·临床病理报告·

IgG4相关性脑膜病变

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【摘要】 **目的**　探讨IgG4相关性脑膜病变的临床病理学特征以及诊断与鉴别诊断要点。**方法与结果**　男性患者，49岁，临床表现为头痛近2年并进行性加重1月余，头部MRI显示左侧顶叶占位性病变，增强扫描可见“脑膜尾征”，手术完整切除病灶。组织学形态，左侧顶叶硬脑膜和脑实质大量胶原纤维增生，其间散在灶状细胞浸润，多为较成熟的浆细胞，部分浆细胞内可见匀质红染的Russell小体，其间散在淋巴细胞和少量嗜酸性粒细胞，局部可见小灶状坏死，间质纤维母细胞和小血管增生，未见包膜，病变累及周围脑组织。免疫组织化学染色，浆细胞胞质弥漫性表达IgG和IgG4（>60%）、胞膜表达CD38和CD138，淋巴细胞胞膜表达CD3、CD4或CD20。血清IgG4为1.05 g/L。最终病理诊断为（左侧顶叶）IgG4相关性脑膜病变可能性大。术后予抗感染、抗癫痫、营养支持治疗，症状明显好转，出院后未按医嘱定期随访。**结论**　IgG4相关性脑膜病变临床少见，且缺乏典型临床表现和特征性影像学改变，术前诊断与鉴别诊断困难，血清IgG4水平升高是其诊断的重要线索，明确诊断仍需依靠特征性的组织学形态和免疫组织化学表型。

【关键词】 免疫球蛋白G；　脑膜；　病理学；　免疫组织化学

IgG4-related meningeal disease

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【**Abstract**】 **Objective**　To explore the clinical and pathological characteristics, diagnosis and differential diagnosis of IgG4-related meningeal disease. **Methods and Results**　A 49-year-old male patient suffered from headache for nearly 2 years and the symptom was aggravated progressively for over one month. MRI revealed space-occupying lesion in left parietal lobe, with irregular signal and clear borderline. Contrast-enhanced MRI showed homogeneous enhancement, and obvious meningeal thicking just like "dural tail sign". The patient underwent operation, and the lesion was totally removed. Histologically, it showed a large amount of hyperplastic collagen fibrous tissue with inflammatory cells, including a large number of plasmocytes, as well as scattered lymphocytes and a small number of eosinophilic granulocytes. Russell bodies which were homogenously positive for eosin could be seen in some plasmocytes. Focal necrosis, and proliferation of interstitial fibroblasts and small vessels were found. The lesion had no capsules and invaded surrounding tissues. Immunohistochemical staining showed the plasmocytes were diffusely positive for IgG and IgG4 (> 60%), and were positive for CD38 and CD138 on the membrane. Lymphocytes were positive for CD3, CD4 or CD20 on the membrane. The serum IgG4 level was 1.05 g/L. Final pathological diagnosis was IgG4-related meningeal disease in left parietal lobe. After operation, the patient received anti-infectious, anti-epileptic and nutrition support treatment, and the symptoms were markedly improved. The patient was discharged after 26 d, but was lost to follow-up. **Conclusions**　IgG4-related meningeal disease is a rare disease. Due to the atypical imaging features, it may be difficult to differentiate IgG4-related meningeal disease from other diseases with prominent inflammatory cells and

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stromal fibrosis. The elevated serum IgG4 level may provide diagnostic cues. However, a definite diagnosis depends on characteristic histological and immunohistochemical features.

【Key words】 Immunoglobulin G; Meninges; Pathology; Immunohistochemistry

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IgG4相关性疾病是新近定义的自身免疫性疾病类型，最初被Sarles等[1]报告为自身免疫性胰腺炎（AIP），病理学检查可见胰腺系统性硬化。后续研究发现，此类患者除外胰腺的其他器官如胆道、唾液腺、眼眶、淋巴结、后腹膜、纵隔、软组织和中枢神经系统均存在相似病变，且具有相同的组织学形态特点，即间质广泛纤维化、弥漫性淋巴细胞和浆细胞浸润伴闭塞性静脉炎，以及血清IgG4水平升高。2010-2012年召开的一系列国际会议已达成共识，接受这一疾病类型并正式命名为IgG4相关性疾病。我们报告1例IgG4相关性脑膜病变患者，通过复习文献，对其临床表现、影像学特点、组织学形态和免疫组织化学表型进行分析，以期提高对该病的诊断与鉴别诊断能力。

病历摘要

患者 男性，49岁，主因头痛近2年并进行性加重1月余，于2014年10月17日入南方医科大学南方医院。患者近2年前（2013年2月）无明显诱因出现头痛（具体情况不详），无恶心、呕吐，无视物模糊、双眼肿胀，无头晕，无发热、寒战，无胸痛，无咳嗽、咳痰，无胸闷、气短。1个月前头痛症状加重，至当地医院就诊，头部CT检查显示，左侧顶叶占位性病变，予脱水治疗（具体方案不详）后，头痛症状好转。为求进一步诊断与治疗，至我院就诊。患者自发病以来，精神疲惫，睡眠、饮食尚可，大小便正常，体重无明显改变。

既往史、个人史及家族史 患者既往体格健康，否认急性传染性疾病病史，否认外伤史和手术史，否认食物和药物过敏史，否认输血史；无疫区、疫水等接触史，无毒物和放射性物品等接触史。个人史和家族史无特殊。

体格检查 患者体温36.3 ℃、脉搏88次/min、呼吸12次/min、血压105/66 mm Hg（1 mm Hg = 0.133 kPa）。神志清楚，语言流利，主动体位，步态平稳。双侧瞳孔等大、等圆，直径约3 mm，对光反射、调节反射和辐辏反射灵敏，鼻唇沟对称，伸舌居中。脑神经检查未见异常。脑膜刺激征阴性，病理征未引出。四肢肌力和肌张力无明显异常。

辅助检查 实验室检查各项指标未见明显异常。头部MRI显示，左侧顶叶占位性病变，最大横截面积约2.20 cm × 1.70 cm，宽基底贴近颅骨，边界清晰，信号不均匀，T_1WI呈等信号，T_2WI呈高或低信号，增强扫描病灶明显均匀强化，硬脑膜明显增厚，可见“脑膜尾征”，相邻颅骨骨质信号不均匀，余未见明显异常（图1）。^{18}F-脱氧葡萄糖（^{18}F-FDG）PET扫描显示，左侧顶枕叶放射性摄取减少伴左侧顶枕叶弧形放射性聚集，右侧上颌窦、上颌骨牙槽突放射性摄取增加。综合考虑颅内感染可能性大。

诊断与治疗经过 临床诊断为左侧顶叶占位性病变，性质待查。遂于2014年10月27日于气管插管全身麻醉下行左侧顶叶占位性病变切除术，术中可见颅骨下硬脑膜增厚，打开硬脑膜，可见病变呈灰白、灰红色，大小约5.00 cm × 4.60 cm × 0.50 cm，形状不规则，质地柔软，血供丰富，无包膜，与周围脑组织界限不清，部分侵犯脑实质，完整切除病变，行组织病理学检查。（1）大体标本观察：手术切除标本为灰黄、灰褐色硬脑膜组织，大小约5.00 cm × 4.50 cm × 0.50 cm，并可见一灰黄、灰褐色突起，大小约2.50 cm × 2.00 cm × 2.00 cm，质地较韧，无包膜，血供尚可，未见出血。标本经体积分数为10%中性甲醛溶液固定，常规脱水、浸蜡、石蜡包埋，制备4 μm脑组织连续切片，行HE染色和免疫组织化学染色。（2）HE染色：左侧顶叶硬脑膜和脑实质大量胶原纤维增生，部分呈“席纹”样，其间散在灶状细胞浸润（图2a），大部分为较成熟的浆细胞，部分浆细胞内可见匀质红染的Russell小体（图2b），其间散在淋巴细胞和少量嗜酸性粒细胞，局部可见小灶状坏死，间质纤维母细胞和小血管增生（图2c），未见包膜，病变侵及周围脑组织（图2d）。（3）免疫组织化学染色：ABC三步法检测试剂盒由北京中杉金桥生物技术有限公司提供，检测所用抗体包括S-100蛋白

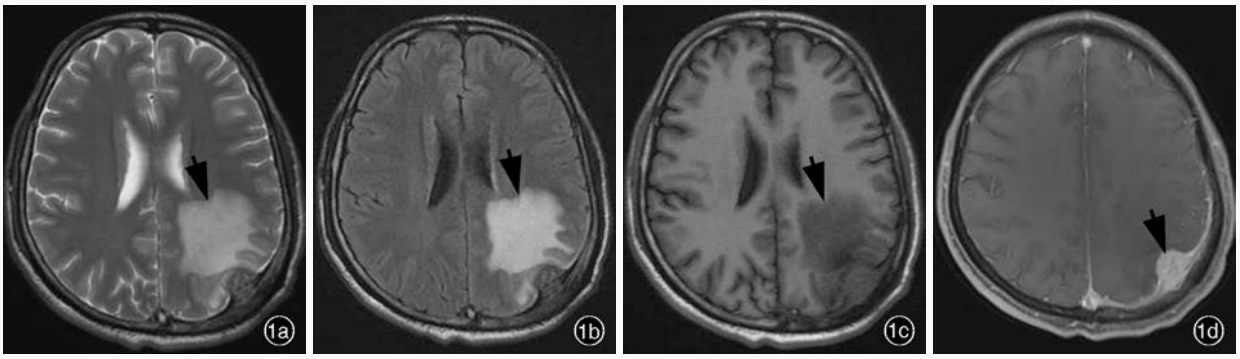

图1　头部MRI检查所见　1a　横断面T_2WI显示，左侧顶枕叶不规则高或低混杂信号影（箭头所示），界限清晰，可见大面积片状水肿　1b　横断面FLAIR成像显示，病灶呈高或低混杂信号（箭头所示），界限清晰，周围可见大面积片状水肿　1c　横断面T_1WI显示病灶呈稍低信号（箭头所示）　1d　横断面增强T_1WI显示，病灶呈明显均匀强化，邻近脑膜线样强化，并同时可见“脑膜尾征”（箭头所示）

Figure 1　Head MRI findings　Axial T_2WI (Panel 1a) and FLAIR (Panel 1b) showed irregular mixed signal (high- or low-intensity signal) of the lesion in left parieto-occipital lobe with a clear borderline and large sheet edema (arrows indicate). Axial T_1WI showed slight hypointensity signal of the lesion (arrow indicates, Panel 1c). Axial contrast-enhanced T_1WI showed homogenous enhancement of the lesion, and linear enhancement of surrounding meninges. "Dural tail sign" could be seen (arrow indicates, Panel 1d).

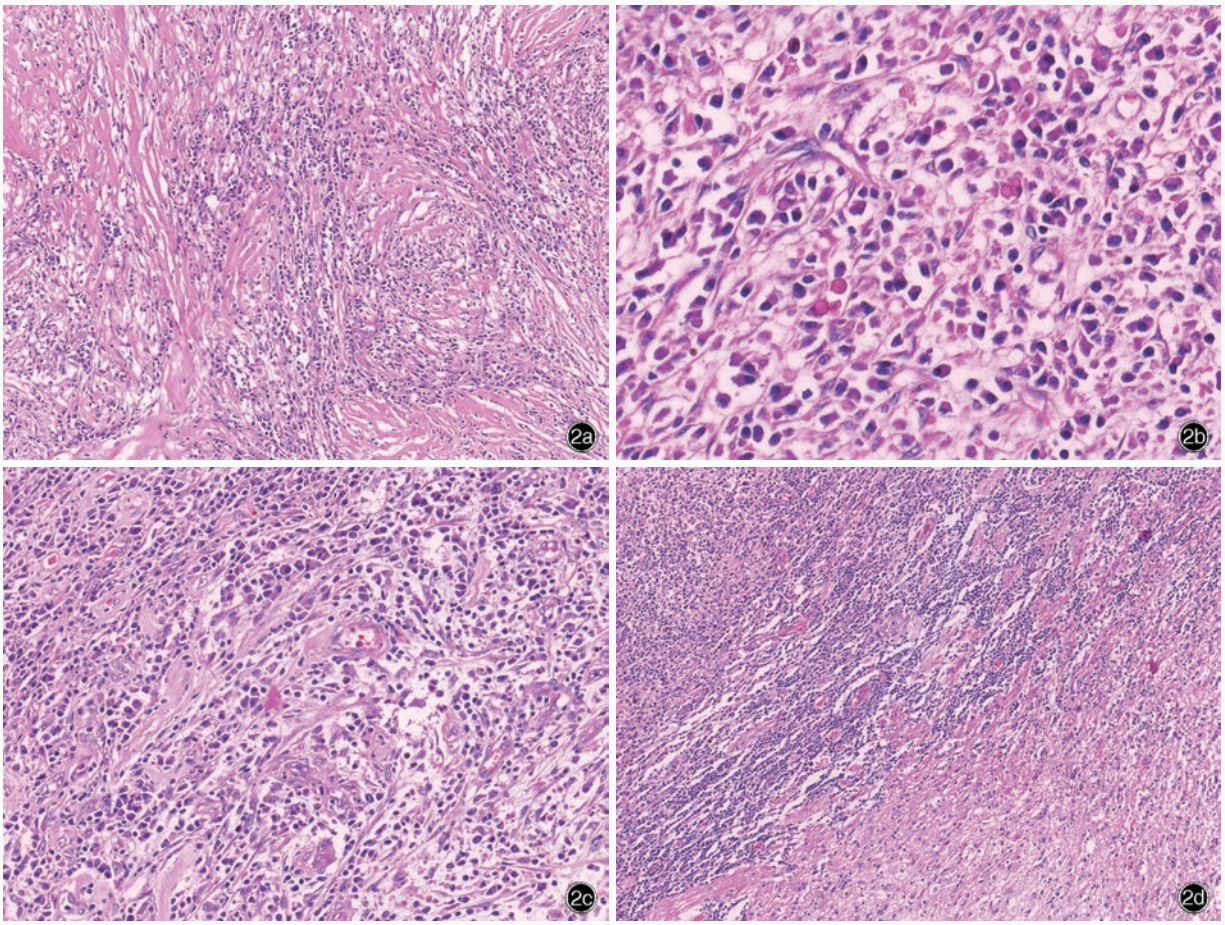

图2　光学显微镜观察所见　HE染色　2a　可见胶原纤维增生呈“席纹”样，伴淋巴细胞和浆细胞浸润　×100　2b　浸润的炎性细胞中可见较多成熟浆细胞，部分浆细胞胞质内可见Russell小体　×400　2c　可见小血管增生，呈闭塞性静脉炎表现　×200　2d　病变侵及周围脑组织，与周围脑组织界限不清　×100

Figure 2　Optical microscopy findings　HE staining　There were a large amount of hyperplastic storiform-arranged collagen fibrous tissue with infiltration of lymphocytes and plasmocytes.　×100 (Panel 2a)　Most of the inflammatory cells were mature plasmocytes, and Russell bodies could be seen in the cytoplasm of some plasmocytes.　×400 (Panel 2b)　There were hyperplastic small vessels resembling obliterative phlebitis.　×200 (Panel 2c)　The lesion involved surrounding brain tissues.　×100 (Panel 2d)

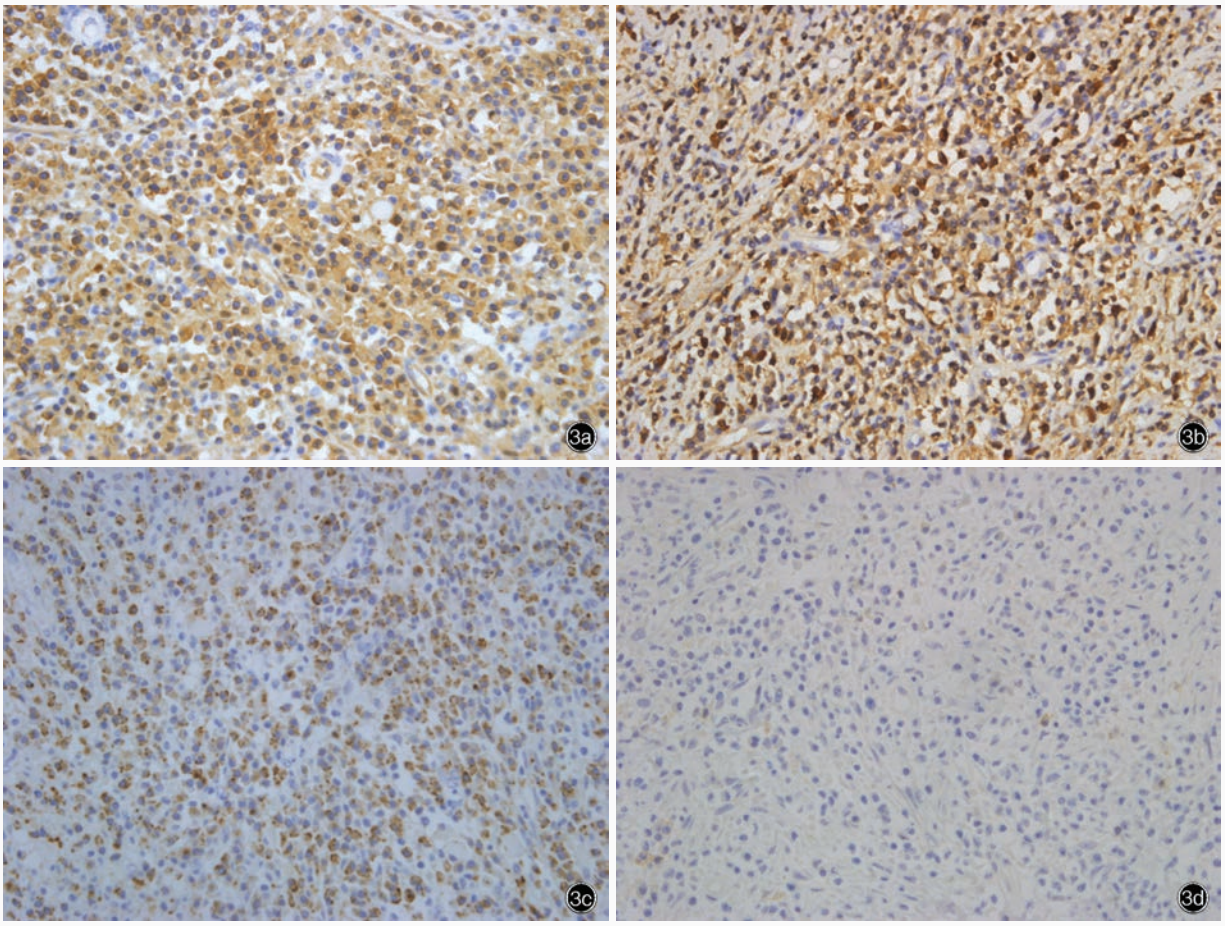

图3　光学显微镜观察所见　免疫组织化学染色(ABC三步法)　×400　3a　浆细胞胞质IgG呈弥漫性阳性　3b　浆细胞胞质IgG4呈弥漫性阳性(>60%)　3c　浆细胞胞膜CD138阳性　3d　浆细胞不表达EMA

Figure 3　Optical microscopy findings　Immunohistochemical staining (ABC)　×400　The cytoplasm of plasmocytes was diffusely positive for IgG (Panel 3a) and IgG4 (>60%, Panel 3b). The membrane was positive for CD138 (Panel 3c). Plasmocytes were negative for EMA (Panel 3d).

(S-100,1∶150)、平滑肌肌动蛋白(SMA,1∶150)、CD3(1∶100)、CD4(1∶100)、CD20(1∶100)、CD38(1∶100)、CD68(1∶100)、CD138(1∶100)、波形蛋白(Vim,1∶200)、上皮膜抗原(EMA,1∶100)、IgG(1∶100)和IgG4(1∶100)均购自北京中杉金桥生物技术有限公司。结果显示,梭形细胞胞质弥漫性表达Vim、部分表达SMA,淋巴细胞胞膜表达CD3、CD4或CD20,组织细胞胞质表达CD68,神经元胞质表达S-100,浆细胞胞质弥漫性表达IgG(图3a)和IgG4(>60%,图3b)、胞膜表达CD38和CD138(图3c),不表达EMA(图3d)。遂建议行血清免疫球蛋白测定,结果显示,血清IgG4为1.05 g/L(>1.35 g/L)。最终病理诊断为(左侧顶叶)IgG4相关性脑膜病变可能性大。术后予抗感染(头孢曲松钠2 g/d静脉滴注)、抗癫痫(丙戊酸钠400 mg/d静脉滴注)、营养支持治疗,未再出现头痛、头晕、癫痫发作。患者住院26 d,出院时一般状况良好,但未按医嘱定期随访。

讨　论

IgG4相关性疾病是以血清IgG4水平升高、受累组织IgG4$^+$浆细胞浸润和纤维化为特征的疾病[2],好发于中老年男性,激素治疗效果较好[3]。最初被Sarles等[1]报告为自身免疫性胰腺炎,2003年Kamisawa等[4]首次提出"IgG4相关性疾病"的概念,2010年*Autoimmun Rev*对其正式命名[5]。自身免疫性胰腺炎并非单纯胰腺炎,而是一种系统性疾病伴IgG4$^+$浆细胞、CD4$^+$T细胞和CD8$^+$T细胞浸润,研究提示该病为自身免疫性疾病,故归入IgG4相关性疾病范畴,表明该病发病机制与自身免疫因子相关[6]。血液循环中存在的辅助性T细胞2(Th2)与IgG4相

关性疾病密切相关，前者分泌的IL-4、5和13参与IgG4相关性疾病的特异性免疫应答[7]。IL-10在IgG4相关性疾病中的表达高于其他自身免疫性疾病[8]，亦可诱导IgG4$^+$细胞的形成[9]。

IgG4相关性疾病的特征性病理改变为淋巴细胞和浆细胞浸润，伴纤维化；嗜酸性粒细胞浸润，伴血清IgG4水平升高；常合并闭塞性静脉炎；大量淋巴细胞浸润时可形成肿瘤样团块或滤泡；可形成“席纹”样纤维化；组织结构和功能相对完整；免疫组织化学染色IgG4$^+$浆细胞浸润明显（IgG4$^+$浆细胞/IgG$^+$细胞＞40%）。结合临床表现、血清IgG4水平和组织病理学结果可以分为确诊（definite）、很可能（probable）、可能（possible）等诊断层次[10]。IgG4相关性疾病可发生于全身多个组织和器官，如胰腺、泪腺、唾液腺、垂体、甲状腺、肺部、主动脉、冠状动脉、肝脏、胆道、肾脏、前列腺、皮肤和淋巴结等。

发生于中枢神经系统的IgG4相关性疾病多侵及垂体[11-12]，亦可见关于IgG4相关性脑膜炎的文献报道[13-14]。IgG4相关性脑膜病变临床表现不典型，缺乏特征性影像学改变，故血清IgG4水平升高是诊断的重要线索，但明确诊断仍需依靠组织病理学检查。该例患者组织学形态可见病变组织内大量胶原纤维增生，其间散在灶状细胞浸润，大多为较成熟的浆细胞，部分浆细胞内可见均质红染的Russell小体，其间散在淋巴细胞和少量嗜酸性粒细胞，局部可见小灶状坏死，间质纤维母细胞和小血管增生，未见包膜，病变侵及周围脑组织，符合IgG4相关性脑膜病变的病理改变。

IgG4相关性脑膜病变应注意与以下疾病相鉴别：（1）富于淋巴细胞浆细胞型脑膜瘤。不具有典型脑膜瘤的影像学特征，如“脑膜尾征”、脑外肿瘤“镶嵌征”、宽基底实性肿物等，但可见脑膜广泛性不均匀增厚，增强扫描可见明显强化征象[15]。组织学形态表现为大量淋巴细胞和浆细胞浸润，并可见不同比例的典型脑膜上皮细胞区域；免疫组织化学染色显示，Vim、S-100、EMA呈强阳性，可资鉴别。（2）Rosai-Dorfman病（RDD）。好发于儿童和青少年，常累及淋巴结，亦可累及硬脑膜。组织学形态表现为淋巴细胞和浆细胞聚集，并可见较多组织细胞，组织细胞胞质透亮或空泡化，具有显著吞噬特性；免疫组织化学染色，具有吞噬特性的组织细胞CD68和S-100呈强阳性，可资鉴别。（3）Castleman病。尤其是浆细胞型Castleman病的特征性改变为滤泡间区各级浆细胞片状增生，可见Russell小体，浆细胞表达免疫球蛋白λ和κ轻链，同时可见少量淋巴细胞和免疫母细胞。累及硬脑膜时常为局灶型Castleman病，临床主要表现为持续性头痛，可伴有视力障碍和癫痫发作等[16]。IgG4相关性疾病可伴发Castleman病样改变[17]。（4）炎性肌纤维母细胞瘤/炎性假瘤。好发于儿童和青少年，肺部为最常发生部位，主要由增生的纤维母细胞和肌纤维母细胞构成，其间可见大量炎性细胞浸润，多为淋巴细胞、浆细胞和组织细胞。梭形细胞（纤维母细胞和肌纤维母细胞）不同程度表达Vim、SMA、肌特异性肌动蛋白（MSA）、肌动蛋白和结蛋白（Des）。

由于IgG4相关性脑膜炎临床相对少见，若MRI呈现“鼠尾征”，术前极易误诊为脑膜瘤。术后组织病理学检查可因病变组织内存在广泛淋巴细胞和浆细胞浸润伴纤维母细胞增生而误诊为炎性肌纤维母细胞瘤等疾病。此时，血清IgG4水平升高是诊断的重要线索，明确诊断仍需依靠组织病理学检查。该例患者免疫组织化学染色淋巴细胞胞膜表达CD3、CD4或CD20，浆细胞胞膜表达CD38和CD138，证实病灶中的炎性细胞呈多克隆性，此外，IgG和IgG4（＞60%）均呈弥漫性阳性；血清IgG4为1.05 g/L，均有助于IgG4相关性脑膜病变的诊断与鉴别诊断。充分了解疾病的临床表现、影像学特点和组织病理学特征，方可避免可能出现的诊断陷阱而得出正确结论。

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Society for Neuro-Oncology Conference on Meningioma

Time: June 17–18, 2016

Venue: Toronto, Ontario, Canada

Website: www.soc–neuro–onc.org/

The Society for Neuro-Oncology (SNO) Conference on Meningioma will be held on June 17–18, 2016 at the Yorkville InterContinental Hotel in Toronto, Ontario, Canada. This meeting is being jointly organized by SNO and the Consortium on Meningioma in Toronto. Chaired by Dr. Gelareh Zadeh, this 2-day educational event seeks to bring together a focused multidisciplinary group of researchers and clinician scientists who are committed to improving the outcome of patients with meningioma through the translation of research into clinical practice. A mix of oral and poster presentations, as well as invited speakers will be part of this exciting conference.

Neurosurgeons, interventional neuroradiologists, neuro-oncologists, medical oncologists, radiation oncologists, neuropathologists, scientists/laboratory researchers, industry representatives and trainees with an interest in the research and treatment of meningiomas are encouraged to attend.

12th European Congress on Epileptology

Time: September 11–15, 2016

Venue: Prague, Czech Republic

Email: prague@epilepsycongress.org

Website: www.epilepsyprague2016.org/

The 12th European Congress on Epileptology (ECE) will take place in Prague, Czech Republic on September 11–15, 2016. The congress is now a landmark in the epilepsy community agenda and the Prague 2016 promises to be innovative and engaging. The congress is organized by the Commission on European Affairs (CEA) of the International League Against Epilepsy (ILAE).