Palsy, B/L papilledema	Normal	Normal	4 th ventricle	No	Same	Occ. Desmosomes	Total excis _ RT [prophylactically]	No recurrence
Dominic et al 1988 ^[11]	16/F	Headache, visual disturbances	Normal	Elevated Ig M only	Rt. frontal and supra sellar. Meningioma	No	Same	No birbeck granules	Sub total excision, Recurrence - RT	Recurred in 6 months
Ferrer et al - 2 cases 1989 ^[12]	19/F	Headach, DI, optic atrophy, growth retardation	Normal	Elevated	Hypothalamus and 4 th ventr floor	No	Same	Scarcely seen Russell bodies	Biopsy + RT	Recurred in 1 year
	48/F	Headache, disorientation,homonymous hemianopia	Normal	Normal	Hypothalamusand supra chiasmaic	No	Same	Same	Biopsy + RT	Died – intolerance to RT, Fistula, meningitis
Gangemi et al 1989 ^[13]	16/M	Left leg paresis	Normal	Normal	Right frontoparietal convexity	No	Same	----	Total excision	No recurrence
Figarella et al 1990 ^[14]	60/M	Generalised weakness	Microcytic hypochr anemia	Elevated	Right temporal lobe and horn	No	Same	Occasional multinucleated giant cells,Hemorrhagic foci at periphery and Whorl pattern of spindle cells	Total excision	No recurrence
Tang et al 1990 ^[15]	13/M	Headache	Microcytic hypochromic anemia Elevated ESR	Elevated	Left parieto-occipital	Lung	Same	Emperipolesis seen	Total excision + RT thinking as mets from lung	No recurrence
Pimentel et al 1993 ^[16]	18/M	Headache, iintellectual deterioration, right hemiparesis	Normal	Normal	Left lateral ventricle	No	Same	Occasional psammomatous bodies	Total excis	No recurr
Al sarraj et al 1995 ^[9]	58/M	Headache, vomittings, nystagmus	Elevated ESR	Elevated	4 th ventricle choroid plexus	polymyositis	Same	Areas of necrosis, foci of calcificationslymphocyte predomin,multinucleate giant cells	Biopsy	Died due to Atrial fibrillation and pulmonary embolism

Hadour et al 1995 ^[27]	28/M	Headache	Leucocytosis, Raised ESR	Elevate	Petrous apex and cavernous sinus	Lung – detected in routine chest X ray	Same	Plasma cells +ve for anti EMA	Numerous biopsies taken + steroids	No recurrence
Makino et al 1995 ^[28]	11/M	Headache	Normal	Not done	Left frontal convexity	No	Same	Small hemorrhagic areas	Total excision	No recurr
Tresser et al 1996 ^[17]	5/F	Behavioural changes, seizures	Leucocytosis	Elevated	Right parieto occipital intra axial mass	No	Same	Occasional germinal centres	Total excision	No recurrence, Last seizure 3 months after excision. Later no seizures
Saxena et al 2000 ^[29]	18/F	Headache, blurring, right homonymous hemianopia	ESR elevated	Elevated	Right temporal convexity	No	Same	--	Total excision	No recurr
Kilinc et al 2002 ^[30]	34/M	Headache, weakness both lower limbs	ESR elevated	Normal	Multiple cerebral, cerebellar and spinal cord	Spinal cord	Same	--	Done for spinal lesion and steroids for brain lesion as patient refused surgery	Reduction of perilesional edema in brain after excising spinal lesion
Buccoliero et al 2003 ^[18]	70/M	Visual disturbances	Normal	Normal	Frontal region, sella and 3 rd ventricle	No	Same	Plasma cells reactive for EMA	Biopsy + steroids	No increase in size of the lesion for 14 months
Brandsma et al- 3 cases 2003 ^[19]	36/F	Headache, hearing loss, tinnitus, optic atrophy	--	--	Left CP angle mass extending upto cavernous sinus	No	same	1 st operated { partial excision } and reported as meningioma with plasma cell infiltrate. 2 nd operation for increased size and reported as PCG and retrospect examination of previous specimen reported as PCG.		Developed cervical lymphnode enlargement 3 years later and diagnosed to have rosai dorfman. Died 5 years after initial presentation
	41/M	Seizures, left hemianopia	--	--	Right occipital tumour, developed frontal lesion 11 years later	No	same	Necrotic brain tissue, Lymphophagocytosis, polytypic B lymphocytes		Occipital lesion diagnosed as primary B cell lymphoma and cytarabine + methotrexate with RT given. Frontal lesion showed T cell infiltration. Hence previous specimen re-examined and diagnosis changed from B cell NHL to PCG. Treated as NHL
	33/F	Hearing loss	---	--	Infratentorial mass extending into bilateral internal acoustic meatus	Spinal cord	Same	lymphophagocytosis		Treated as TB meningitis but biopsy of craniovertebral junction lesion done due to rapid deterioration of neurological status showed PCG. Well maintained on RT and

									corticosteroids but granuloma increased in size after 3 years	
Greiner et al 2003 ^[20]	13/M	Headache, seizures	Normal	Normal	Right frontal	Lung 4 years back	Same	--	Total excis	No recurr
Shah et al 2005 ^[21]	14/F	Headache, 5 th , 6 th , 7 th cranial nerve palsy	Normal	Elevated	Right middle cranial fossa, cavernous sinus and infratemporal fossa	No	Same	Steroids given after biopsy and she is asymptomatic for 8 months. Later recurrence of lesion observed and she was kept on steroids, 6-mercaptopurine and methotrexate for 2 years and lesion resolved. Later recurred after 1 year radiologically but patient is neurologically asymptomatic.		
Sato et al 2006 ^[22]	51/M	Headache	Leucocytosis	Normal	Left parasagittal mass	No	Same	--	Sub total excis + RT	No recurrence
Miyahara et al 2008 ^[23]	73/M	Disorientation, right hemiparesis, gait disturbances	Elevated C-reactive protein	Normal	Trigone left lateral ventricle	No	Same	--	sub total excision + steroids	Residual lesion completely resolved on steroids. Died in 2 months due to DIC due to hemorrhagic gastric ulcer
Suri et al 2008 ^[24]	5/M	Seizures, headache	Normal	--	Basifrontal	No	Same	--	Total excis	No recurr
Lui et al – 4 cases 2009 ^[25]	60/F	Blurring	Normal	Normal	Tentorial mass	No	Same	--	Near total excis	No recurr
	52/F	Left superior quadrantanopia	Normal	Normal	Right lateral ventricle and corpus call	No	Same	-	Near total excis	No recurr
	45/M	Left hemiparesis	Normal	Normal	Right frontal	No	Same	-	Near total excis	No recurr
	26/F	Headache, diplopia	Normal	Normal	Left frontotemporal extending into orbit	No	Same	-	Biopsy + steroids	No recurr
Present case	19/M	Seizures, headache, behavioural distr.	Elevated leucocytes	Normal which was done 1 month after surgery	Left frontal lobe	No	Same		Total excis under corticographic guidance	2 months post op. no new symptoms

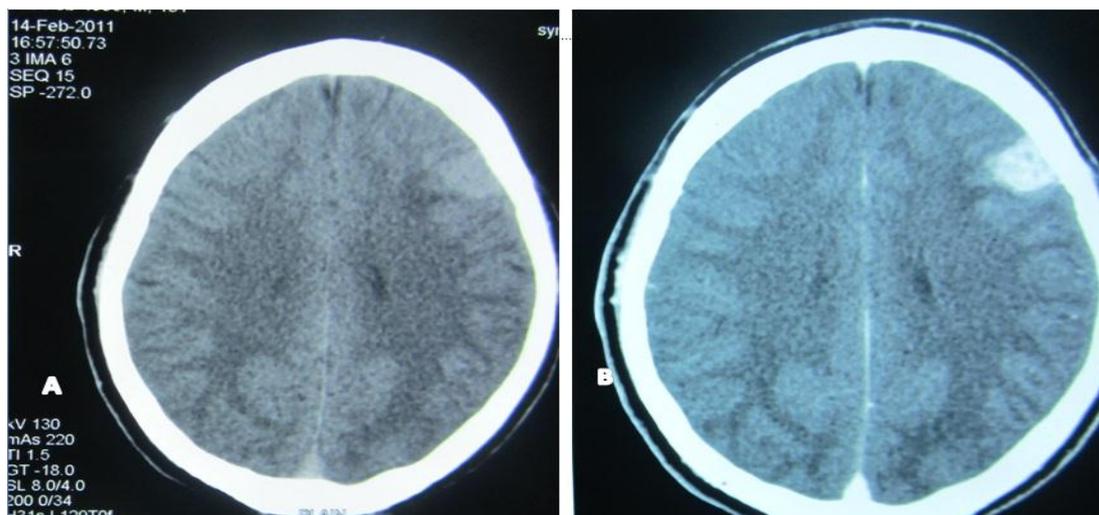


Figure 1: CT Brain plain (A) and contrast (B) taken in 2011 showing hyperdense lesion which is enhancing on contrast administration present in left frontal region with perilesional edema.

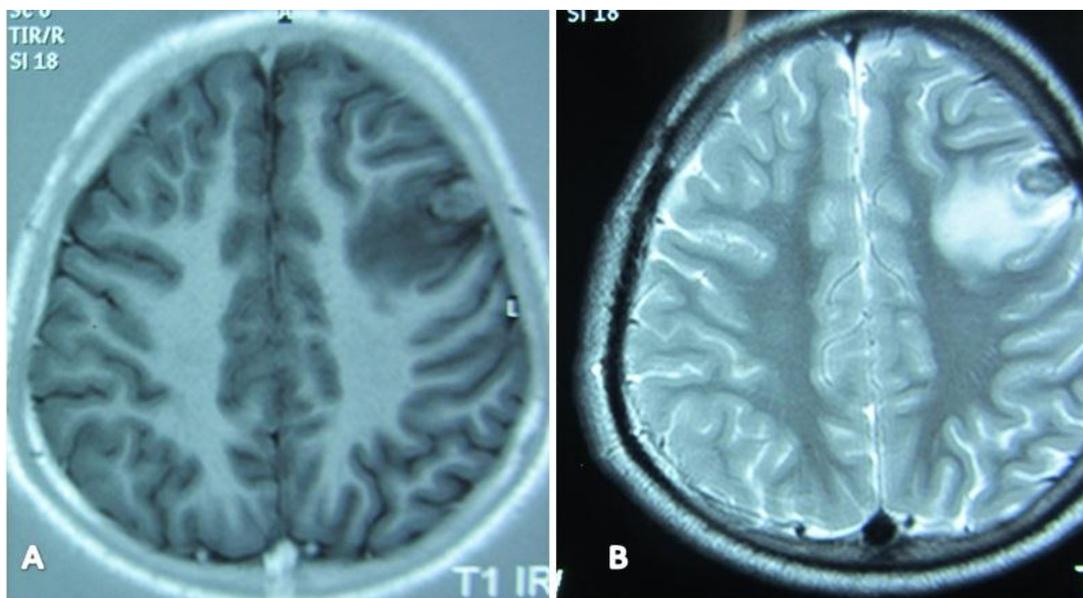


Figure 2: MRI Brain T1W (A) and T2W (B) taken in 2011 showing a lesion in the left middle frontal gyrus, of size 14 X 20 X 26 mm, isointense with peripheral hypointensity on T1W and hyperintense on T2W with a hypointense rim all around. MRI Brain contrast (C, D) showing enhancement.



Figure 3: MRI Brain T1W (A) and contrast (B) taken in 2012 showing no radiological change in lesion size OR enhancement pattern even after 18 months of ATT.



Figure 4: Tumor [T] excising from brain parenchyma.

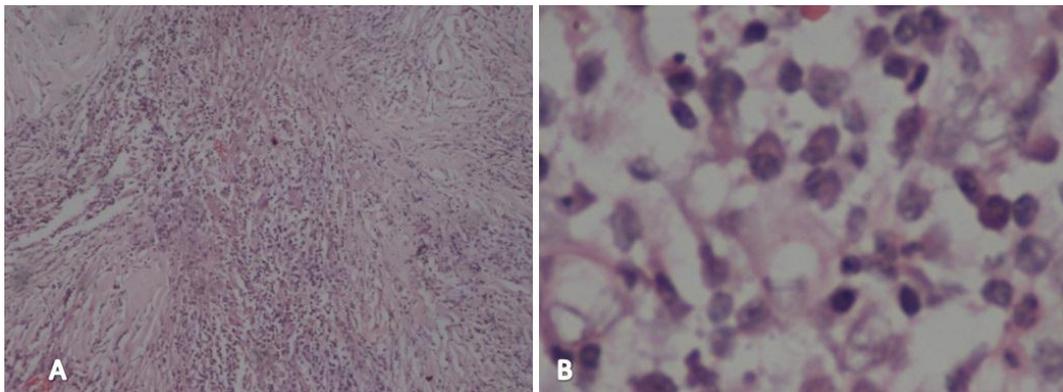


Figure 5: Magnification: 40 X and 100 X: Photomicrograph depicting mixed cellular components of plasma cell granulomas. H & E Left: Predominant plasma cells and eosinophils, lymphocytes under a background of fibrocollagenous stroma. Right: enlarged view showing plasma cells with eccentric nuclei.

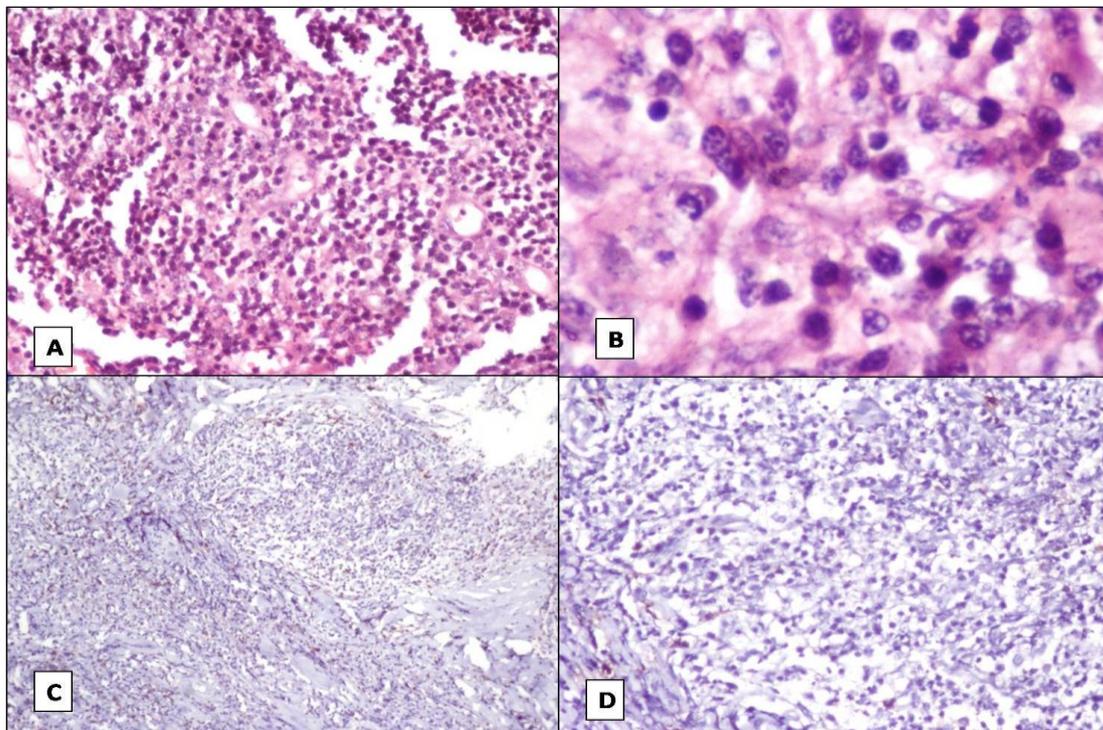


Figure 6 (A &B): Magnification: 40 X and 100 X. The microphotograph showing a lesion composed predominantly of inflammatory cells with most of them being plasma cells. (C&D) The cells are negative for EMA (HRP Polymer; EMA)

Reviewing the literature by Pubmed search with key words inflammatory pseudo-tumour, plasma cell granuloma, primary intracranial, myofibroblastic, central nervous system, 28 cases of intracranial PCG were reviewed (16 male and 12 female, mean age 32.71 years). Major presenting complaints were headache (in 18 patients) followed by visual disturbances (in 13 patients), paresis (5 patients), intellectual disturbances (5 patients), seizures (5 patients) and growth retardation (1 patient). [Table 1]

Hematological abnormalities described are microcytic hypochromic anemia, raised ESR, elevated leucocyte counts, and elevated polyclonal immunoglobulins in isolation or combination. However, urinary Bence Jones proteins were reported negative in all the cases. [Table 1]

PCG's were reported to occur in various parts of the cerebrum (10 in frontal regions, 5 in ventricles with 2 in 4th ventricle and 3 in lateral ventricles, 3 near tentorium, 3 in parieto-occipital region, 2 in hypothalamic region, each one in temporal region, CP angle, parasagittal, middle cranial fossa and near petrous apex.

Total resection was possible in 39.2%, gross total and subtotal in 21.42% of the patients (Table 1). Biopsy was done in 25% of the patients. In few cases, biopsy followed with radiotherapy and / or steroid supplementation given. In one patient recurrence occurred after giving RT and steroids and patient was subjected to chemotherapy with 6-mercaptopurine and methotrexate and the granuloma size increased in MRI after 1 year but patient remained asymptomatic.^[21] Treatment is mainly surgical as biopsy or incomplete excision followed by adjuvant therapy is confined only to the lesions which are diffuse and involving vital regions or complex anatomical regions like skull base.

Histopathologically PCG's shows predominance plasma cell with single nuclei, no mitosis, and scattered Russell bodies. In addition to lymphoid follicles, focal aggregates of epithelial-like cells with vesicular nuclei and distinct cytoplasmic bodies were intermingled with diffuse infiltration of plasma cells. The cells were interspersed in a hyaline fibrous tissue which showed positive trichrome stain for collagen and negative Congo red stain for amyloid. Exceptionally some PCG's showed occasional multinucleated giant cells,^[9,14] germinal centres,^[17] areas of necrosis,^[9,19] areas of haemorrhages,^[14,28] EMA positivity,^[18,27] whorl pattern of spindle cells^[14] and psammomatous bodies.^[16] Greiner *et al*^[20] demonstrated 7% Ki / MIB index in his case.

Sixty seven percent of PCG's not recurred within 3 months, 3 patients died,^[9,12,23] one because of trans sphenoidal fistula and meningitis following biopsy,^[12] one due to pulmonary embolus, and other died due to disseminated intravascular coagulation.^[Table 1] No recurrence was observed in all patients who underwent gross total resection. Three patients presented with

recurrences in which one occurred following subtotal excision and steroid therapy and two recurrences occurred following biopsy and steroid therapy. Survival analysis with Kaplan meier survival curve predicted that a probability of recurrence at 2 years is 0.885 (\pm 0.063 standard error) and the mean survival time 22.38 months (\pm 0.897 standard error). Patients who underwent biopsy followed with RT or steroids were maintained for 3-6 months without increase in the tumour volume. Most of them on repeat imaging showed resolving granuloma. In very few patients granuloma progressed and required further management with steroids and chemotherapy.

CONCLUSION

Radiological diagnosis of granulomas, treated empirically as tuberculosis should be followed cautiously and reviewed in case there is no response to chemotherapy and be subjected to biopsy/excision and histopathology/culture for there are many radiological mimics. Intracranial plasma cell granuloma is a rare entity and should be considered as differential diagnosis in meningiomas and granulomatous lesions.

REFERENCES

1. Bahadori M, Liebow AA. Plasma cell granulomas of the lung. *Cancer*, 1973; 31: 191-208.
2. Wentworth P, Lynch MJ, Fallis JC, Turner JA, Lowden JA, Conen PE. Xanthomatous pseudo-tumour of lung. A case report with electron microscope and lipid studies. *Cancer*, 1968; 22: 345-55.
3. Leonardo E, Palestro G. Intracellular Ig identification on paraffin-embedded solitary plasmacytoid proliferation: a routine method to define the biological nature of plasma cells. *Tumori*, 1978; 64: 457-61.
4. Acevedo A, Buhler JE. Plasma-cell granuloma of the gingiva. *Oral Surg Oral Med Oral Pathol*, 1977; 43: 196-200.
5. Holck S. Plasma cell granuloma of the thyroid. *Cancer*, 1981; 48: 830-2.
6. Isaacson P, Buchanan R, Mephram BL. Plasma cell granuloma of the stomach. *Hum Pathol*, 1978; 9: 355-8.
7. Fisch AE, Brodey PA. Plasma cell granuloma of kidney. *Urology*, 1976; 8: 89-91.
8. Wu JP, Yunis EJ, Fetterman G, Jaeschke WF, Gilbert EF. Inflammatory pseudo-tumours of the abdomen: plasma cell granulomas. *J Clin Pathol*, 1973; 26: 943-8.
9. al-Sarraj S, Wasserberg J, Bartlett R, Bridges LR. Inflammatory pseudotumour of the central nervous system: clinicopathological study of one case and review of the literature. *Br J Neurosurg*, 1995; 9: 57-66.
10. West SG, Pittman DL, Coggin JT. Intracranial plasma cell granuloma. *Cancer*, 1980; 46: 330-5.
11. Cannella DM, Prezyrna AP, Kapp JP. Primary intracranial plasma-cell granuloma- Case report. *J Neurosurg*, 1988; 69: 78 5-8.

12. Ferrer I, Garcia Bach M, Aparicio MA, Acebes JJ, Twose J, Isamat F. Plasma cell granuloma of the hypothalamic region. *Acta Neurochir (Wien)*, 1989; 99: 152-6.
13. Gangemi M, Maiuri F, Giamundo A, Donati P, De Chiara A. Intracranial plasma cell granuloma. *Neurosurgery*, 1989; 24: 591-5.
14. Figarella-Branger D, Gambarelli D, Perez-Castillo M, Garbe L, Grisoli F. Primary intracerebral plasma cell granuloma: a light, immunocytochemical, and ultrastructural study of one case. *Neurosurgery*, 1990; 27: 142-7.
15. Tang TT, Segura AD, Oechler HW, Harb JM, Adair SE, Gregg DC, Camitta BM, Franciosi RA. Inflammatory myofibrohistiocytic proliferation simulating sarcoma in children. *Cancer*, 1990; 65: 1626-34.
16. Pimentel J, Costa A, Távora L. Inflammatory pseudo-tumour of the choroid plexus. Case report. *J Neurosurg*, 1993; 79: 939-42.
17. Tresser N, Rolf C, Cohen M. Plasma cell granulomas of the brain: pediatric case presentation and review of the literature. *Childs Nerv Syst*, 1996; 12: 52-7.
18. Buccoliero AM, Caldarella A, Santucci M, Ammannati F, Mennonna P, Taddei A, Taddei GL. Plasma cell granuloma--an enigmatic lesion: description of an extensive intracranial case and review of the literature. *Arch Pathol Lab Med.*, 2003; 127: e220-3.
19. Brandsma D, Jansen GH, Spliet W, Van Nielen K, Taphoorn MJ. The diagnostic difficulties of meningeal and intracerebral plasma cell granulomas--presentation of three cases. *J Neurol*, 2003; 250: 1302-6.
20. Greiner C, Rickert CH, Möllmann FT, Rieger B, Semik M, Heindel W, Wassmann H. Plasma cell granuloma involving the brain and the lung. *Acta Neurochir (Wien)*, 2003; 145: 1127-31.
21. Shah MD, McClain KL. Intracranial plasma cell granuloma: case report and treatment of recurrence with methotrexate and 6-mercaptopurine. *J Pediatr Hematol Oncol*, 2005; 27: 599-603.
22. Sato K, Kubota T, Kitai R, Miyamori I. Meningeal plasma cell granulomas with relapsing polychondritis- Case report. *J Neurosurg*, 2006; 104: 143-6.
23. Miyahara K, Fujitsu K, Yagishita S, Takemoto Y, Ichikawa T, Matsunaga S, Takeda Y, Niino H, Shiina T. Inflammatory pseudo-tumour of the choroid plexus- Case report. *J Neurosurg*, 2008; 108: 365-9.
24. Suri V, Shukla B, Garg A, Singh M, Rishi A, Sharma MC, Sarkar C. Intracranial inflammatory pseudo-tumour: report of a rare case. *Neuropathology*, 2008; 28: 444-7.
25. Lui PC, Fan YS, Wong SS, Chan AN, Wong G, Chau TK, Tse GM, Cheng Y, Poon WS, Ng HK. Inflammatory pseudotumors of the central nervous system. *Hum Pathol*, 2009; 40: 1611-7.
26. Maeda Y, Tani E, Nakano M, Matsumoto T. Plasma-cell granuloma of the fourth ventricle. Case report. *J Neurosurg*, 1984; 60: 1291-6.
27. Le Marc'hadour F, Lavieille JP, Guilcher C, Brambilla E, Brichon PY, Lebas JF, Charachon R, Pasquier B. Coexistence of plasma cell granulomas of lung and central nervous system. *Pathol Res Pract*, 1995; 191: 1038-45.
28. Makino K, Murakami M, Kitano I, Ushio Y. Primary intracranial plasma-cell granuloma: a case report and review of the literature. *Surg Neurol*, 1995; 43: 374-8.
29. Saxena A, Sinha S, Tatke M. Intracranial plasma cell granuloma--a case report and review of the literature. *Br J Neurosurg*, 2000; 14: 492-5.
30. Kiliç M, Ertürk IO, Uysal H, Birler K, Evrenkaya T, Akkalyoncu BB. Multiple plasma cell granuloma of the central nervous system: a unique case with brain and spinal cord involvement. Case report and review of literature. *Spinal Cord*, 2002; 40: 203-6.
31. Bredahl WH, Robbins PD, Ives FJ, Wong G. Intracranial plasma cell granuloma. *Neuroradiology*, 1996; 38: S86-9.
- 32.