·临床病理报告·

间变型多形性黄色瘤型星形细胞瘤

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【摘要】 **目的**　报道1例男性间变型多形性黄色瘤型星形细胞瘤患儿的临床资料，探讨其临床病理学、免疫表型、基因突变特征，以及诊断与鉴别诊断要点。**方法与结果**　男性患儿，11岁，头痛伴左上肢无力15 d。头部CT和MRI显示右侧颞叶和基底节区巨大占位性病变，提示胶质瘤。遂行右侧颞叶和基底节区占位性病变切除术，术中可见肿瘤呈囊实性，实性部分呈灰黄色，质地柔软，血供丰富，无包膜，与周围组织界限清晰。术中冰冻病理学提示低级别胶质瘤，遂分块全切除肿瘤。组织学形态观察，肿瘤细胞呈多形性，由梭形和圆形星形胶质细胞以及单核细胞和多核瘤巨细胞组成，核分裂象罕见；局部可见较成熟的神经元或节细胞分化成分，伴淋巴细胞浸润；部分区域肿瘤细胞呈间变特征，细胞密度增加，异型性明显，以圆形和梭形细胞为主，核分裂象＞5个/10高倍视野，血管内皮细胞增生，伴血管周围假“菊形团”样结构，局灶性坏死。免疫组织化学染色，低级别肿瘤细胞胞质表达胶质纤维酸性蛋白（GFAP）和BRAF V600E、胞质和胞核表达S-100蛋白、胞膜表达CD34，少数肿瘤细胞胞质表达突触素和非磷酸化神经丝重链SMI-32，Ki-67抗原标记指数为3%；低级别和高级别肿瘤细胞胞核均表达P53；高级别肿瘤细胞胞质表达GFAP和BRAF V600E，Ki-67抗原标记指数为30%。网织纤维染色可见肿瘤细胞周围包绕基底膜样物质。基因检测显示，低级别和高级别肿瘤均存在*BRAF* V600E杂合突变。**结论**　2016年世界卫生组织中枢神经系统肿瘤分类将间变型多形性黄色瘤型星形细胞瘤定义为核分裂象＞5个/10高倍视野，属WHOⅢ级，预后较WHOⅡ级多形性黄色瘤型星形细胞瘤差。鉴别诊断主要包括胶质母细胞瘤、毛细胞型星形细胞瘤和节细胞胶质瘤，尽管上述肿瘤临床表现、组织学形态、免疫表型和基因突变有重叠，但生物学行为、治疗及预后各异。

【关键词】 黄瘤病；　星形细胞瘤；　间变；　免疫组织化学；　病理学

Anaplastic pleomorphic xanthoastrocytoma

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【**Abstract**】 **Objective**　To investigate the clinicopathological features, immune phenotype and gene mutation characteristics, and diagnosis or differential diagnosis of anaplastic pleomorphic xanthoastrocytoma (PXA). **Methods and Results**　A 11-year-old male patient presented with more than half month of headache, and left upper limb weakness. Cranial CT and MRI revealed a large space-occupying lesion in the right parietal lobe and basal ganglia which was suggested as glioma. During operation the tumor was examined. It was a cystic-solid lesion. The solid part was soft and greyish yellow with rich blood supply and without membrane, and the boundary was clear. Intraoperative freezing pathologic examination showed the tumor was a low grade glioma. The right parietal glioma was completely removed piece by piece under the microscopy. Histologically, the tumor cells were polymorphism, including spindle or round astrocytes, monocytes and multinuclear tumor giant cells. Mitoses were rarely seen. Differentiation of mature neuronal cells or ganglion cells with lymphocyte infiltration were seen in focal region. In some regions, tumor cells were anaplastic, and cellularity were increased. Atypical round or spindle cells were seen, and atypical mitoses ＞5/10 high power field (HPF) were found. Microvascular proliferation, perivascular pseudorosettes and localized necrosis were also evident. Immunohistochemically, tumor cells were positive for glial fibrillary acidic protein (GFAP), BRAF V600E, S-100 protein (S-100), CD34, synaptophysin (Syn), non-phosphorylation neurofilament heauy chain SMI-32 and P53, but negative for isocitrate dehydrogenase 1

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(IDH1), neurofilament protein (NF) and epithelial membrane antigen (EMA). Ki-67 labeling index was about 3% in low grade tumor cells, while Ki-67 labeling index was about 30% in high grade tumor cells. In reticular fibre tissue staining, a lot of reticular fibre tissue were seen. *BRAF* V600E heterozygous mutation c.1799A > T was detected by Sanger sequencing. **Conclusions** Anaplastic PXA in grade Ⅲ is defined as mitoses > 5/10 HPF in World Health Organization (WHO) classification of tumors of the central neuvous system, 2016. Its prognosis is worse than grade Ⅱ tumor. The differential diagnosis from glioblastoma (GBM), pilocytic astrocytoma (PA) and ganglioglioma (GG) should be kept in mind, because all of them having some overlaps in clinicopathological presentations, imaging manifestation, immunophenotype features and genetic mutation, but quite different in their biological behavior, treatment and prognosis.

【**Key words**】 Xanthomatosis; Astrocytoma; Anaplasia; Immunohistochemistry; Pathology

多形性黄色瘤型星形细胞瘤(PXA)是临床罕见的原发性星形细胞肿瘤,占所有星形细胞肿瘤的1%以下,具有特征性临床病理学和影像学特点,好发于儿童和青年,中位发病年龄为22岁,通常位于大脑浅表位置,累及软脑膜和脑组织,约98%发生于幕上,尤以颞叶常见,多数患者存在长期癫痫病史[1-2]。组织病理学以肿瘤细胞多形性为特征,伴单核细胞或多核瘤巨细胞、嗜酸性小体,血管周围可见丰富淋巴细胞浸润,肿瘤组织附着于网状纤维。2016年世界卫生组织(WHO)中枢神经系统肿瘤分类取消"伴间变特征的多形性黄色瘤型星形细胞瘤"命名,将核分裂象≥5个/10高倍视野(HPF)者定义为"间变型多形性黄色瘤型星形细胞瘤",属WHOⅢ级,伴或不伴坏死[1,3]。近年报道的间变型多形性黄色瘤型星形细胞瘤病例逐渐增多[4-11]。本文报道1例发生于颞叶和基底节区的间变型多形性黄色瘤型星形细胞瘤患儿,并复习相关文献,初步探讨其临床病理学特点、免疫表型和*BRAF* V600E突变形式,并与组织学形态、免疫表型和基因突变相似的肿瘤进行鉴别诊断。

病历摘要

患儿 男性,11岁。主因头痛伴左上肢无力15 d,于2016年1月15日入院。患者15 d前无明显诱因出现头痛伴左上肢无力,右侧肢体正常,无肢体抽搐、意识障碍、大小便失禁。外院头部CT检查显示,右侧颞叶和基底节区巨大囊实性占位性病变伴幕上积水,考虑高级别胶质瘤(图1)。头部MRI显示,右侧颞叶和基底节区巨大囊实性占位性病变伴幕上积水,T_1WI可见病变界限相对清晰,呈以低信号为主的混杂信号影,周围水肿带呈低信号;T_2WI可见病变呈不均匀高信号;FLAIR成像呈稍高信号;增强扫描病变呈环形和斑片状不均匀明显强化,囊性变区未见明显强化(图2)。为求进一步诊断与治疗,遂至我院就诊,门诊以"右侧颞叶巨大占位性病变"收入院。患者自发病以来,精神良好,睡眠、饮食可,体重无明显变化,大小便正常。

既往史、个人史及家族史 既往身体健康,身高和体重发育正常,智力发育正常,否认手术、外伤和输血史,否认药物和食物过敏史,预防接种史不详。生于山西省,久居本地,无疫区居住史,无疫情、疫水接触史,无牧区、矿山、高氟区、低碘区居住史,无化学性物质、放射物、毒物接触史,无毒品接触史,无吸烟、饮酒史。父母和兄长身体健康,家族中无传染性疾病和遗传性疾病病史。

体格检查 患儿体温36.4 ℃,脉搏80次/min,呼吸19次/min,血压102/66 mm Hg(1 mm Hg = 0.133 kPa);身高150 cm,体重35 kg,体重指数(BMI)15.60 kg/m^2,发育正常,营养良好,面容表情正常,自主体位;神志清楚,语言流利,查体合作,胸腹部检查未见异常。神经系统检查:双侧瞳孔等大、等圆,直径约2.50 mm,对光反射灵敏,眼球各向活动充分;鼻唇沟对称,口角无歪斜,伸舌居中;左上肢肌力4级、余肢体5级,肌张力均正常,无肌萎缩、肌束颤;痛温觉和轻触觉正常,两点辨别觉、图形觉、位置觉和音叉震动觉正常;双侧快复轮替动作、指鼻试验、跟-膝-胫试验稳准,Romberg征阴性,直线行走试验阴性;病理征阴性,脑膜刺激征阴性。自主神经系统检查:全身皮肤温度和湿度适中,皮肤弹性好,皮肤划痕试验阴性。

诊断与治疗经过 实验室检查各项指标均于正常值范围。结合外院影像学检查,临床诊断为胶质瘤。完善术前准备,于2016年1月19日在全身麻醉下行MRI导航下右侧颞叶胶质瘤切除术。术中可见肿瘤位于大脑皮质下1 cm处,呈囊实性,实性部分呈灰黄色,质地柔软,血供丰富,无包膜,与周

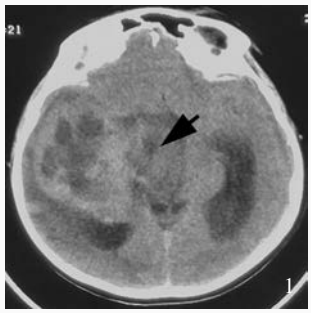
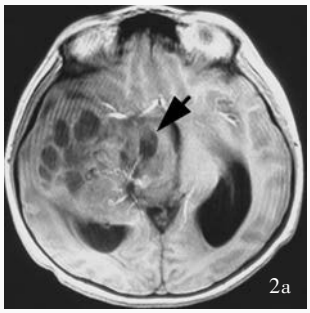
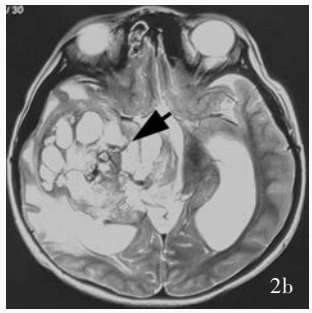
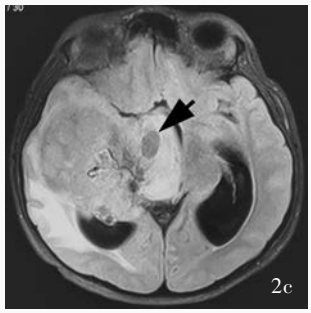
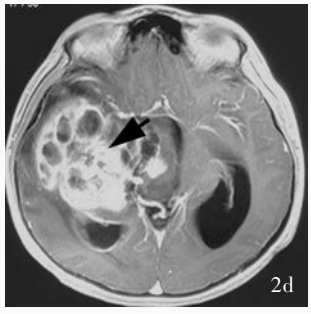

图1　头部CT检查显示，右侧颞叶和基底节区巨大囊实性占位性病变，呈不规则混杂密度影，其内可见多房性低密度囊性变区，周围可见片状低密度水肿带（箭头所示）；右侧侧脑室受压，中线结构向左侧偏移　**图2**　头部MRI检查所见　2a　横断面T_1WI显示，病变界限相对清晰，呈以低信号为主的混杂信号影，周围水肿带呈低信号（箭头所示）　2b　横断面T_2WI显示，病变呈不均匀高信号（箭头所示）　2c　横断面FLAIR成像显示病变呈稍高信号（箭头所示）　2d　横断面增强T_1WI显示，病变呈环形和斑片状不均匀明显强化（箭头所示），囊性变区未见明显强化

Figure 1　Cranial CT scan showed a huge space-occupying cystic-solid lesion in right temporal lobe and basal ganglia (arrow indicates), which was an irregular mixed density with multiple low-density cystic changes, surrounded by patchy low-density edema. Obvious space-occupying effect was found. The right ventricle was compressed and the midline was shifted to left.　**Figure 2**　Cranial MRI findings. Axial T_1WI showed the lesion boundary was relatively clear, and mixed signal lesion with predominant hypointense signal were seen (arrow indicates). Periphal edema zone with low-signal was seen (Panel 2a). Axial T_2WI showed a heterogeneous hyperintense signal lesion (arrow indicates, Panel 2b). Axial FLAIR imaging displayed slight hyperintense signal lesion (arrow indicates, Panel 2c). Axial enhanced T_1WI revealed heterogeneous ring or patchy enhancement lesion (arrow indicates), whereas enhancement was not seen in cystic region (Panel 2d).

围组织界限清晰，瘤体主要位于右侧颞叶并突入侧脑室，中线结构向左侧偏移。切取实性部分行快速冷冻病理学检查，术中报告低级别胶质瘤，遂于手术显微镜下分块全切除肿瘤，行组织病理学检查。（1）大体标本观察：术中和术后送检标本为灰白色破碎组织，大小分别为1.50 cm×1.00 cm×0.30 cm和9 cm×5 cm×2 cm，质地柔软，血供丰富，无包膜。经体积分数为10%中性甲醛溶液固定、常规脱水、石蜡包埋、4 μm连续切片，分别行HE染色、免疫组织化学染色和网状纤维染色。（2）HE染色：肿瘤组织可见两种组织学形态，术中送检组织和部分术后送检组织肿瘤细胞呈多形性，由梭形和圆形星形胶质细胞以及单核细胞和多核瘤巨细胞组成，核分裂象罕见，可见较成熟的神经元或节细胞分化成分，伴淋巴细胞浸润，局灶性泡沫样细胞聚集，未见血管内皮细胞增生和坏死灶，病变累及软脑膜，呈现低级别肿瘤（WHOⅡ级）组织学形态改变（图3，4）。部分术后送检组织肿瘤细胞密度增加、异型性明显，以圆形和梭形星形胶质细胞成分为主，不典型核分裂象>5个/10高倍视野，可见血管内皮细胞增生，伴血管周围假“菊形团”样结构，局灶性坏死，呈高级别肿瘤（WHOⅢ级）组织学形态改变（图5）。（3）免疫组织化学染色：采用SP二步法，检测用试剂盒购自北京中杉金桥生物技术有限公司，检测用抗体包括突触素（Syn，1∶100），胶质纤维酸性蛋白（GFAP，1∶400），R132H-突变的异柠檬酸脱氢酶1（IDH1，1∶200），P53（1∶75），S-100蛋白（S-100，1∶2000），CD34（1∶100），CD68（1∶100），神经微丝蛋白（NF，1∶100），非磷酸化神经丝重链SMI-32（1∶7500），上皮膜抗原（EMA，工作液），BRAF V600E（工作液）和Ki-67抗原，均购自北京中杉金桥生物技术有限公司。结果显示，低级别肿瘤细胞胞质表达GFAP（图6a）和BRAF V600E（图6b）、胞质和胞核表达S-100、胞膜表达CD34（图6c），少数肿瘤细胞胞质表达Syn（图6d）和SMI-32（图6e），Ki-67抗原标记指数为3%（图6f），不表达NF和EMA；高级别和低级别肿瘤细胞胞核均表达P53（图7a）；高级别肿瘤细胞胞质表达GFAP（图7b）和BRAF V600E（图7c），不表达R132H-突变的IDH1和CD34，Ki-67抗原标记指数为30%（图7d）。（4）特殊染色：网织纤维染色显示肿瘤细胞周围包绕基底膜样物质。（5）基因检测：采用聚合酶链反应（PCR）扩增*BRAF* V600E位点，再行Sanger测序，结果显示，低级别和高级别肿瘤均存在*BRAF* V600E杂合突变（图8）。最终病理诊断为：（右侧颞叶和基底节区）间变型多形性黄色瘤型星形细胞瘤（WHOⅢ级）。术后未辅

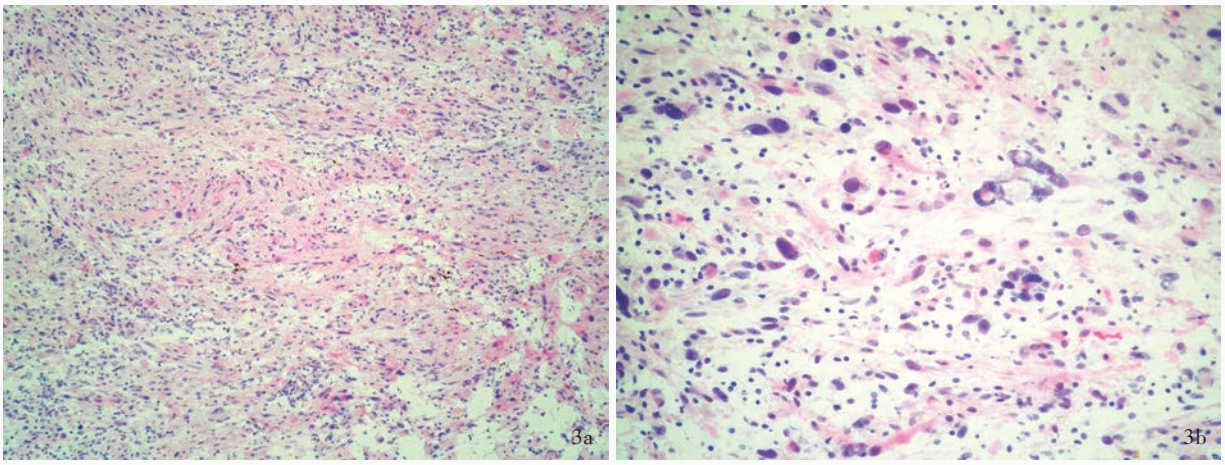

图3 术中送检标本组织学形态光学显微镜观察所见 HE染色 3a 肿瘤细胞呈多形性，由梭形细胞以及单核细胞和多核瘤巨细胞组成 ×100 3b 可见单核细胞和多核瘤巨细胞，肿瘤细胞异型性明显 ×200

Figure 3 Optical microscopy findings of histological patterns of sample of intra-operation HE staining Pleomorphic histological appearance of tumor was characterized by spindled cells being intermingled with mononucleated or multinucleated giant cells (Panel 3a). ×100 Nuclear pleomorphism and bizarre were seen in mononucleated or multinucleated tumor giant cells (Panel 3b). ×200

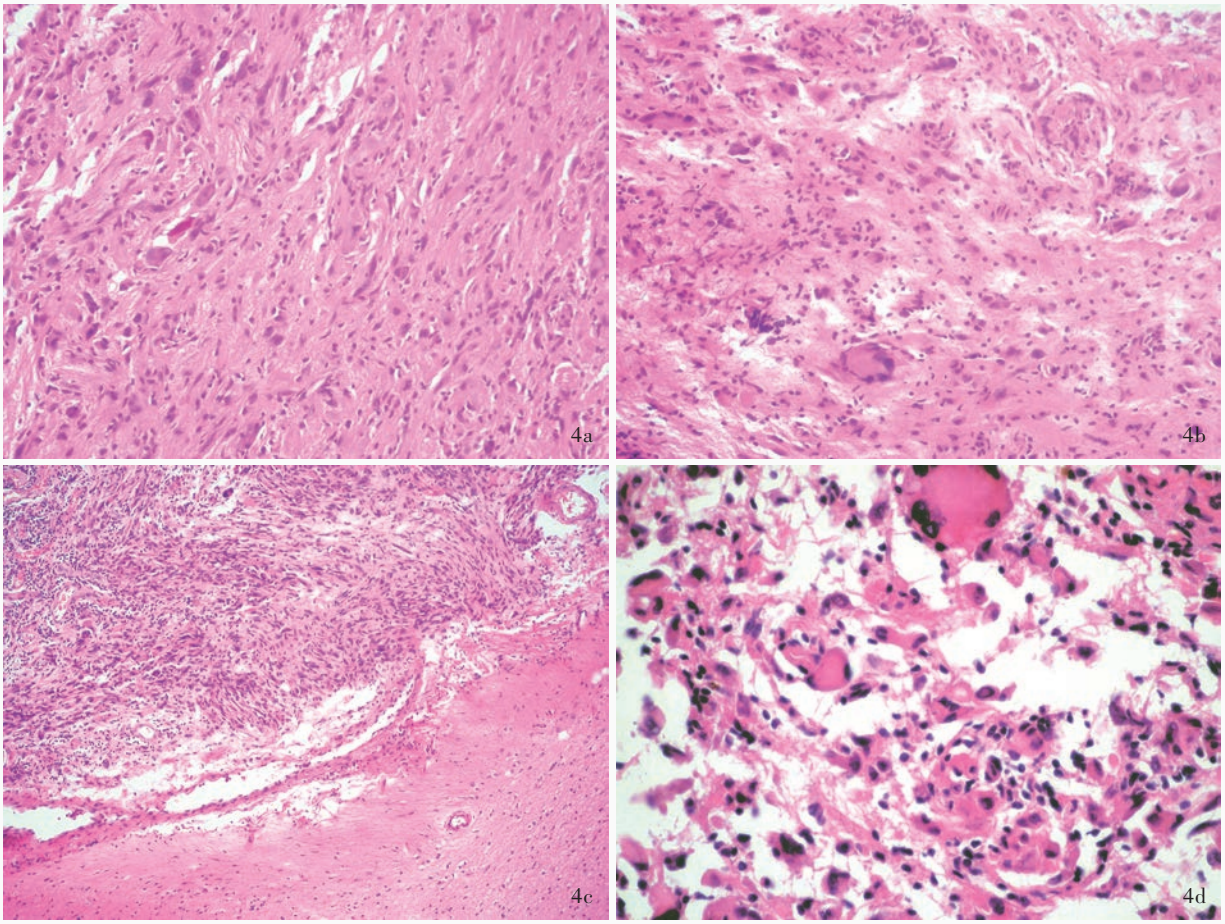

图4 部分术后送检标本(低级别肿瘤)组织学形态光学显微镜观察所见 HE染色 4a 肿瘤组织由梭形细胞以及单核细胞和多核瘤巨细胞组成 ×200 4b 多核瘤巨细胞胞核呈"马蹄"样排列 ×200 4c 肿瘤位于脑表浅位置，侵及软脑膜，与大脑皮质分界清晰 ×100 4d 部分区域可见较多单核细胞和多核瘤巨细胞聚集 ×400

Figure 4 Optical microscopy findings of histological patterns of sample (low grade tumor) of surgical excision HE staining Tumor was composed of spindled cells, mononucleated and multinucleated tumor giant cells (Panel 4a). ×200 Nuclei of multinucleated tumor giant cell were arranged in a horseshoe-like pattern at edge of the cell (Panel 4b). ×200 Tumor cells invaded pia mater (leptomeningeal) and clearly delineated from the underlying cerebral cortex (Panel 4c). ×100 Prominent mononucleated or multinucleated tumor giant cells were seen in part of the region (Panel 4d). ×400

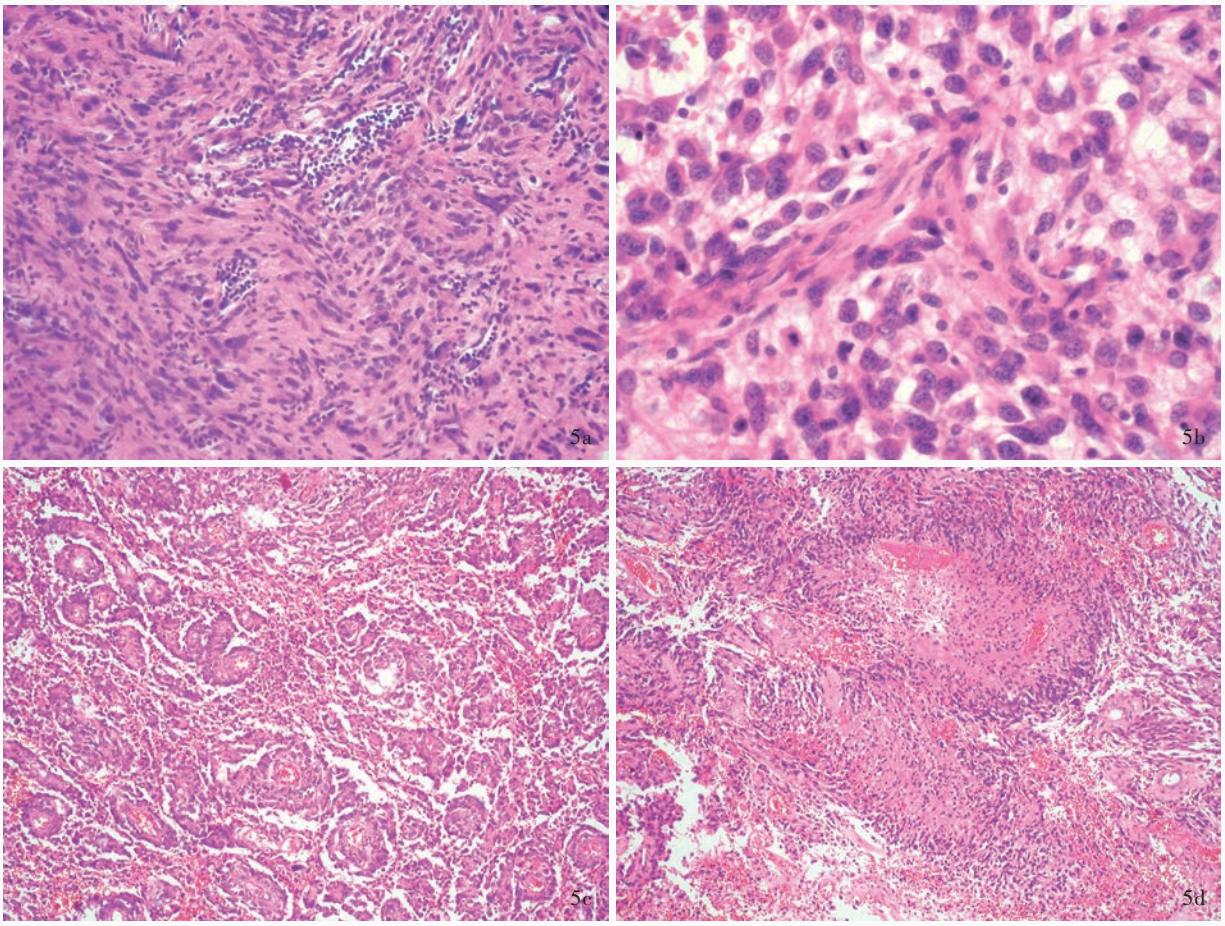

图5　部分术后送检标本(高级别肿瘤)组织学形态光学显微镜观察所见　HE染色　5a　可见典型多形性黄色瘤型星形细胞瘤成分　×200　5b　肿瘤细胞呈相对一致的圆形或卵圆形,可见不典型核分裂象　×400　5c　肿瘤细胞围绕血管周围排列,可见血管周围假"菊形团"样结构　×100　5d　可见局灶性坏死,肿瘤细胞呈"栅栏"样排列　×100

Figure 5　Optical microscopy findings of histological patterns of sample (high grade tumor) of surgical excision　HE staining　Typical histologic feature of PXA, WHO grade Ⅱ was seen (Panel 5a).　×200　Atypical mitotic activity was seen in the round or oval shape tumor cells (Panel 5b).　×400　Tumor cells were arranged around the vascular as perivascular pseudorosettes (Panel 5c).　×100　Focal necrosis was seen and tumor cells were arranged in column (Panel 5d).　×100

助放射治疗和药物化疗。患者共住院22 d,出院时病情平稳,恢复良好。出院后9个月失访。

讨　论

Kepes等[2]于1979年首次报告并命名多形性黄色瘤型星形细胞瘤,是临床罕见的原发性星形细胞肿瘤,具有特征性组织病理学特点。肿瘤细胞呈梭形细胞、单核细胞和多核瘤巨细胞的多形性特点,伴胞体宽大的多核瘤巨细胞,胞质表达GFAP,肿瘤细胞周围包绕致密网织纤维,是一种特殊类型的星形细胞肿瘤,故命名为"多形性黄色瘤型星形细胞瘤"。多形性黄色瘤型星形细胞瘤仅占颅内肿瘤的0.09%,占所有星形细胞肿瘤的<1%[1],好发于儿童和青年,亦有老年患者的报道[12],无性别和种族差异。

多形性黄色瘤型星形细胞瘤组织病理学特点主要表现为[13]:(1)肿瘤细胞呈多形性,即梭形细胞、肥胖细胞以及单核细胞和多核瘤巨细胞多种成分,胞核和核仁大小不一,常见核内包涵体(INIs)。(2)可见黄色瘤型星形细胞,即脂质聚集于肿瘤细胞内,融合并充满或占据大部分胞质,其星形胶质细胞特性经GFAP染色证实。(3)可见网织纤维,即肿瘤细胞周围包绕的基底膜样物质,经网织纤维染色证实。此外,还可见嗜酸性小体,间质或血管周围淋巴细胞和浆细胞浸润,后者形成血管周围淋巴"袖套"或局灶性聚集。

在2007年WHO中枢神经系统肿瘤分类中,多形性黄色瘤型星形细胞瘤属WHOⅡ级,伴明显核分

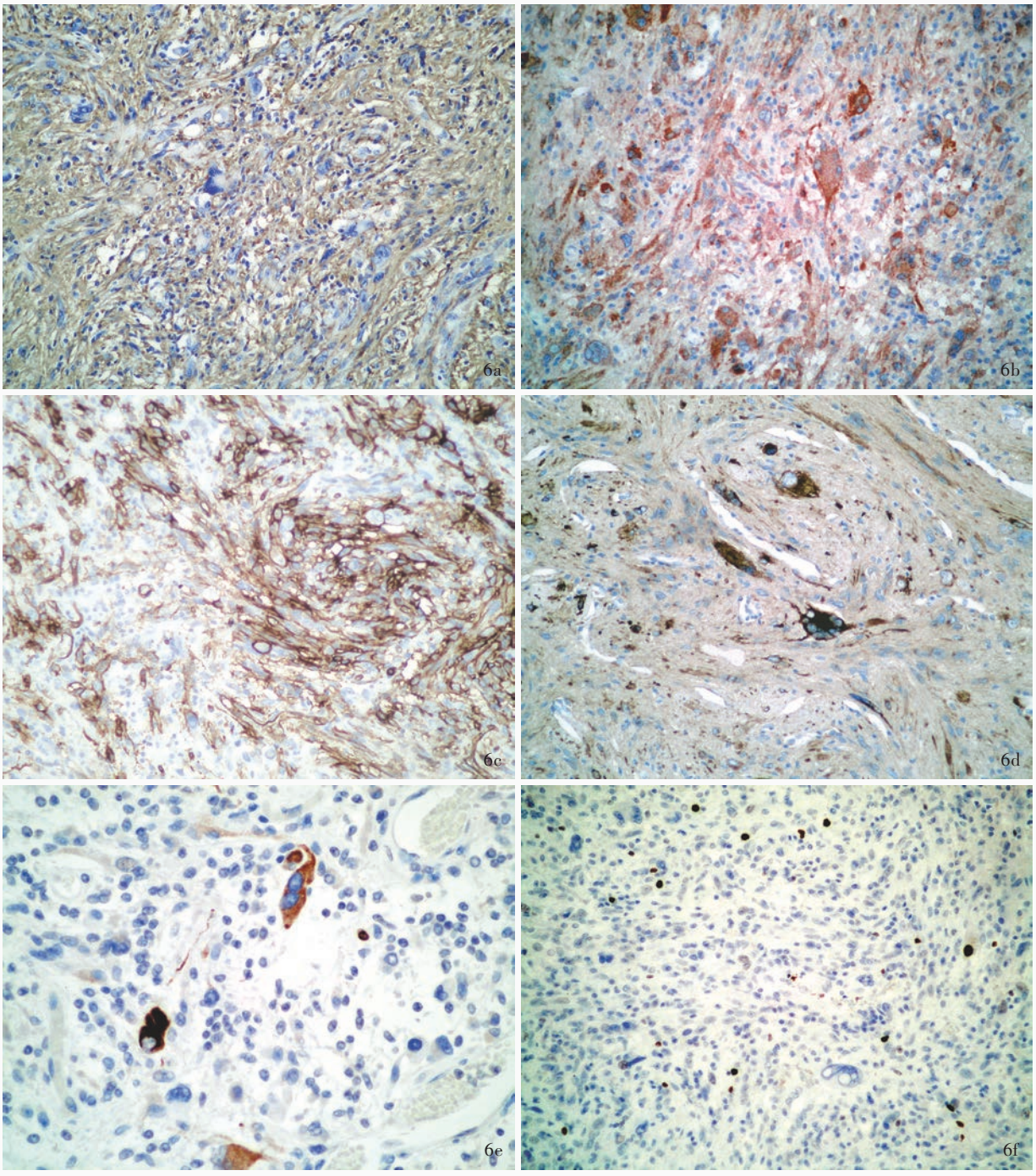

图6 低级别肿瘤光学显微镜观察所见 免疫组织化学染色(SP二步法) 6a 肿瘤细胞胞质表达GFAP ×200 6b 肿瘤细胞胞质表达BRAF V600E ×200 6c 肿瘤细胞胞膜表达CD34 ×200 6d 少数肿瘤细胞胞质表达Syn ×200 6e 个别神经元样细胞胞质表达非磷酸化神经丝重链SMI-32 ×400 6f Ki-67抗原标记指数约3% ×100

Figure 6 Optical microscopy findings of low grade tumor Immunohistochemical staining (SP) The cytoplasm of tumor cells was positive for GFAP (Panel 6a) and BRAF V600E (Panel 6b). ×200 Tumor cells membrane was positire for CD34 (Panel 6c). ×200 The cytoplasm of a few tumor cells was positive for Syn (Panel 6d). ×200 The cytoplasm of individual neuron-like cells was positive for SMI-32 (Panel 6e). ×400 Ki-67 labeling index was about 3% (Panel 6f). ×100

裂象或坏死者命名为"伴间变特征的多形性黄色瘤型星形细胞瘤"[14]。2016年WHO中枢神经系统肿瘤分类取消该命名,将间变型多形性黄色瘤型星形细胞瘤(WHOⅢ级)列为一种明确类型,其诊断标准为:核分裂象≥5个/10高倍视野,可伴坏死[13]。间变型多形性黄色瘤型星形细胞瘤病变部位、临床表

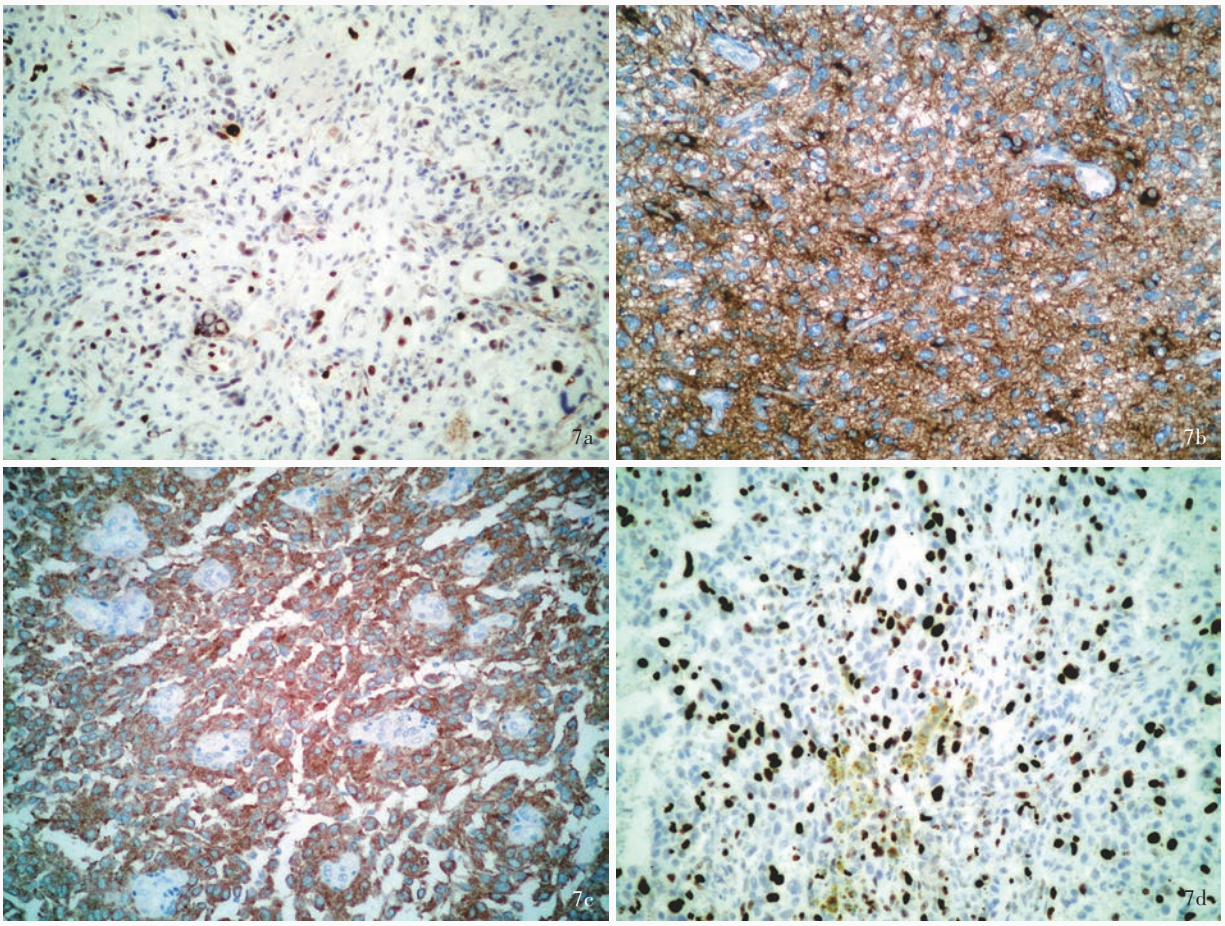

图7　高级别肿瘤光学显微镜观察所见　免疫组织化学染色(SP二步法)　7a　肿瘤细胞胞核表达P53　×100　7b　肿瘤细胞胞质表达GFAP　×200　7c　肿瘤细胞胞质表达BRAF V600E　×200　7d　Ki-67抗原标记指数约30%　×100

Figure 7　Optical microscopy findings of high grade tumor　Immunohistochemical staining (SP)　Tumor cells nucleus was positive for P53 (Panel 7a).　×100　The cytoplasm of tumor cell was positive for GFAP (Panel 7b) and BRAF V600E (Panel 7c).　×200　Ki-67 labeling index was about 30% (Panel 7d).　×100

现和影像学特点均与WHOⅡ级的多形性黄色瘤型星形细胞瘤相似[13]。组织病理学典型特征是核分裂活跃,呈局灶性或弥漫性分布。常见的坏死灶与高核分裂活性密切相关。微血管增生不明显,常与核分裂活性和坏死相关。与WHOⅡ级的多形性黄色瘤型星形细胞瘤相比,间变型多形性黄色瘤型星形细胞瘤细胞多形性不明显,突出表现为弥漫性和浸润性生长模式[13]。有文献报道,间变型多形性黄色瘤型星形细胞瘤可见小细胞、纤维样、上皮样/横纹肌样成分[15-17]。

多形性黄色瘤型星形细胞瘤细胞表达GFAP和S-100[1];存在明显的神经元分化特征,表达相应的神经元标志物[18],如Syn、嗜铬素A(CgA)、NF、β-微管蛋白(β-tubulin)和微管相关蛋白-2(MAP-2);部分表达P53。大多数多形性黄色瘤型星形细胞瘤核分裂象罕见或缺如,Ki-67抗原标记指数<2%[1]且随着肿瘤恶性程度的增加而升高,间变型多形性黄色瘤型星形细胞瘤达10%甚至20%[5]。多形性黄色瘤型星形细胞瘤细胞表达CD34,阳性率达84%,但间变型多形性黄色瘤型星形细胞瘤CD34阳性率下降,仅为44%,可资鉴别[19]。

有50%~78%的WHOⅡ级多形性黄色瘤型星形细胞瘤存在*BRAF*基因突变,尤以*BRAF* V600E突变最为常见,故肿瘤细胞表达BRAF V600E,免疫组织化学染色检测BRAF V600E敏感性和特异性均较高[20]。研究显示,约75% WHOⅡ级多形性黄色瘤型星形细胞瘤存在*BRAF* V600E突变,WHOⅢ级间变型多形性黄色瘤型星形细胞瘤*BRAF* V600E突变率较低(47.4%)[10],且儿童与成人无明显差异[3]。*BRAF* V600E突变亦见于其他原发性中枢神经系统

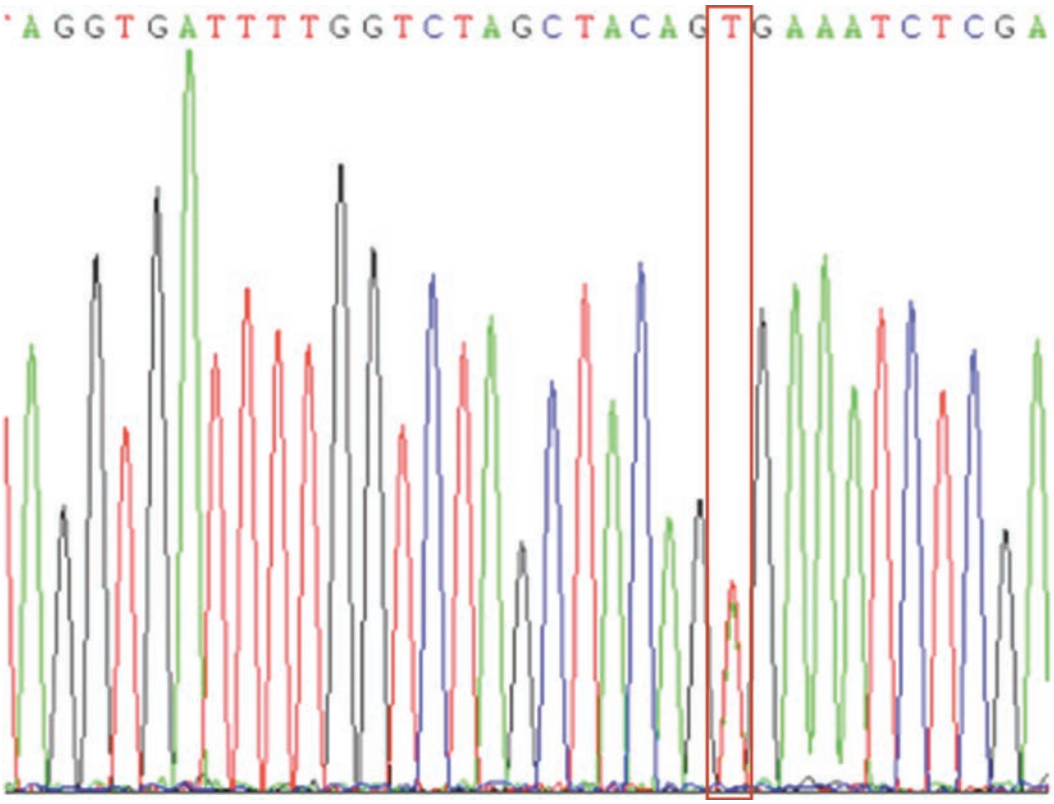

图8 Sanger测序显示，*BRAF*基因存在第15外显子杂合突变c.1799A > T

Figure 8 The 1799 basic group of 15th exon of *BRAF* occurred heterozygosis mutation: A > T.

肿瘤，特别是胶质母细胞瘤（GBM）、毛细胞型星形细胞瘤（PA）和节细胞胶质瘤（GG）[21]。

应注意与胶质母细胞瘤两种亚型巨细胞型胶质母细胞瘤和上皮样胶质母细胞瘤、毛细胞型星形细胞瘤及节细胞胶质瘤相鉴别。（1）巨细胞型胶质母细胞瘤：肿瘤组织以大量异形多核瘤巨细胞、丰富网状纤维和淋巴细胞浸润为特征，亦可见血管周围假“菊形团”样结构，与多形性黄色瘤型星形细胞瘤组织学形态相重叠，应予以鉴别[22]。多形性黄色瘤型星形细胞瘤核分裂象罕见，未见坏死，Ki-67抗原标记指数＜2%，易区分。间变型多形性黄色瘤型星形细胞瘤和巨细胞型胶质母细胞瘤均表现为核分裂活跃，特别是当前者出现广泛坏死时，难以区分。Reifenberger等[19]研究显示，多形性黄色瘤型星形细胞瘤表达CD34，而巨细胞型胶质母细胞瘤不表达，可资鉴别。此外，与多形性黄色瘤型星形细胞瘤不同，巨细胞型胶质母细胞瘤几乎不表达神经元标志物[NF、神经元核抗原（NeuN）、Syn等]，可资鉴别[22]。（2）上皮样胶质母细胞瘤：好发于儿童和青年，肿瘤细胞可见多核瘤巨细胞、脂肪化、促纤维增生反应，约50%患者存在*BRAF* V600E突变[23-24]。二者不同之处在于，间变型多形性黄色瘤型星形细胞瘤缺乏肿瘤细胞形态一致性特点，可见嗜酸性小体[25-26]和局灶性典型低级别多形性黄色瘤型星形细胞瘤成分[13]。（3）毛细胞型星形细胞瘤：约9%患者存在*BRAF* V600E突变，主要见于幕上毛细胞型星形细胞瘤患者[27]，免疫组织化学染色BRAF V600E阳性。二者鉴别诊断要点在于，毛细胞型星形细胞瘤的致密纤维区域由特征性细长突起的双极肿瘤细胞组成，含丰富Rosenthal纤维；稀疏区可见微囊结构和规则的假少突胶质细胞结构[28]，并伴透明样变性的厚壁血管和海绵状血管瘤样等退行性变成分。（4）节细胞胶质瘤：系发育异常的神经元和肿瘤性胶质细胞组成的神经元及混合性神经元-胶质肿瘤，约70%以上的WHO Ⅰ级节细胞胶质瘤位于颞叶，临床症状主要为局限性癫痫发作[29]。肿瘤性神经元的大小和分布差异较大，可见双核神经元、钙化、促纤维增生反应、淋巴细胞浸润和嗜酸性小体；梭形细胞成分与弥漫性星形细胞瘤、毛细胞型星形细胞瘤或多形性黄色瘤型星形细胞瘤等组织学形

态相似。肿瘤性神经元成分表达神经元标志物，如MAP-2、NF、CgA、Syn和CD34；肿瘤性胶质细胞表达GFAP[29-31]。有20%～60%的节细胞胶质瘤存在*BRAF* V600E突变[27,32-33]。节细胞胶质瘤细胞成分缺乏多形性黄色瘤型星形细胞瘤的细胞多形性特点，即梭形细胞及单核细胞和多核瘤巨细胞的混合成分，以及特征性富含脂质的多核瘤巨细胞，可资鉴别。

多形性黄色瘤型星形细胞瘤有相对良性的临床病程。一项纳入74例多形性黄色瘤型星形细胞瘤患者的研究显示，5年无进展生存率和总生存率分别为70.9%和90.4%；手术切除范围是最具意义的肿瘤复发预测因素[3]。间变型多形性黄色瘤型星形细胞瘤患者预后较差、生存期较短，5年总生存率显著低于多形性黄色瘤型星形细胞瘤患者（55.6%对89.4%），且伴坏死的患者5年总生存率低于不伴坏死的患者（42.2%对90.2%）。儿童和成人的5年无进展生存率（67.99%对62.4%）和总生存率无显著差异（87.4%对76.3%）；*BRAF* V600E突变的预后意义不明[3]。多形性黄色瘤型星形细胞瘤患者应进行长期随访。

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The Annual Meeting of American Academy of Addiction Psychiatry 2017

Time: December 7–10, 2017

Venue: San Diego, California, USA

Website: www.aaap.org/annual–meeting

The Annual Meeting of American Academy of Addiction Psychiatry 2017 will be held on December 7–10, 2017 in San Diego, California, USA. The Annual Meeting and Scientific Symposium provide the latest scientific developments in addiction psychiatry for physicians and allied health professionals who treat patients with substance use disorders (SUD) and mental health disorders. The meeting is structured to encourage interaction among clinicians from various disciplines, approaches and settings.

The Meeting aims to recognize emerging issues and trends in addiction psychiatry and be on the forefront of diagnosis and treatment of substance use disorders and co-occurring mental disorders. Participants should be able to: 1) identify how to recognize, diagnose, and treat substance use disorders as they change in society. 2) Increase their competency in using evidence based psychotherapy, medications and other treatments. 3) Improve their knowledge using didactic lectures, skill building workshops, and unique educational formats to support concepts in addiction psychiatry. 4) Increase skills to educate peers, colleagues, trainees, patients and the community about addiction psychiatry.

The Meeting can also provide support and education to addiction psychiatrists and clinicians treating patients with substance use disorders. Therefore, participants should be able to: 1) utilize and promote evidence-based approaches and current treatment guidelines for biopsychosocial treatment of substance use disorders and co-occurring mental disorders. 2) Network with peers and mentors to find support and guidance in the field of addiction psychiatry. 3) Develop and expand current educational curriculum in the field of addiction psychiatry.

The Meeting will demonstrate for trainees the various evidence-based approaches, treatments and settings applicable to the field (or practice) of addiction psychiatry. Trainees should be able to: 1) identify various career paths in addiction psychiatry available to them. 2) Increase their familiarity with career options and pathways by networking with leaders in addiction psychiatry. 3) Enhance their knowledge relevant to early careers in addiction psychiatry.