·CASE ANALYSIS· ·临床病例讨论·

以尿崩症起病累及垂体柄的罕见Rosai-Dorfman病1例

黎露莎[1]，何红晖[1]，赵少俐[1]，初令[2]，金萍[1]

(1. 中南大学湘雅三医院内分泌科，长沙 410013；2. 中南大学湘雅三医院病理科，长沙 410013)

[摘 要] Rosai-Dorfman病(Rosai-Dorfman disease，RDD)是一类罕见的非朗格汉斯细胞组织细胞增生症。2024年8月中南大学湘雅三医院内分泌科收治1例38岁男性，以尿崩症为首发症状，病情迁延15年，逐渐进展为全垂体功能减退症。影像学提示垂体柄明显增粗、2处硬脑膜瘤样结节、多发骨质破坏、少许间质性肺炎及泌尿系统受累。经垂体柄活体组织检查后确诊为RDD，伴有*BRAF*^{V600E}突变，予以BRAF抑制剂达拉非尼靶向治疗和垂体激素替代治疗。治疗8个月后复查头部磁共振，提示垂体柄增粗较前好转，2处硬脑膜瘤样结节消失，且患者生活质量明显改善。对本病例进行回顾性分析，可帮助临床医师对垂体柄增粗进行病因诊断及鉴别诊断，提高对RDD这一罕见疾病的认识。

[关键词] Rosai-Dorfman病；尿崩症；垂体柄增粗；多系统受累；*BRAF*突变

A rare case of Rosai-Dorfman disease involving the pituitary stalk and presenting initially with diabetes insipidus

LI Lusha[1], HE Honghui[1], ZHAO Shaoli[1], CHU Ling[2], JIN Ping[1]

(1. Department of Endocrinology, Third Xiangya Hospital, Central South University, Changsha 410013;
2. Department of Pathology, Third Xiangya Hospital, Central South University, Changsha 410013, China)

ABSTRACT Rosai-Dorfman disease (RDD) is a rare form of non-Langerhans cell histiocytosis. In August 2024, a 38-year-old man was admitted to the Department of Endocrinology, The Third Xiangya Hospital of Central South University. He initially presented with diabetes insipidus, and his condition gradually progressed over a 15-year disease course, eventually leading to panhypopituitarism. Imaging examinations revealed marked thickening of the pituitary stalk, 2 meningioma-like dural nodules, multiple osteolytic lesions, mild interstitial pneumonia, and urinary system involvement. RDD was confirmed by a pituitary stalk biopsy, and a *BRAF*^{V600E} mutation was identified. The patient subsequently received targeted therapy with the BRAF inhibitor dabrafenib in combination with pituitary hormone replacement therapy. Follow-up brain magnetic resonance imaging after 8 months

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第一作者(First author)：黎露莎，Email: lilusha19890806@sina.com, ORCID: 0000-0003-1863-8603
通信作者(Corresponding author)：金萍，教授，Email: ping.jin06@csu.edu.cn, ORCID: 0000-0002-7835-076X
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of treatment demonstrated a reduction in pituitary stalk thickening and complete disappearance of the 2 meningioma-like dural nodules. The patient's quality of life also improved substantially. This retrospective analysis of the case may assist clinicians in determining the etiology and differential diagnosis of pituitary stalk thickening and enhance awareness of RDD as a rare disease.

KEY WORDS Rosai-Dorfman disease; diabetes insipidus; pituitary stalk thickening; multisystem involvement; *BRAF* mutation

临床上中枢性尿崩症和垂体柄增粗可同时出现或先后出现，其病因诊断复杂且困难。很多患者在初次就诊时不一定能得到正确的诊断[1]。本研究报道1例以尿崩症为首发症状、病程长达15年的侵犯垂体柄、硬脑膜、骨骼等多系统的Rosai-Dorfman病(Rosai-Dorfman disease，RDD)患者，以提高内科医师对垂体柄增粗的诊断及鉴别诊断的能力，并认识这一疾病。

1 病例资料

患者，男，38岁，因“口干、多饮、多尿15年余，言语不利1个月余”于2024年8月2日入住中南大学湘雅三医院内分泌科(以下简称“我科”)。患者疾病自然史如图1所示。患者15年前无明显诱因出现口干、多饮、多尿，每日饮水3 000~4 000 mL，夜尿3~4次，曾在外院考虑“肾性尿崩症?”，出院后长期服用氢氯噻嗪和卡马西平，但疗效欠佳，遂于2013年3月14日第1次入住我科，禁水加压试验结果提示禁水后最高尿比重达1.020，尿渗透压616 mOsm/kg，垂体磁共振(magnetic resonance imaging，MRI)平扫及增强未见异常(图2A和2B)，诊断为“精神性烦渴”出院。出院后患者仍感口干多饮多尿，近年饮水量及尿量均为6 000~7 000 mL，夜间需起床上厕所及饮水3~4次。半年前患者自觉体力下降，性欲减退，体毛脱落，体重减轻约15 kg。入院前1个月无明显诱因出现言语不利，主要表现为吐词欠清，无头痛和视力下降，在外院MRI检查提示鞍区占位，为求进一步治疗遂第2次入我科。既往体健，否认传染病史，否认重大外伤、手术史。

体格检查：身高为169 cm，体重为62 kg，体重指数(body mass index，BMI)为21.7 kg/m^2，脉搏为100次/min，血压为125/94 mmHg。发育正常，营养中等，四肢、腋毛及阴毛等部位毛发稀疏，未见皮肤黏膜皮疹，全身浅表淋巴结未扪及肿大，心肺腹未见阳性体征。辅助检查：尿比重<1.005(参考值范围1.005~1.030)；甲状腺功能3项：游离三碘甲状腺原氨酸3.79 pmol/L(参考值范围3.1~6.8 pmol/L)、游离甲状腺素10.72 pmol/L(参考值范围10.3~22.65 pmol/L)、促甲状腺激素2.5 μU/mL(参考值范围0.27~4.2 μU/mL)。促黄体生成素0.1 mU/mL(参考值范围1.7~8.6 mU/mL)、卵泡生成素1.04 mU/mL(参考值范围1.5~12.4 mU/mL)、垂体泌乳素51.49 ng/mL(参考值范围4.04~15.2 ng/mL)、睾酮2.5 ng/dL(参考值范围193~740 ng/dL)。促肾上腺皮质激素(adrenocorticotropic hormone，ACTH)节律：ACTH 8点19.63 pg/mL，0点13.25 pg/mL，16点17.16 pg/mL。皮质醇节律：皮质醇0点1.3 μg/dL，8点2.2 μg/dL，16点2.12 μg/dL；血常规、肝肾功能、电解质、骨代谢标志物均正常。禁水加压试验：禁水8 h后血钠即明显升高至152.7 mmol/L，但尿比重仍波动在1.002~1.004，禁水13 h后予以垂体后叶素10 U皮下注射后1 h测尿比重仍无明显升高。腹部彩色多普勒超声提示：脂肪肝。双肾集合系统分离，不排除膀胱充盈所致；膀胱壁毛糙，增厚。垂体增强MRI(图2C和2D)示垂体柄弥漫性增粗，呈结节样膨大，等T_1稍长T_2信号灶改变，大小约10 mm×12 mm×12 mm，增强扫描明显强化，提示垂体萎缩、后叶短T_1信号消失，伴垂体柄异常增粗、膨大，性质待定。头部增强MRI(图2E和2F)提示：大脑镰、右侧额部硬脑膜瘤样结节伴明显强化，Erdheim Chester病(Erdheim Chester disease，ECD)脑膜侵犯？脑膜瘤？

为明确垂体柄病变的病因，进一步完善相关检查，结果显示，红细胞沉降率105 mm/h(↑)，免疫球蛋白G4(immunoglobulin G4，IgG4)3.46 g/L(0.03~2.01 g/L)，余免疫全套、结缔组织全套、血甲胎蛋白、人绒毛膜促性腺激素均正常。全身骨显像示：颅骨、双膝关节、双股骨下段、双胫腓骨、双踝关节骨质代谢异常，性质待定。全身正电子发射计算机断层显像(positron emission tomography-computed tomography，PET/CT)提示：1)垂体柄增粗呈结节样改变，糖代谢明显增高(图2G)；2)下颌骨右侧支溶骨性破坏，双侧上颌窦壁、蝶窦壁及蝶骨翼、双侧股骨远端、胫骨近端及远端对称性成骨性骨质破坏，糖代谢增高(图2H)；3)双肺少许间质性炎症；4)双侧肾盂、输尿管及膀胱壁稍不均匀增厚，双肾轻度积

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水。以上多系统异常改变，结合临床考虑非朗格汉斯细胞组织细胞增生性疾病(Langerhans cell histiocytosis，LCH)可能性大。完善骨髓涂片，结果显示，骨髓增生活跃，各阶段细胞形态正常。双能X线骨密度检查提示骨量减少。

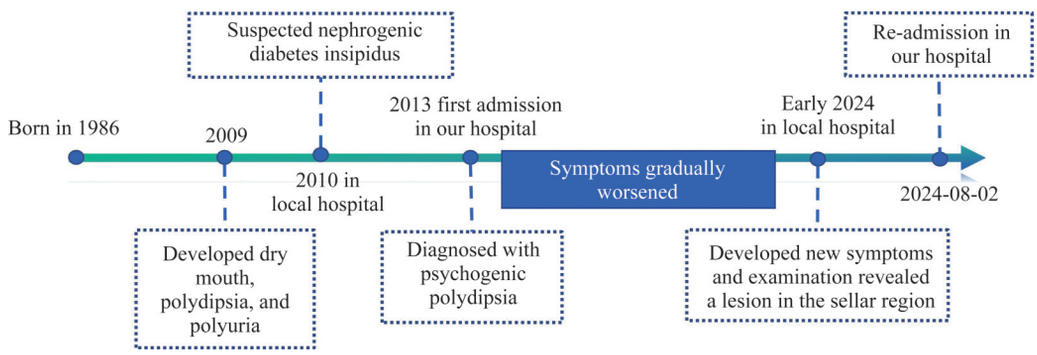


图1　患者疾病自然史

Figure 1　Natural history of the patient's disease

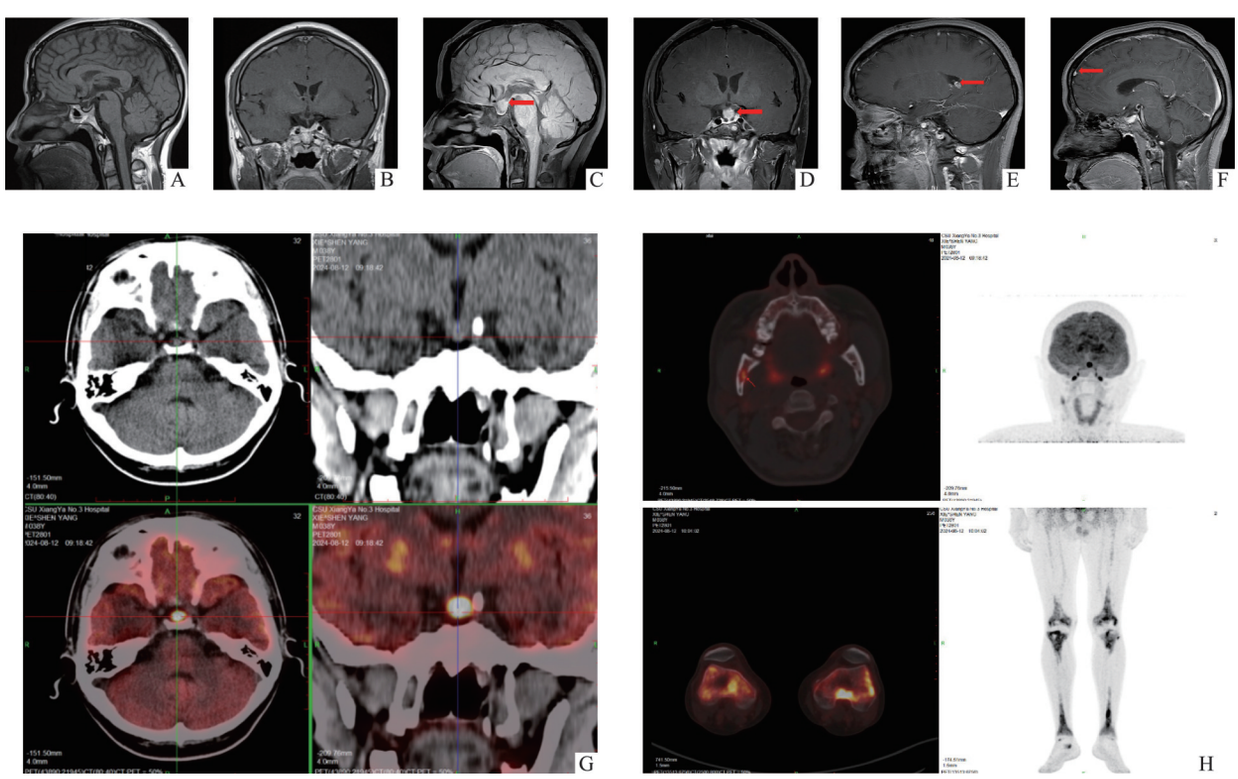


图2　患者典型的影像资料

Figure 2　Typical imaging findings of the patient

A and B: Sagittal and coronal images of contrast-enhanced pituitary MRI in 2013, showing normal pituitary size and morphology, midline pituitary stalk, but absence of the normal high signal of the posterior pituitary on T_1-weighted imaging. C and D: Sagittal and coronal images of contrast-enhanced pituitary MRI in 2024, showing diffuse thickening of the pituitary stalk with nodular enlargement, measuring approximately 10 mm×12 mm×12 mm (red arrow), marked enhancement on contrast, pituitary atrophy, and loss of the posterior pituitary high signal on T_1-weighted imaging. E: Contrast-enhanced head MRI shows nodular thickening of the falx cerebri above the splenium of the corpus callosum with obvious homogeneous enhancement (red arrow). F: Contrast-enhanced head MRI shows a subdural nodule in the right frontal region with marked enhancement (red arrow). G: PET/CT shows marked thickening of the pituitary stalk with increased glucose metabolism uptake. H: PET/CT shows osteolytic destruction of the right ramus of the mandible, symmetric bone destruction of the bilateral distal femurs, proximal and distal tibiae, with increased glucose metabolism. MRI: Magnetic resonance imaging; PET/CT: Positron emission tomography-computed tomography.

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为进一步明确病因，行垂体柄活体组织检查，病理回报(图3)：镜下见神经组织，局灶较多组织细胞和淋巴细胞浸润，且可见典型的“伸入现象”，免疫学表型较符合RDD的特征。免疫学表型：分化簇(cluster of differentiation，CD)163(+)，SRY相关HMG盒转录因子10(SRY-related HMG-box 10，SOX-10)(−)，甲状腺转录因子1(thyroid transcription factor 1，TTF-1)(灶+)，突触素(synaptophysin，Syn)(灶+)，嗜铬粒蛋白(chromogranin A，CgA)(−)，细胞角蛋白(cytokeratin，CK)(−)，CD207(−)，CD1a(−)，S100(灶+)，增殖指数Ki-67(15%+)，P53(40%+，弱阳性，提示可能为野生型)，CD68(+)，胶质纤维酸性蛋白(glial fibrillary acidic protein，GFAP)(+)，免疫球蛋白G(immunoglobulin G，IgG)(−)，IgG4(−)，CD138(−)。基因检测：*BRAF*^{V600E}基因突变型。结合患者临床表现及病理结果最终诊断为RDD，转至血液内科。用药方案为达拉非尼(150 mg，每日2次)、氢化可的松片(20 mg，每日1次)、左甲状腺素钠片(12.5 μg，每日1次)、去氨加压素片(早上0.05 mg，晚上0.1 mg)，辅以补钙、护胃等治疗。

规律治疗8个月后复诊，患者无明显口干，口服去氨加压素0.05 mg隔日1次，尿量2 000~3 000 mL/d，夜尿1次，乏力症状好转，复查垂体增强MRI(图4)可见垂体柄弥漫性增粗较前改善，由原来矢状位10 mm×12 mm×12 mm缩小至6 mm×7 mm×6 mm，且强化程度较前减低。此外，原有的大脑镰、右侧额部硬脑膜瘤样结节伴明显强化也未见显示。患者规律行达拉非尼靶向治疗1年后，转为使用人干扰素α-2b 600万U隔日1次皮下注射，动态复查头部增强MRI均提示垂体柄增粗、膨大较前次复查无明显变化，提示疾病未进展。以上病历资料的使用已获得患者的知情同意。

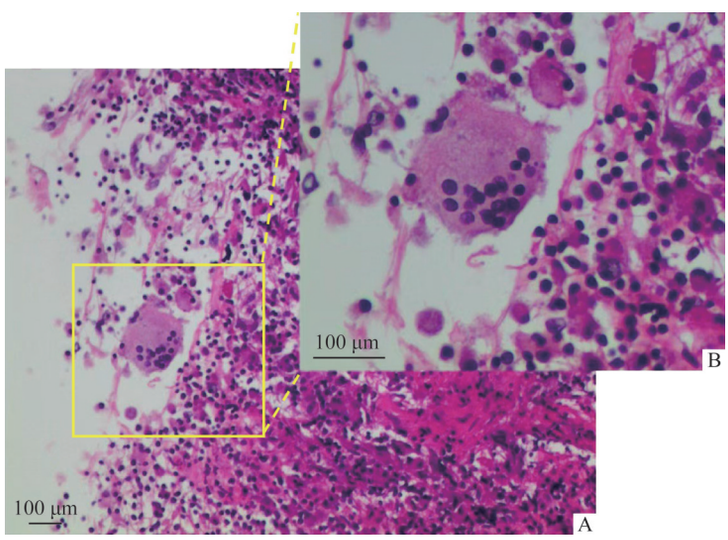


图3 垂体柄活检组织的病理结果典型图(HE染色)

Figure 3 Typical pathological findings of the pituitary stalk biopsy tissue (HE staining)

A: Focal infiltration of numerous histiocytes and lymphocytes. B: Magnified view in the upper right corner corresponds to Figure A, showing the typical “emperipolesis” phenomenon. HE: Hematoxylin and eosin.

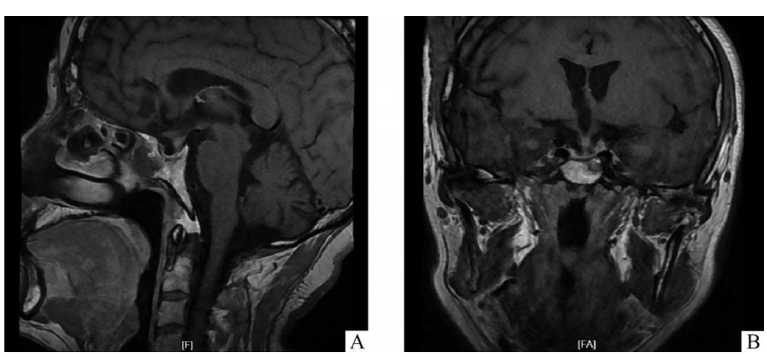


图4 靶向治疗8个月后复查垂体增强MRI

Figure 4 Follow-up enhanced MRI scan of the pituitary after 8 months of targeted therapy

A and B: Sagittal (A) and coronal (B) images from the follow-up contrast-enhanced pituitary MRI, showing reduced diffuse thickening of the pituitary stalk, decreased nodular enlargement (measuring approximately 6 mm× 7 mm× 6 mm), and diminished enhancement compared with the previous examination.

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2 讨　论

垂体柄增粗多以尿崩症为首发症状，发病时或进展过程中出现单一或多种垂体前叶功能减退症的表现，严重者甚至可出现功能障碍、下丘脑综合征等症状。垂体柄增粗的病因包括肿瘤性疾病、自身免疫性疾病、先天性疾病及感染性疾病，其中肿瘤性病变是其中一个重要的病因。2项针对垂体柄增粗患者的队列研究[1-2]显示，儿童青少年患者以生殖细胞瘤最为常见，成人患者以其他部位恶性肿瘤转移、LCH最为常见。

本例患者以尿崩症起病，病情迁延10余年，逐渐出现垂体柄增粗、硬脑膜和骨骼多系统受累，垂体柄活体组织检查提示RDD。分析该患者延迟诊断的原因可能是患病早期垂体后叶功能并未完全缺失，鞍区病变早期影像学改变不明显，以及出院后患者多年未遵医嘱复诊。以上提示对于尿崩症起病而垂体影像学改变不明显的患者需要定期观察随访。Di Iorgi等[3]总结分析了43例青少年特发性中枢性尿崩症患者，其中40例在半年内出现垂体柄影像学增粗变化；3例在中位随访9年(8.5~10年)后诊断为LCH，1例随访13年后诊断为霍奇金淋巴瘤，上述病例在发病或随访过程中均出现不同程度的腺垂体功能减退及侵犯骨骼、肺或淋巴结的表现。而在另一项研究中，30例儿童中枢性尿崩症患者中有11例在随访1~126个月确诊为组织细胞增生症，1例在57个月后被诊断为生殖细胞瘤[4]。因此推荐在垂体柄增粗病因诊断不明确时，建议每3~6个月[5]或每6个月[3, 6]进行MRI检查，以便及时发现垂体柄变化并进一步行病因诊断。

国内虽有数例鞍上区、垂体RDD的报道[7-8]，但垂体柄的RDD侵犯尚无文献报道。RDD是一种罕见的非LCH，根据临床表现可分为散发性、家族性和皮肤性3类。散发性又分为经典淋巴结型、结外型、肿瘤相关型、自身免疫性疾病相关型[9]。最常见的是经典淋巴结RDD，表现为颈部无痛性淋巴结肿大，伴或不伴间歇性发热、盗汗和体重减轻，病变还可累及纵隔、腋窝和腹股沟淋巴结，腹膜后淋巴结受累少见[10]。大约43%的病例表现为结外RDD，常见的受累部位包括皮肤(10%)、鼻腔(11%)、骨(5%~10%)、眼眶组织(11%)和中枢神经系统(5%)[11]。RDD起病或急或隐匿，临床表现具有高度异质性，从无症状到播散性乃至危及生命。RDD诊断主要依赖于病理学特征及免疫组织化学。RDD在镜下的特征性表现为“伸入现象”，即在体积较大、胞质丰富且核仁突出的组织细胞中，可见吞噬完整的淋巴细胞和浆细胞，背景可见大量淋巴细胞和浆细胞浸润[12]。RDD免疫组织化学表型为S-100、CD68、CD163阳性和CD1α阴性。本病例有典型的CD1α阴性，可与LCH相鉴别；ECD也是一种非LCH，常见垂体柄浸润合并成骨性骨破坏，但本病例的病理S-100阳性、典型“伸入现象”且未见典型的组织泡沫样细胞和Touton巨细胞，可与ECD相鉴别；亦有文献[13-14]报道ECD合并局灶RDD病变，但多发生于老年男性且ECD先于RDD病灶被诊断，多侵犯睾丸组织和骨组织。RDD可模拟IgG4相关疾病，在病理局灶中IgG4阳性浆细胞可达30个/高倍镜视野以上[15]，本病例血液中IgG4水平升高，但病理标本中无阳性IgG4浆细胞，不支持IgG4相关疾病。而结核、淋巴瘤等因无相关临床表现故不考虑。

有丝分裂原活化蛋白激酶(mitogen-activated protein kinase，MAPK)信号通路在组织细胞增生性疾病中的突变率高达80%，其中以*BRAF*^{V600E}最为常见，为50%~60%，其他突变还包括*KRAS/NRAS*、*MAP2K1*(或者*MEK*)、*ARAF*基因突变[16-17]。MAPK信号通路中MEK抑制剂考比替尼(cobimetinib)和BRAF抑制剂维莫非尼(vemurafenib)现已被批准用于治疗组织细胞疾病，临床证据主要集中于LCH和ECD[18-20]。以往报道的RDD病例中无*BRAF*突变[21]，但近年来有数例RDD合并*BRAF*突变的报道[12]，且在应用*BRAF*抑制剂和/或*MEK*抑制剂后进展的病情得到部分缓解[22-23]。本例患者多系统受累合并*BRAF*^{V600E}突变，经与血液科医师会诊，予以达拉非尼作为一线治疗方案，在靶向治疗1年后转为干扰素治疗。上述治疗显著改善了患者的症状、头部影像学表现及生活质量，远期预后还有待进一步随访。

综上，本文报道了1例累及垂体柄、硬脑膜、骨骼等多部位的RDD伴有罕见的*BRAF*^{V600E}突变，病情呈持续缓慢进展，无自发缓解趋势，需要进行积极干预。这一病例有助于临床医师对垂体柄增粗进行病因诊断及鉴别诊断，并认识RDD这一罕见疾病。

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