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Plasma Cell Granulomas of the Central Nervous System

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Abstract. Plasma cell granulomas of the central nervous system are rare benign forms of inflammatory pseudotumor of unknown etiology. They are often characterized by nonneoplastic proliferation of plasma cells and mimicking neoplastic processes. The author describes the management of six patients with histological proven plasma cell granulomas presented primarily with headache. Three were males with their age ranged from 9 to 43 years and a mean follow up of 40 months. The preoperative imaging studies revealed 3 cases located in the cerebral convexity and 3 were located in the posterior fossa region. Craniotomy was performed in 5 patients and in three patients complete resection was achieved. The resection was subtotal in 2 patients and they received postoperative steroids and radiation therapy. Complete resolution of the residual lesion was observed in one, and in another, the residual lesion remained stable during follow-up. One patient with infiltrating petroclival lesions had image-guided trans-nasal biopsy followed by radiation therapy with partial response. The author reviewed the literature of this rare entity and identified 46 reported cases of plasma cell granulomas. The clinicopathological and radiological findings, and the differential diagnoses were discussed and the management was outlined.

Keywords: Plasma cell granuloma, Inflammatory pseudotumors, Brain tumors.

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Introduction

Plasma cell granulomas (PCGs) are rare inflammatory lesions of unknown etiology composed of non-neoplastic proliferation of plasma and lymphoid cells. Bahadori and Leibow (1973) were the first to describe them in the lungs[1], which remain to be their most common site of occurrence[2-6]. However, PCGs have been reported to occur almost anywhere in the body. These locations include salivary gland, esophagus, intestines, lymph nodes, breast, skin[3], orbit[7,8], gingiva[9], thyroid gland[10], tonsil[11], stomach[12], liver[13], spleen[14] and pancreas[15]. Redondo-Fernandez and colleagues (1977) were the first to describe this rare entity in literature as a primarily intracranial located lesion[16]. The terminology used in the literature to describe PCGs in the past has been confusing, *i.e.* pseudolymphoma[16], inflammatory pseudotumor[17], fibrohistiocytoma[18], xanthomatous pseudotumor[2] and plasma cell-histiocytoma complex[6].

The author reports his experience of six cases of primary intracranial PCGs. The literature of the 46 reported cases of this rare entity was reviewed[17-52]. Cases were analyzed in an attempt to understand their epidemiology, radiological characteristics, histological appearance, response to different treatment modalities and outcome.

Materials and Methods

During the period from July 1998 to December 2005, a total number of 6 patients with PCGs were treated at King Abdulaziz University (KAU) and King Faisal Specialist Hospital and Research Center (KFSH&RC) in Jeddah, Saudi Arabia. The retrospective analysis of all brain tumors treated at both institutions presented an incidence of PCGs of about 0.02%. The medical records, with detailed analysis of the radiographic and the histopathological studies, were reviewed.

Clinical Presentations

The patients' data was summarized in Table 1. The age at presentation varied from 9- to 43-years-old (mean: 25-years), with equal sex distribution. In all, headache was the most common presenting symptom, and seizures were present in the 3 patients with supratentorial tumors. Ataxic gait was present in 2 patients and hoarseness in one with posterior fossae locations (Fig. 1). The neurological examination was normal in one patient, hemiparesis was present in a patient with a large left temporal lesion, and papilledema and lower cranial nerves palsy was present in 2 patients, respectively. Frontal swelling was seen in one patient with invasive frontal lesion (Fig. 2).

Table 1. Summary of the six patients with intracranial PCGs treated at KAU and KFSH&RC.

Case	Age / Sex	Clinical presentation	Location	Treatment	Outcome and follow-up (months)
1	9 year / F	Headache dysphagia, 9th to 12th cranial neuropathy, and ataxia	Left cerebello-pontine angle	Subtotal resection + steroids + radiotherapy	No recurrence (75)
2	19 year / M	Headache and ataxia	Midline cerebellar	Complete resection (+ cranioplasty)	No recurrence (56)
3	43 year / M	Headache, seizures and papilledema	Left temporal	Subtotal resection + steroids + radiotherapy	Stable residual (45)
4	32 year / M	Headache, seizures, frontal swelling and papilledema	Right frontal	Complete resection (+ cranioplasty)	No recurrence (38)
5	30 year / F	Headache, hoarseness, and 9th to 12th cranial neuropathy	Right petroclival	Biopsy + steroids + radiotherapy	Stable residual (18)
6	15 year / F	Headache and seizures (normal	Right parietal	Complete resection	No recurrence (6)

Investigations

The routine preoperative laboratory investigations and chest X-rays were unremarkable for all the patients. Preoperative testing for serum immunoglobulin levels was not requested, and in one, the postoperative testing was done and was negative (Case 5). All the patients had preoperative computed tomography (CT) scans, which demonstrated a hyperdense lesion with variable degrees of perilesional cerebral edema. The lesion in all had intense homogenous enhancement after intravenous contrast administration. In two patients, the skull bone was markedly eroded by the underlying mass. Magnetic resonance imaging (MRI) scans in all patients demonstrated the lesions to be an iso- to hypointense on T1 and hypointense on T2 scans with homogenous enhancement pattern after intravenous injection with gadolinium contrast agent (Gadopentetate Dimeglumine). Cerebral angiogram was done in one patient for preoperative embolization (Fig. 1e). The preoperative radiological diagnosis in all the cases was suggestive of meningioma.

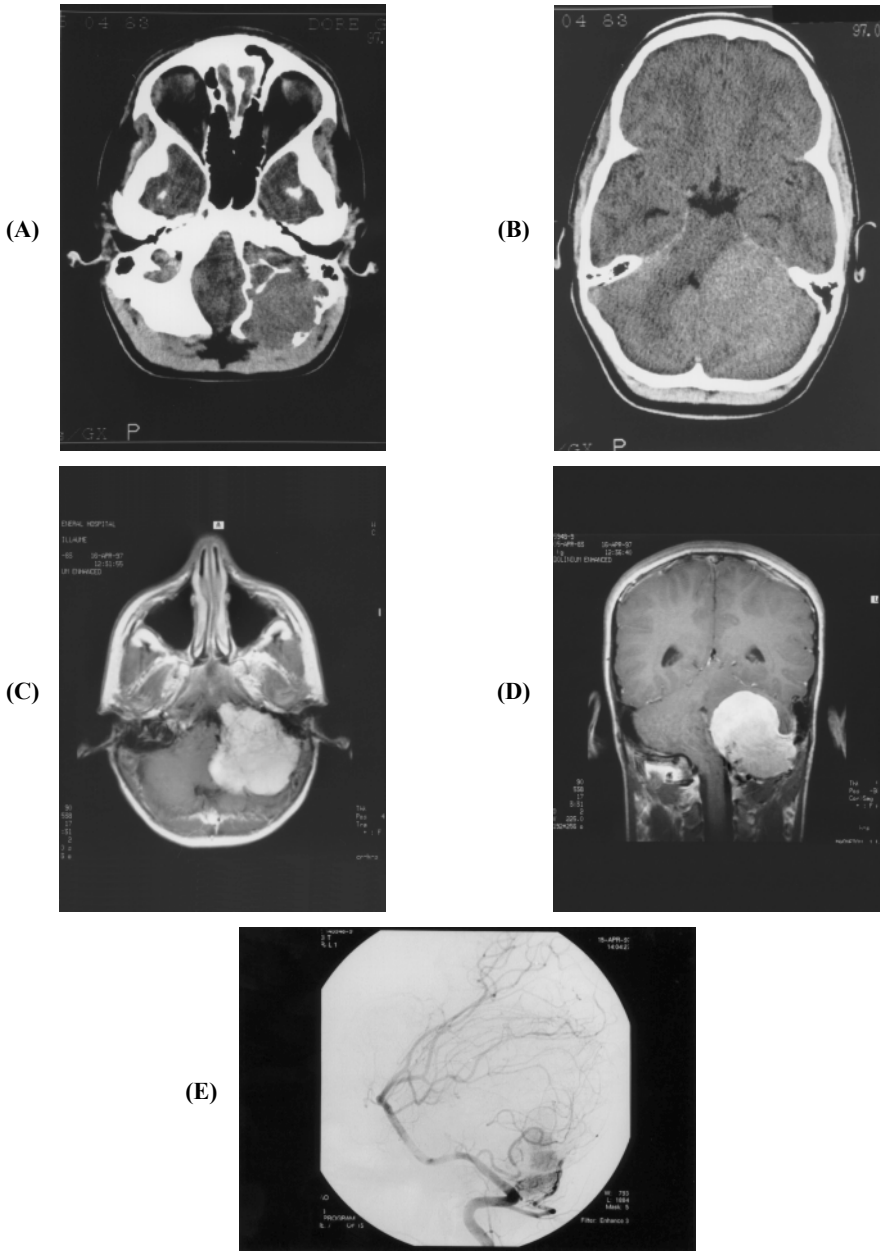

Fig. 1. Case illustration #1: A 9-yr-old female presented with headache and lower cranial neuropathy. CT scan demonstrated destruction of suboccipital bone (A) from a 50 × 45 × 45 mm hyperdense right cerebellopontine tumor with marked mass effect (B). MRI scans demonstrated homogenous enhancement and compression on the brain stem (C. D). Cerebral angiogram demonstrated marked tumor plush that was preoperatively embolized (E).

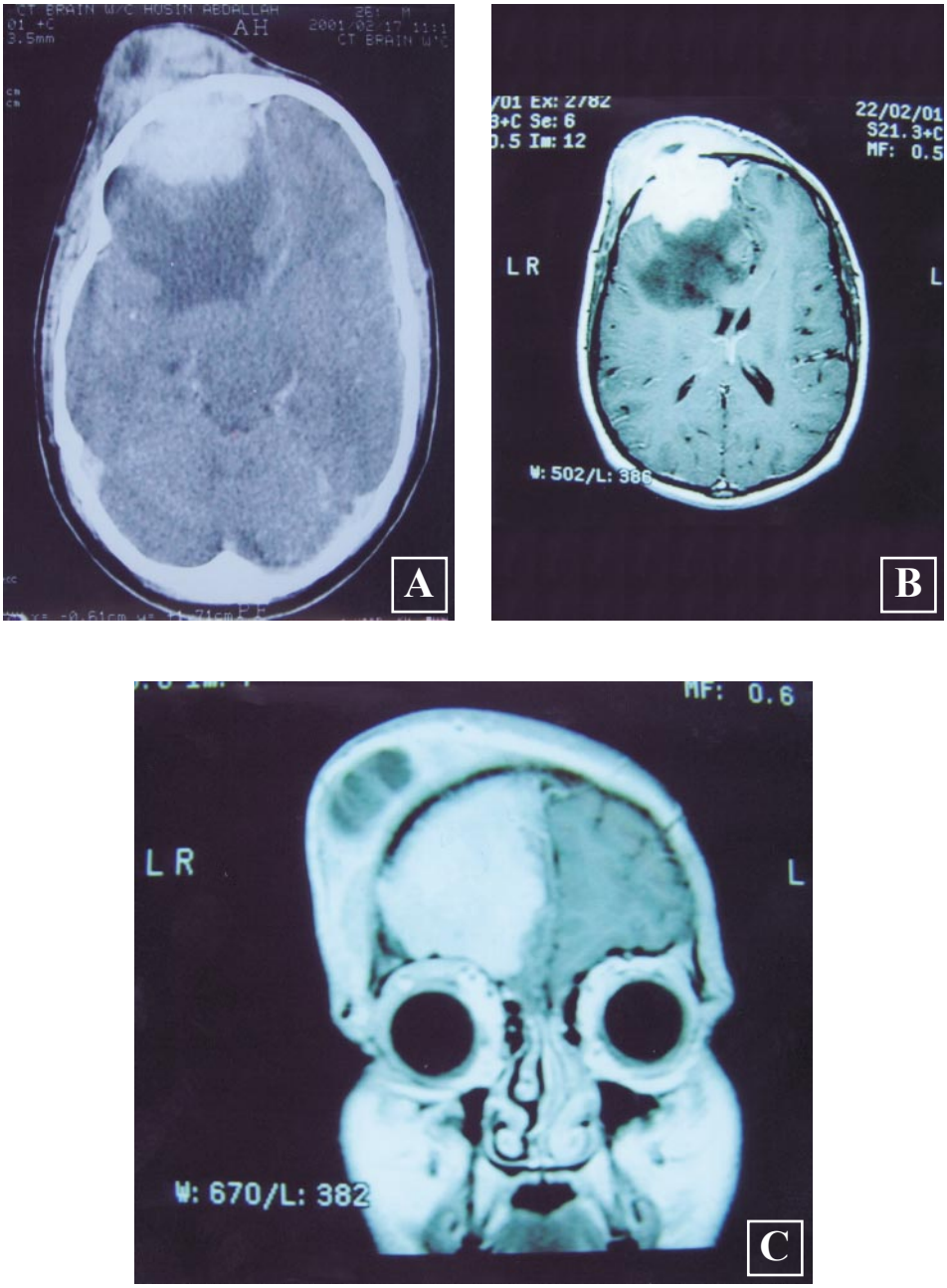

Fig. 2. Case illustration #4: A 32-yr-old male presented with headache, seizures, and frontal swellling. CT scan (A) and MRI (B,C) demonstrated 40 × 45 × 45 mm enhancing right frontal mass with underlying brain edema and destruction of the frontal bone with subcutaneous extension.

Operative Procedures

Craniotomy was done in 5 patients; 3 in a supratentorial and 2 in a posterior fossa located PCGs (Table 1). The intraoperative findings were consistent with a white-grayish relatively vascular mass invading the dura and to some extend the underlying brain tissue; a finding usually seen with meningiomas. Complete surgical excision including the involved dura was achieved in 3 patients, and the 2 patients in who had bony infiltration, cranioplasty was performed. The resection was subtotal in 2 patients and one patient with extensive petroclival infiltrating lesion had image-guided trans-nasal biopsy.

Histopathological Examinations

Routine Hematoxylin and Eosin (H&E) stain of all the specimens revealed a fibrous chronic inflammation of the dura and the arachnoid membranes, which produced an inflammatory pseudotumor. The inflammatory infiltrates included abundant mature plasma cells and polyclonal non-neoplastic T- and B-lymphocytes, which were either diffusely scattered or focally aggregated into lymphoid follicles, admixed with a few mononuclear histiocytes, mast cells and eosinophils. There was hyalinization and thickening of the small vessels wall with perivascular lymphocytic infiltrates. However, there were no foci of necrosis, and special staining (Periodic acid-Schiff (PAS), Ziehl-Neelsen (ZN); Gomori's Methenamine Silver (GMS)) did not reveal any infectious organisms. In three cases, sections from the brain underlying the inflammatory tumor showed an inflamed cortex with focal microcalcifications and increased capillary vessels with mild perivascular collections of lymphocytes. Immunohistochemical examination revealed polyclonal plasma cells with positive kappa- and lambda-light chain staining. The polyclonal nature of the abundant plasma cell population ruled out plasmocytoma. The meningioma markers (Epithelial membrane antigen (EMA), Vimentin, and cytokeratin) were all negative. These histopathological findings were consistent with PCGs.

Results

The postoperative course was uneventful in 5 patients; 3 with supratentorial and 2 with posterior fossa located PCGs (Table 1). The follow-up period of these cases ranged from 6- to 75-months (average, 40-months). All patients had follow-up MRI scans at 3-, 6-, and 12-months from surgery and annually thereafter. The 3 cases that had complete resection (Cases #2, 4, and 6) remained with no recurrence. The two patients that had subtotal resection and received postoperative radiotherapy and 3 months of steroids therapy reported complete resolution in one (Case #1) and partial response in another (Case # 3). The patient who had a biopsy received radiotherapy with partial regression

(50%) and remained stable for the 24 months of follow-up period. Of the 3 cases which presented with seizures, 2 became seizure free, and one still on one antiepileptic therapy. One patient (Case #1) developed postoperative worsening of her lower cranial palsy with difficulty swallowing and had repeated aspiration that required temporary tracheotomy and gastrostomy feeding for 6 months.

Discussion

Intracranial PCGs are rare forms of idiopathic inflammatory pseudotumors often characterized by non-neoplastic proliferation of plasma cells, and clinically mimicking a neoplastic process. The involvement of the central nervous system, however, is exceptional and rare. The current literature review for the 46 cases revealed that the age at presentation ranged between 1.5- to 80 years of age (average: 25-years) with 50% occurrence under the age of 20, with male predominance of 2:1. There was also an unusual racial predilection; 7 were black[16,17,30,36,39,40]; 7 were oriental[19,23,32,35,38,47]; 5 were Hispanic [21,22,24,27,50]; 3 were Indian[34,42,51]; one was Native American[18]; with no reports coming from Arab region. Although there is a high possibility that those with no description of their race were Caucasian, only 3 were clearly noted to be of white race[7,28,30,37,41,43-46,49].

Intracranial Locations

PCGs have been reported to occur anywhere in the cranial cavity. The most common locations were the cerebral convexity, followed by supra and para-sellar region, then the posterior fossae region. They are almost always dural based lesions, with only 3 reported cases without any dural attachments occurred in the insula, the cerebellar hemisphere and parietal white matter, respectively[25,31,39]. Only one case had multiple intracranial lesions[34], and another had multiple brain and spinal cord simultaneous involvement[48].

Clinical Presentations

While the presenting symptoms often correlated with the location, headache remains the most common presenting symptom. Seizure disorders were the presenting symptoms in 10 patients[23,25,27,28,34,36,39,44,49]. Diabetes insipidus was a common finding with suprasellar lesions[22,24,31]. Five (15%) patients had associated pulmonary and/or mediastinal PCGs[18,29,30,34,44], while one had a maxillary sinus PCGs without any intraorbital or intracranial extension [36]. In two of the cases with pulmonary PCGs, the cerebral lesion presented 5 years after the surgical treatment of the pulmonary lesion[35,44].

Laboratory Findings

Of the 15 cases with detailed information on complete blood count (CBC) [7,16-18,21,25,30,33,34,36,39,40,49], a hypochromic and microcytic anemia was reported in 4 (25%). In addition, mild leukocytosis[17,30,39,40] and thrombocytosis[18,40] were also documented. Preoperative erythrocyte sedimentation rate (ESR) values were documented for only 4 patients with values ranging between 58 and 70 mm/hr[16,18,20,40]. Preoperative immunoglobulinopathy was demonstrated in 10 patients of a thoroughly investigated 13 patients[7,16,18,21,25,30,33,34,40,49]. The most common abnormality was elevation of IgG (n=8), IgA (n=6) and IgM (n=6) levels. Of these 13 patients, 5 were checked postoperatively and four had normalized IgG values[7,18,25,40,49]. West and colleagues were the only researchers to test their patients for complement values, which revealed an increase in both in C3 and C4 factors[40]. The postoperative C4 levels returned to normal values, while C3 remained slightly elevated.

Radiological Diagnosis

All the reported cases except one, who had cerebral angiogram, had a preoperative CT scan. On non-enhanced CT scan, PCGs often appeared as hyperdense lesions causing mild to diffuse perilesional cerebral edema. In two cases, PCGs appeared hypodense[19,39] and rarely as an isodense lesion[26]. The contrast enhanced CT scan often demonstrated marked homogenous enhancement. In two cases, there was a peripheral cystic enhancement[19,30]. Calcification was described in one case only[49] and hyperostosis of the adjacent bone was never noted as a radiological finding, although Redondo-Fernandez *et al.,* mentioned it in their report as a surgical finding[16]. Hyperostosis in the case reported by Mirra *et al.,* was due to the coexisting frontal meningioma[36]. Twenty-five patients had detailed description of the preoperative MRI scan, among these the PCGs appeared as iso- to hypointense on T1 and hypointense on T2 MRI scans[7,20,25,30,33,35,44,46-51]. The low signal intensity has been suggested to result from the calcifications, the presence of free radicals produced by active inflammatory cells as well as the numerous small hemorrhages and the fibrous components. The enhancement pattern after gadolinium contrast injection was homogenous, except for the case in which mucus-like fluid filled cystic PCG[30]. The suggested preoperative radiological diagnosis was a meningioma in all the cases with dural-based lesions. Three cases were exceptional: the insular lesion in a 60-year-old man without any dural attachment and with significant perilesional edema was thought to represent a secondary brain tumor[25]. The radiological diagnosis of 2 cases with 4th ventricular PCGs was thought to be medulloblastoma, ependymoma or astrocytoma[31,50]. Aozasa *et al.,* suspected that their case of a 76-year-old male

with hypodense and peripherally enhancing suprasellar mass was either a craniopharyngioma or a pituitary adenoma[19]. Hildebrandt *et al.,* thought of a chondroma in their first case with a suprasellar tumor[29]. Positron emission tomography (PET) scan findings in one case demonstrated marked accumulation of 11 C-methyl-L-methionine in the frontally located PCGs[35]. However, it was not clear from that report how PET scan findings could have contributed to the diagnosis.

Histopathological Studies

The histological description of PCGs was classically of a mixed inflammatory response dominated by plasma cells in the background of a collagenous stroma in the absence of microorganisms. Stroma in the case reported by Nazek *et al.,* was hyalinized and contained giant-cells[37]. Necrosis was uniformly absent in the reported cases except for the one reported by Gochman *et al.,* who had identified several foci of necrosis between the collections of plasma cells, which contained granular debris[28]. The Immunohistochemical examination was reported for all but three patients and remains the main diagnostic tool of PCGs[16,18,27]. The staining for lambda and kappa light chains was positive as well as heavy chains. The cerebral parenchyma in direct contact with the PCGs revealed reactive gliosis [18,27,28,49]. In the cases reported by Tang *et al.,* and Gochman *et al.,* the brains were infiltrated by numerous collections of inflammatory cells, not only lymphoplasmacytoid cells but also neutrophils[18,28]. Gochman *et al.,* also reported vascular wall changes in the adjacent brain parenchyma in the form of intimal hyperplasia[28].

Differential Diagnosis

A wide variety of lesions may have clinical and radiological features similar to that of PCGs, but the most common tumor to be considered is meningioma. In the cases by reported Brandsma *et al.,* one case was first diagnosed as meningioma, but 2 years later the patient required a reoperation and the diagnosis of PCGs was made[45]. A second patient was treated for a CNS B-cell lymphoma, which proved 11 years later to be PCGs on re-examination of the pathological specimen. The diagnosis in the third patient was considered tuberculosis meningitis because pachymeningitis were found together with a high protein level in the cerebrospinal fluid and a positive Mantoux test. Despite detailed histological examination, the following are a variety of inflammatory lesions that can mimic the histological picture of PCGs:

1. **Lymphoplasmocytoid meningioma (LPM):** Horten *et al.,* (1979) first described a peculiar subtype of meningioma with plasma cell infiltrates[52]. They are rare and less than 20 cases were reported in literature[52-56]. This

entity encompasses a range of inflammatory reaction together with a range of meningeal cell proliferation. The cell population even within different LPM cases could vary from predominant meningeal cells with massive plasma cell infiltrates to small foci of meningothelial cells within predominant inflammatory cells. Such a wide spectrum may create some uncertainty and difficulty in differentiating LPM from PCG. In such cases, electron microscopic (EM) and immunohistochemical examinations and the awareness and experience of the pathologist has the most important impact for an accurate diagnosis. On EM, meningothelial cells, known for their interdigitating process and well-formed desmosomes are demonstrated. Immunohistochemical staining for EMA is the most accurate and useful in detecting the meningeal cells, as well as for Vimentin, cytokeratin and S-100.

2. **Intracranial solitary plasmocytoma:** Are rare lesions reported in literature (15 cases) by Benli and Inci which should also be distinguished from PCGs[52]. They are composed of a monotonous mature plasma cell population without the mixed inflammatory appearance of PCGs. In Benli and Inci's review, the presenting age ranged from 30 to 70 years (mean: 49.5 years), with a striking 4:1 female preponderance[57]. Their immunohistochemical examination displays monoclonal proliferation of plasma cells, in contrast to polyclonality of PCGs.

3. **Sinus histocytosis with massive lymphadenopathy (SHML):** Should also be considered in the differential diagnosis of PCGs. Eleven patients were reported in the literature[58]. Rosai and Dorfman (1969) first described this entity, which has somewhat confusing nomenclature since the lymph node involvement is not a requirement[59]. The etiology is still obscure, although speculation revolves around two possibilities, Epstein Barr virus infection and/or abnormality of the host immune response. They occur commonly in black children and there is a two-fold male preponderance. They could have neutrophilic leukocytosis, elevated ESR and polyclonal hypergamma-globulinaemia. SHML and PCGs share many similar features such as proliferation of plasma cells, small lymphoid aggregates and even presence of Russell bodies. However, the main cell population in and SHML would be mature histiocytes.

4. **Histocytosis-X (H-X):** Which combines three inflammatory disorders: eosinophilic granulomas; Letterer-Siwe disease and Hans-Schüller-Christian syndrome may mimic several clinical and even histological features of PCGs. The etiology of this inflammatory disease process is unknown. The proliferating and pathognomonic cells in H-X are Langerhans' histiocytes like PCGs, children and young adults are most commonly affected. Radiographically, an infiltrative lesion is more common with H-X. The disease process rarely manifests as an isolated dural-based lesion[60-61]. H-X has a predilection for the

hypothalamo-pituitary axis and causes endocrinologic symptoms (like diabetes insipidus) similar to PCGs. Considering the fact that our review has documented a relative propensity of PCGs for supra and parasellar region, in cases with such tumors the possibility of H-X should be kept in mind. Histologically, H-X infiltrate contains a significant number of Langerhans' histiocytes and a mixed population of inflammatory cells including plasma cells. Focal aggregates of eosinophils are a very important diagnosis. At the ultrastructural level, the diagnostic feature is racket-shaped bodies also called Birbeck's granules.

5. **Castleman's disease (CD):** Is a rare lymphoproliferative disorder that usually arises from mediastinal lymph nodes[62]. The etiology is unknown, but it is considered a chronic inflammatory process possibly of infectious origin. However, the pathogenesis must be quite different from other inflammatory processes since CD has been reported to transform into plasmocytoma and even to malignant lymphoma[62]. Intracranial involvement is most unusual with only 6 cases reported up to date[63-65]. The clinical and radiological diagnosis, except the one reported by Lacombe *et al.,* was a meningioma[66]. The age ranged from 25- to 82-years (mean: 47.8-years), and two-thirds were females. On histological examination, CD consists of lymphoid follicles and interfollicular areas filled with large clusters and sheets of mature plasma cells and smaller lymphocytes. Of 6 patients with intracranial lesions, 3 had plasma-cell type lesions[63-66], 2 had hyaline-vascular lesions[64] and one had an intermediate lesion[64]. Of those 5 cases with immunoassaying, plasma cells were found to be polyclonal in 4 and monoclonal in 1.

Treatment

Surgery was performed in 44 cases of the reported PCGs, all but two underwent craniotomy, and in 28, PCGs were gross totally resected. Two patients with suprasellar lesions were considered for transsphenoidal surgery [24,30]. In five patients (2 with cavernous sinus lesions, 1 each with suprasellar, tentorial and frontal skull base lesions) surgery was limited to a biopsy. While in another subset of six patients (3 with suprasellar, 1 with anterior clinoid, 1 with basal temporal and another with entire flax lesions) a partial to subtotal resection could be accomplished. External radiation therapy (XRT) following surgery was considered necessary for two of those undergoing biopsy[24,35] and six of those with subtotal excision of the tumor[20-22,37,41,46]. The radiation doses ranged between 20-54 Gy (mean: 40 Gy). Tang *et al.,* was the only one to give his patient XRT after total excision because they suspected the lesion to be metastasis from the lung mass[18]. Late XRT for recurrence was considered in only one patient who initially underwent a partial excision[29]. The remaining

biopsied lesions (n=3) which were not considered for XRT were treated with postoperative steroids for 2-, 2- and 12-months respectively[7,30]. Shah and McClain have usefully treated a 14-year-old girl with PCGs, involving the cavernous sinus and the pterygopalatine and infratemporal fossae, with chemotherapy (methotrexate and 6-mercaptopurine) after recurrence following radiation therapy and steroids[41].

Outcome

Follow-up was mentioned in 44 cases, ranging from 3 weeks to 8 years. No patient with a total excision recurred at a mean follow-up period of 16.5 months. In patients with known residual PCGs, one declined treatment[21]. Steroid therapy resulted in decrease of the size in one[31], while in another it remained unchanged at 2 years follow-up[30]. There were 3 deaths, one accidental, one patient died due to postoperative meningitis from trans-sphenoidal surgery, and one died from pulmonary thromboembolism [17,24,31]. Autopsy was possible in two of these three cases, and in both no other systemic lesions were demonstrated[17,31].

Conclusions

The author reports six cases of PCGs and reviews the literature of this rare entity for a better understanding of these atypical lesions. Patients, particularly children, presenting with meningioma-like lesions should be studied if possible with detailed imaging for staging and serum immunoglobulin levels. Detailed histopathological studies are very important tools in providing accurate diagnosis and for excluding other intracranial inflammatory lesions. Total excision invariably leads to cure, however less than complete excisions complemented with radiation, steroid therapy, and chemotherapy has been shown to be effective. None has progressed to a neoplastic.

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الورم الحُبيبي لخلايا مصل الدم في الجهاز العصبي المركزي

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المستخلص. إنّ الورم الحبيبي لخلايا مصل الدم في الجهاز العصبي المركزي نادراً ما يحدث ، وهو من الأورام الالتهابية الكاذبة غير المعروف سببها ، والتي غالبًا ما تتميز بتكاثر ورم حميد في خلايا مصل الدم. يصف المؤلف علاج ستة من المرضى الذين لديهم أورام حبيبية لخلايا مصل الدم ، وكان نصف عدد هؤلاء المرضى من الذكور الذين تراوحت أعمارهم ما بين ٩ ـ ٤٣ سنة ومتابعتهم لمدة ٤٠ شهراً ، وقد أظهرت الفحوصات الإشعاعية لما قبل إجراء الجراحة إلى وجود ثلاثة أورام في السطح المحدب من المخ ، وثلاثة أخرى في الحفرة الخلفية القريبة من المخيخ . تم استئصال كامل الورم لثلاثة مرضى ، وتم استئصال جزئي منه في مريضين ، وتم علاجهما بجرعات من مادة الستروويد والعلاج الإشعاعي بعد العملية الجراحية . وكان أحد المرضى يعاني من ورم في الجزء الصلب المنحدر من الجمجمة ، وتم أخذ عينة منه عبر الأنف من خلال جهاز الملاح الجراحي وأُتبع بالعلاج الإشعاعي مع استجابته للعلاج جزئيًا . راجع المؤلف كل ماكُتب عن ندرة وجود هذا الورم ، وأعدّ تقاريراً لحوالي ٤٦ حالة تعاني من الورم الحُبيبي لخلايا مصل الدم في الجهاز العصبي المركزي ، نشرت في عدة مجلات طبية . وتمت مناقشة الفحوصات السريرية والإشعاعية والنسيجية مع تأكيد التباين في التشخيص وطريقة العلاج .