

## 胸部CT显示梗阻性肥厚型心肌病引起 肺部树芽征1例

张龙富<sup>1,2</sup> 沈勤军<sup>2</sup> 叶伶<sup>2</sup> 朱蕾<sup>2△</sup>

(<sup>1</sup>上海市徐汇区中心医院呼吸科 上海 200031; <sup>2</sup>复旦大学附属中山医院呼吸科 上海 200032)

**【摘要】** 患者咳嗽、胸闷、气促2月余,胸部CT表现为树芽征(tree-in-bud pattern, TIB)和磨玻璃斑片影,考虑感染引起的小气道病变可能,予以抗感染及对症治疗,患者症状无好转。入院后查氨基末端利钠肽前体(amino-terminal pro-brain natriuretic peptide, NT-proBNP)升高,结合肺部磨玻璃影,考虑心源性肺水肿可能,进一步完善心超,明确为梗阻性肥厚型心肌病,左室流出道压差明显升高;经针对性改善心功能治疗,患者症状好转,肺部病灶吸收。本文重点介绍TIB形成的病因、机制及鉴别诊断思路,提高临床医师对TIB的认识。

**【关键词】** 树芽征(TIB); 梗阻性肥厚型心肌病; 慢性心功能不全; 微血管

**【中图分类号】** R563 **【文献标志码】** B **doi:** 10.3969/j.issn.1672-8467.2021.04.024

## Tree-in-bud pattern on chest CT due to obstructive hypertrophic cardiomyopathy: a case report

ZHANG Long-fu<sup>1,2</sup>, SHEN Qin-jun<sup>2</sup>, YE Ling<sup>2</sup>, ZHU Lei<sup>2△</sup>

(<sup>1</sup>Department of Pulmonary Medicine, Central Hospital of Xuhui District, Shanghai 200031, China;

<sup>2</sup>Department of Pulmonary Medicine, Zhongshan Hospital, Fudan University, Shanghai 200032, China)

**【Abstract】** The patient had cough, chest tightness and shortness of breath for more than 2 months, and chest CT showed tree-in-bud pattern (TIB) and ground glass patchy shadows. Considering the possibility of small airway disorders caused by infection, anti-infection and symptomatic treatment were applied, but the patient's symptoms did not improve. After admission, amino-terminal pro-brain natriuretic peptide (NT-proBNP) was detected to be elevated. Combined with the pulmonary ground glass patchy shadows and the possibility of cardiogenic pulmonary edema, further improved cardiac ultrasound confirmed the diagnosis of obstructive hypertrophic cardiomyopathy. The pressure difference of left ventricular outflow tract was significantly increased. After treatment for improving cardiac function, the symptoms improved and the lung lesions were absorbed. This paper mainly introduces the etiology, formation mechanism and differential diagnosis of TIB, so as to improve the understanding of clinicians on TIB.

**【Key words】** tree-in-bud pattern (TIB); obstructive hypertrophic cardiomyopathy; chronic heart failure; microvessel

树芽征(tree-in-bud pattern, TIB)首次由Im等<sup>[1]</sup>报道,用来描述肺结核沿细支气管扩散,后来发现感染、结缔组织疾病、肿瘤等疾病累及细支气管

时均可引起TIB<sup>[2-3]</sup>,有少数病例报道肺小血管病变亦可引起肺部TIB。2001年Tack等<sup>[4]</sup>在腹部小圆细胞肿瘤的胸部CT发现TIB,进一步活检提示小

<sup>△</sup>Corresponding author E-mail: zhu.lei@zs-hospital.sh.cn

网络首发时间:2021-07-20 17:08:35 网络首发地址: <https://kns.cnki.net/kcms/detail/31.1885.R.20210719.1458.002.html>

叶中央动脉被肿瘤细胞堵塞并管腔扩张。现报道1例由梗阻性肥厚型心肌病伴心功能不全引起肺部微血管扩张导致TIB。

**病例资料** 患者,男,52岁,就职于公安系统。因“咳嗽伴胸闷气促2月余。”于2019年10月底收住复旦大学附属中山医院。患者2月余前出现阵发性咳嗽,干咳为主,伴活动后胸闷、气促,无发热及盗汗,无胸痛,无下肢浮肿。于当地社区医院查血常规:白细胞计数  $12.18 \times 10^9/L$ ,中性粒细胞计数  $9.36 \times 10^9/L$ ,血红蛋白 160 g/L;胸部CT报告细支气管炎;予头孢类抗感染后,咳嗽、胸闷、气促均未好转。遂于3周前至当地上级医院就诊,查血气分析:pH=7.45,  $PaCO_2$  41 mmHg,  $PaO_2$  62 mmHg,  $HCO_3^-$  28.5 mmol/L, BE 4.1 mmol/L,  $SaO_2$  92%;血常规:白细胞计数  $8.74 \times 10^9/L$ ,中性粒细胞计数  $5.81 \times 10^9/L$ ,血红蛋白 145 g/L, C反应蛋白(C-reactive protein, CRP) 16.6 mg/L,降钙素原(procalcitonin, PCT)  $<0.02$  ng/mL,呼吸道病原体九联检、痰细菌培养和1,3-痰细菌培养葡聚糖(G试验)均阴性,氨基末端利钠肽前体(amino-terminal pro-brain natriuretic peptide, NT-proBNP) 437 pg/mL,胸部CT示双肺弥漫性TIB及散在磨玻璃斑片影(图1A、1B)。给予头孢唑肟、莫西沙星抗感染及对症治疗2周,症状无好转,遂收入我院。入院时查体: $SpO_2$  96%(吸空气),BP 114/60 mmHg,神清,静息坐位或平卧位时呼吸平稳,颈静脉无怒张,双下肺闻及少许湿啰音,HR 108次/min,律齐,心尖部可闻及收缩中晚期喷射性杂音,双下肢无浮肿。

入复旦大学附属中山医院后查血常规:白细胞计数  $6.53 \times 10^9/L$ ,中性粒细胞计数  $4.3 \times 10^9/L$ ,血红蛋白 143 g/L,CRP 7.8 mg/L, PCT 0.12 ng/mL, NT-proBNP 2 922 pg/mL,结核T细胞斑点试验(T cell spot test, T-SPOT)阴性。自身抗体:抗核抗体、抗dsDNA抗体阴性,抗ANCA抗体阴性。血清补体、抗“o”抗体、类风湿因子、肝肾功能、肿瘤指标均正常。血气分析:pH=7.44,  $PaCO_2$  48 mmHg,  $PaO_2$  78 mmHg,  $HCO_3^-$  32.6 mmol/L, BE 7.3 mmol/L,  $SaO_2$  96%。胸部CT提示双肺TIB及磨玻璃斑片影,且与前相似(图1C、1D),肺功能提示通气功能、弥散功能基本正常。心超提示梗阻性肥厚型心肌病,左室流出道压差 180 mmHg,中度二尖瓣反流, LVEF 68%(图2)。心电图:窦性心动过速, T波改

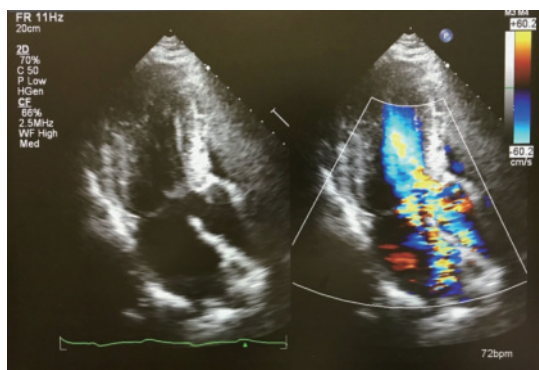
变。因患者心功能不佳,未行支气管镜检查。临床诊断:梗阻性肥厚型心肌病,慢性心力衰竭。临床症状及胸部影像学改变是心肌病的表现。给予伊伐布雷定控制心室率,呋塞米利尿治疗2周后,患者咳嗽明显减轻,胸闷、气促明显改善;复查心超仍符合梗阻性肥厚型心肌病,中度二尖瓣反流,但左室流出道压差降至 80 mmHg, NT-proBNP 降至 1 369 pg/mL。胸部CT提示肺部病灶较前明显吸收(图1E~1F)。患者后在中山医院心外科行左室流出道疏通术及二尖瓣置换术,病理符合肥厚型心肌病改变(图3)。术后予以倍他乐克治疗,偶干咳,日常生活无不适。



A-B: On Oct 10, 2019, chest CT showed diffuse and symmetrical distribution of TIB pattern in both lungs, and ground glass patchy shadows were seen in the apical segment of both upper lungs and the dorsal segment of lower lungs; C-D: On Oct 30, 2019, chest CT showed no absorption of TIB pattern and ground glass patchy shadows; E-F: On Nov 12, 2019, after improved cardiac function, chest CT showed significant absorption of TIB pattern and ground glass patchy shadows.

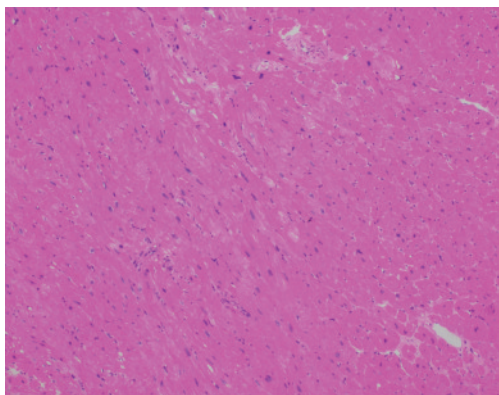
**图1 梗阻性肥厚型心肌病伴心功能不全患者的胸部CT变化**  
**Fig 1 Serial radiographic images of the patient with obstructive hypertrophic cardiomyopathy and cardiac insufficiency**

**讨论** 患者为中年男性,既往体健。本次急性起病,慢性病程,主要表现为干咳,活动后胸闷、气促;无低热、盗汗,无纳差、乏力,无痰血,无胸痛。查体:双下肺少许湿啰音,心尖部可及收缩中晚期



Echocardiography showed thickening of ventricular septum, anterior wall, lateral wall and apex. Pressure difference of left ventricular outflow tract was 180 mmHg. Echocardiography also showed moderate mitral regurgitation.

图2 梗阻性肥厚型心肌病患者的心脏彩色多普勒超声  
Fig 2 Echocardiography of the patient with obstructive hypertrophic cardiomyopathy



Cardiac biopsy showed hypertrophy of some cardiomyocytes, focal fibrosis and endocardial thickening, which were consistent with the changes of hypertrophic cardiomyopathy.

图3 梗阻性肥厚型心肌病患者的心肌活检的  
HE染色结果(×100)

Fig 3 HE staining results of myocardial biopsy in patient with obstructive hypertrophic cardiomyopathy (×100)

喷射性杂音。辅助检查:部分炎症指标轻度升高,病原学检查阴性,NT-proBNP明显升高。胸部CT显示双肺弥漫分布TIB及散在斑片影;抗感染治疗后症状及影像学无好转。入院诊断首先考虑肺弥漫性病变,合并慢性心功能不全。因患者胸部CT见典型TIB,故从TIB入手进行鉴别诊断。

TIB是指CT表现为靠近胸膜下区小叶中心直径为3~5 mm的分支状线影和与其相连的细支气管横断面结节影,状如春天挂满树芽的树。TIB中“树”是因阻塞而扩张的细支气管,“芽”是呼吸性细支气管和肺泡管内充的黏液等物质。该征象由Im

等<sup>[1]</sup>首次报道,主要反映小气道病变,最初用来描述肺结核沿细支气管扩散。后来发现多种疾病累及细支气管均可引起TIB,最常见的是感染(细菌、霉菌、病毒),其他病因包括先天性疾病(囊性纤维化、Kartagener综合征),特发性病变(闭塞性细支气管炎、弥漫性泛细支气管炎),异物吸入,变态反应性支气管肺曲霉病(allergic bronchopulmonary aspergillosis, ABPA)、结缔组织病变(类风湿关节炎、干燥综合征)、过敏性肺炎、肿瘤气道播散<sup>[2-3]</sup>。少数病例报道恶性肿瘤累及肺血管也可以形成TIB改变,其形成机制是肺外恶性肿瘤栓子堵塞肺小叶中央动脉,或肿瘤细胞导致肺小动脉内膜的纤维细胞增生、管腔狭窄,导致远端肺动脉扩张。扩张的肺小动脉类似于“树”,被肿瘤细胞或内膜纤维细胞增生堵塞的血管末端类似于“芽”<sup>[4-6]</sup>。

TIB鉴别可从感染和非感染,细支气管和肺微血管等方面进行。本例患者起病初炎症指标偏高、有咳嗽气促症状,因此TIB需要鉴别感染方面因素。引起TIB的病原体包括结核分枝杆菌、非结核分枝杆菌(nontuberculous mycobacteria, NTM)、支原体、衣原体、曲霉菌、巨细胞病毒(cytomegalovirus, CMV)、金葡菌、流感嗜血杆菌等。本例患者既往体健,社区起病,病程长,头孢唑肟联合莫西沙星抗感染治疗无效;呼吸道病原体九联检及CMV抗体阴性,无法用常见细菌、支原体、衣原体及CMV感染解释。患者无结核毒血症症状,T-SPOT阴性,结核感染也可基本排除。患者TIB弥漫分布,非中叶为主,且无支气管扩张,与NTM感染特点不符。患者免疫功能正常,G试验阴性,曲霉菌感染也可基本排除。非感染因素方面,因患者中年起病,否认慢性咳嗽病史,本次入院自身抗体阴性,嗜酸性粒细胞及IgE正常,故不考虑先天性疾病、结缔组织疾病和ABPA所致TIB。患者否认吸烟史、无粉尘及刺激性气体吸入;不考虑吸入性疾病。闭塞性细支气管炎和泛细支气管炎多有慢性呼吸道症状,影像学可见下叶外带为主TIB及支气管扩张、管壁增厚;与本患者不符。患者急性起病,肺部无占位性病变,肺门及纵膈淋巴结无肿大,肿瘤指标正常,故也不考虑肿瘤气道播散引起TIB。

从上述分析可知,如无明确依据提示累及细支气管的疾病引起TIB,需考虑肺血管病变引起的TIB样改变。本患者肺通气功能、弥散功能基本正

常,PaO<sub>2</sub>和SaO<sub>2</sub>轻度下降,符合轻度肺微血管病变。结合患者梗阻性肥厚型心肌病,左室流出道压差明显增大;NT-proBNP升高,符合心肌病导致轻度心功能不全,肺血管轻度淤血,肺微血管扩张,推测由于心衰偏轻,以肺微静脉和毛细血管扩张为主,引起TIB;同时伴轻度肺间质水肿,出现磨玻璃片状影。患者急性发病,但呈亚急性病程,肺底部血管反射性收缩,间质水肿减轻,故上肺表现更明显。经控制心室率、利尿等改善心功能治疗后,短时间内咳嗽、胸闷、气促症状好转,影像学明显改善,进一步支持轻度肺微血管扩张引起TIB。但是由于患者心功能不稳定,未行组织活检病理协助诊断。

通过本例病例资料及文献复习可熟悉TIB的病因、形成机制及鉴别诊断。尽管细支气管疾病是引起TIB的主要病因,但也不能忽视肺微血管疾病,特别是不典型或轻度心功能不全引起肺血管淤血。

**作者贡献声明** 张龙富 资料收集,论文撰写和修改。沈勤军,叶伶 论文修改。朱蕾 病情分析,论文修改和定稿。

**利益冲突声明** 所有作者均声明不存在利益冲突。

## 参 考 文 献

- [ 1 ] IM JG, ITOH H, SHIM YS, *et al.* Pulmonary tuberculosis: CT findings-early active disease and sequential change with antituberculous therapy[J]. *Radiology*, 1993, 186(3): 653-660.
  - [ 2 ] COLLINS J, BLANKENBAKER D, STERN EJ. CT patterns of bronchiolar disease: what is "tree-in-bud"? [J]. *AJR Am J Roentgenol*, 1998, 171(2): 365-370.
  - [ 3 ] VERMA N, CHUNG JH, MOHAMMED TH. "Tree-in-bud sign"[J]. *J Thorac Imaging*, 2012, 27(2): W27.
  - [ 4 ] TACK D, NOLLEVAUX MC, GEVENOIS PA. Tree-in-bud pattern in neoplastic pulmonary emboli[J]. *AJR Am J Roentgenol*, 2001, 176(6): 1421-1422.
  - [ 5 ] FRANQUET T, GIMENEZ A, PRATS R, *et al.* Thrombotic microangiopathy of pulmonary tumors: a vascular cause of tree-in-bud pattern on CT[J]. *AJR Am J Roentgenol*, 2002, 179(4): 897-899.
  - [ 6 ] NG YL, HWANG D, PATSIOS D, *et al.* Tree-in-bud pattern on thoracic CT due to pulmonary intravascular metastases from pancreatic adenocarcinoma[J]. *J Thorac Imaging*, 2009, 24(2): 150-151.
- (收稿日期:2020-11-23; 编辑:段佳)
- 
- (上接第564页)
- [ 5 ] EDWARDS TM, DURAN MS, MEEKER TM. Congenital infantile fibrosarcoma in the premature infant [J]. *Adv Neonatal Care*, 2017, 17(6): 440-450.
  - [ 6 ] STEELMAN C, KATZENSTEIN H, PARHAM D, *et al.* Unusual presentation of congenital infantile fibrosarcoma in seven infants with molecular-genetic analysis [J]. *Fetal Pediatr Pathol*, 2011, 30(5): 329-337.
  - [ 7 ] HECHTMAN JF, BENAYED R, HYMAN DM, *et al.* Pan-Trk immunohistochemistry is an efficient and reliable screen for the detection of NTRK fusions[J]. *Am J Surg Pathol*, 2017, 41(11): 1547-1551.
  - [ 8 ] KANACK M, COLLINS J, FAIRBANKS TJ, *et al.* Congenital-infantile fibrosarcoma presenting as a hemangioma: a case report [J]. *Ann Plast Surg*, 2015, 74 (Suppl 1): S25-S29.
  - [ 9 ] DURIN L, JEANNE-PASQUIER C, BAILLEUL P, *et al.* Prenatal diagnosis of a fibrosarcoma of the thigh: a case report[J]. *Fetal Diagn Ther*, 2006, 21(6): 481-484.
  - [ 10 ] CANALE S, VANEL D, COUANET D, *et al.* Infantile fibrosarcoma: Magnetic resonance imaging findings in six cases[J]. *Eur J Radiol*, 2009, 72(1): 30-37.
  - [ 11 ] 王玉, 王晓曼, 贾立群, 等. 婴儿型纤维肉瘤与软组织血管瘤的超声鉴别诊断[J]. *中华超声影像学杂志*, 2019, 28(11): 994-998.
  - [ 12 ] 朱倩, 王莽, 钦斌. 婴儿型纤维肉瘤影像学表现[J]. *中国医学影像技术*, 2018, 34(1): 99-102.
- (收稿日期:2020-08-14; 编辑:沈玲)