伴发于急性 Stanford-A 型主动脉夹层的肺动脉鞘血肿的 CT 表现及预后

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摘　要：【目的】探讨肺动脉鞘血肿(HPS)在 Stanford-A 型主动脉夹层(AD)中的检出率及其 CT 表现，评估患者短期预后不良的危险因素。【方法】回顾性分析连续 188 例经 CT 诊断为急性 Stanford A 型 AD 的患者，分析 CT 图像并记录临床资料。【结果】18 例患者合并 HPS，检出率为 9.6%。累及右侧肺动脉 9 例，累及左侧肺动脉 2 例，双侧受累 7 例；9 例血肿(50%)进入肺内支气管血管束周围鞘(Ⅱ型)，其中 7 例伴周围片状实变/磨玻璃影。首诊 CT 后 30 d 内 11 例患者(61.1%)死亡，其中 7 例(63.6%)为Ⅱ型 HPS，未发现病变延伸与死亡率有关联(P=0.335)；腹部分支血管受累在死亡组中 7 例(63.6%)，存活组中 0 例，两组比较有统计学差异(P=0.01)。【结论】急性 Stanford-A 型 AD 合并 HPS 不少见，腹部分支血管受累提示预后不良。

关键词：肺动脉鞘血肿；主动脉夹层；计算机断层扫描；血管造影术
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Hemorrhagic Pulmonary Sheath Due to Ruptured Acute Stanford A Aortic Dissection: Incidence, CT Appearance and Consequences

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Abstract: 【Objective】To retrospectively investigate the incidence, computed tomography (CT) manifestations and consequences of hemorrhagic pulmonary sheath (HPS) in Stanford A aortic dissection (AD) patients. 【Methods】Institutional review board approval and informed consents were obtained. CT aortic angiography images of 188 consecutive acute Stanford A aortic dissection patients (mean age, 59 years; range: 29-78 years; 136 males, 52 females) were reviewed. CT images were interpreted by two independent radiologists. Clinical records were reviewed for outcomes of patients up to 30 days after the initial CT scan. 【Results】18 (9.6%) out of 188 patients had HPS. Right pulmonary artery was involved in 9 (50%), left pulmonary artery in 2 (11.1%) and both in 7 (38.9%) of the 18 patients respectively. HPS extending along bronchovascular sheaths (Type Ⅱ) was identified in 9 (50%) of 18 patients, and 7 (77.8%) of them had alveolar opacity around the thickened bronchovascular sheath. Within 30 days of follow-up, 61.1% (11/18) patients died and 38.9% (7/18) patients survived with absorption of HPS. Type Ⅱ HPS was more prevalent in death group (7/11, 63.6%) than survival group (2/7, 28.6%), but not statistically significant (P=0.335). Patients in death group were more likely to have abdominal visceral arteries involvement (7/11, 63.6%) than patients in survival group (0/7, 0%) (P=0.010). 【Conclusion】HPS was not a rare complication in patients with Stanford A AD. Abdominal visceral arteries involvement indicated poor short-term outcome in this study.

Key words: hemorrhagic pulmonary sheath; aortic dissection; computed tomography angiography
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急性主动脉夹层(aortic dissection,AD)并发肺动脉鞘血肿(hemorrhagic pulmonary sheath,HPS)是由于血流经破裂主动脉壁后进入肺动脉鞘所致,主要见于Stanford A型AD,既往多数为个例报道,且命名各异[1-2]。Sueyoshi等[3]回顾性分析了232例急性Stanford A型AD患者中HPS检出率、CT表现及患者预后,类似研究国内目前尚无报道。本文旨在对此进行初步分析。

1 材料与方法

1.1 病例资料

2011年1月-2016年1月间连续188例经CT诊断为急性Stanford A型AD的患者纳入本研究。患者平均年龄59岁(29~78岁),136例为男性。回顾性分析患者CT图像并记录临床资料。医院伦理委员会批准了该项研究,所有患者均签署知情同意书。

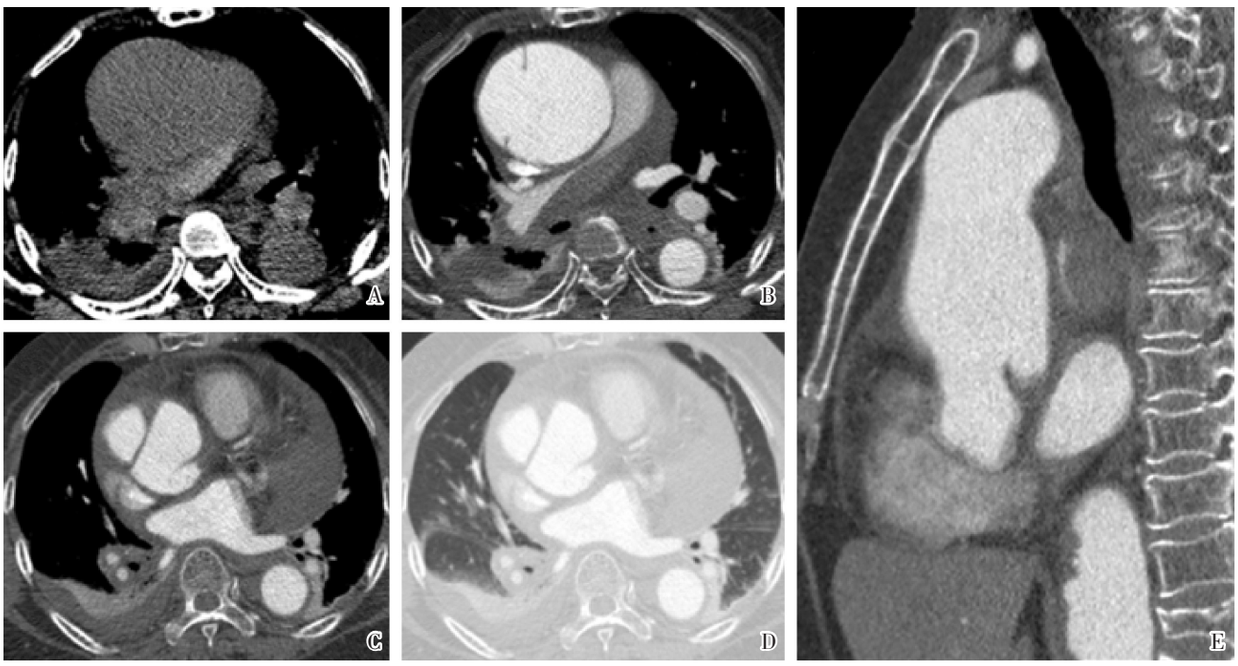
1.2 仪器和材料

采用多层螺旋CT(TOSHIBA)扫描,所有患者均为仰卧位。扫描为头足向,范围由胸廓上口至腹股沟水平。经肘静脉注入对比剂(碘普罗胺,300 mg/mL)80~100 mL,注射流率4.0 mL/s,对比剂注射后以流率4.0 mL/s追加40 mL生理盐水冲刷。扫描中使用对比剂自动追踪技术,感兴趣区置于腹主动脉上段,触发扫描的阈值设置为150 HU。

1.3 CT图像分析

参考文献[1-3]并结合我们的经验,诊断HPS的标准包括:①CT平扫主肺动脉或其分支血管腔周围环状或新月形高密度影包绕;②增强扫描环状影或新月形影相对血管腔呈低密度软组织影,无强化。HPS进一步分为两个类型,Ⅰ型:HPS仅位于主肺动脉和/或肺动脉干周围;Ⅱ型:HPS经肺门进入肺内且包绕支气管血管束,伴或不伴受累支气管血管束邻近肺组织片状实变或磨玻璃影(图1-2)。

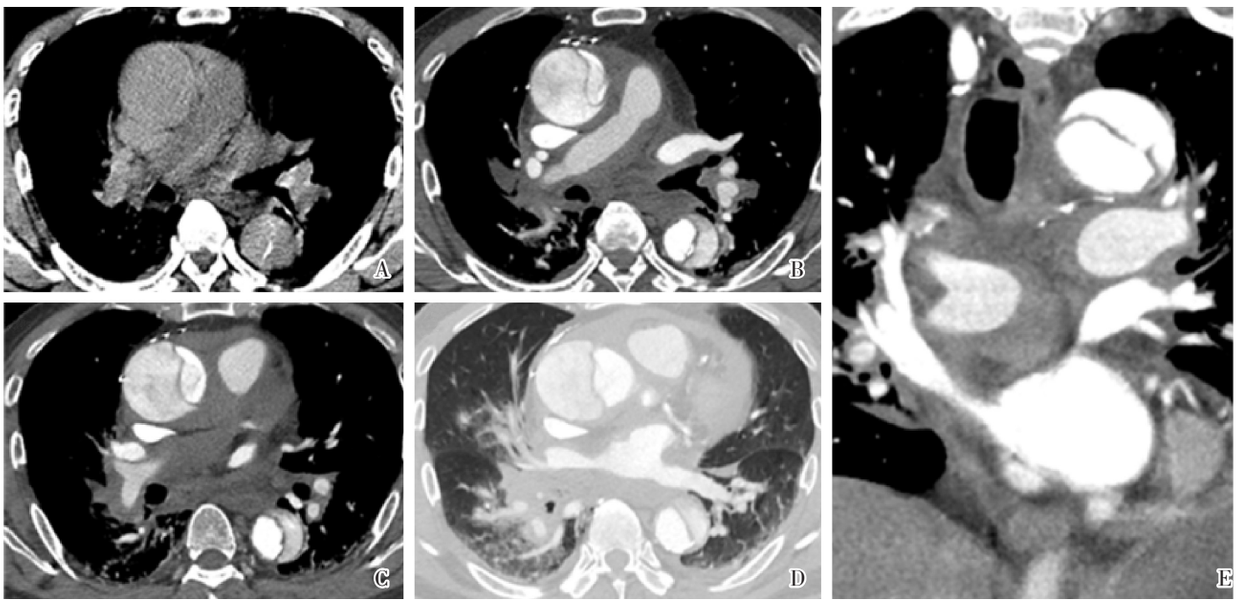
CTA评价的内容包括:1)AD的影像学特征(升主动脉径线测量,夹层累及范围,分支血管受累);2)HPS分类;3)受累主肺动脉和/或肺动脉主干狭窄程度(≥50%或<50%);4)心包、胸膜腔积



A: A high attenuation lesion was seen along the right pulmonary artery (PA) on non-enhanced CT image. B: The high attenuation area showed no contrast enhancement. Stenosis of the PA due to HPS was seen with reduction of diameter > 50%. C: The high attenuation lesion extended beyond the hilum surrounding the branches of PA and bronchi. D: The lung surrounding the involved branches of PA was clear. E: Sagittal sections showed the HPS along the right PA and its marked stenosis.

图1 急性主动脉夹层合并Ⅱ型肺动脉鞘血肿CT影像(73岁女性)

Fig.1 CT image of a 73-year-old female with aortic dissection with Type Ⅱ hemorrhagic pulmonary sheath



A：A high attenuation lesion was seen along the right PA on non-enhanced CT image. B：The high attenuation area showed no contrast enhancement. Stenosis of the PA due to HPS was seen with reduction of diameter < 50%. C：The high attenuation lesion extended beyond the hilum surrounding the branches of PA and bronchi. D：Ground-glass opacities surrounding the involved branches of PA. E：Coronal sections showed high attenuation lesion surrounding the bilateral PA and the branches of right PA.

图2　急性主动脉夹层合并Ⅱ型肺动脉鞘血肿CT影像（74岁男性）

Fig.2　CT image of a 74-year-old male with aortic dissection with Type Ⅱ hemorrhagic pulmonary sheath

液/积血。由两位心血管放射学专业的高年资医师对患者CTA图像进行分析，结果以取得一致意见为准。记录患者临床资料并随访患者在首诊CTA后30 d内自然病程（存活或死亡）。

1.4　统计学分析

统计学分析采用SPSS 19.0软件，采用Fisher确切概率法分别比较Ⅱ型HPS，腹部分支血管受累，头臂分支血管受累，心包/胸膜腔积液/血在存活组和死亡组之间的差异。采用Wilcoxon秩和检验比较升主动脉直径在存活组和死亡组之间的差异，设定检验水准α为0.05。

2　结　果

188例急性Stanford A型AD患者中18例患者合并HPS，检出率为9.6%。

18例患者中HPS仅累及右侧肺动脉9例，仅累及左侧肺动脉2例，双侧肺动脉干受累7例，共16例（88.9%）患者HPS累及右侧肺动脉干。有9例患者HPS进入肺内支气管血管束周围鞘（typeⅡ），表现为支气管血管束增粗，血管和支气管周围见环状软组织影包绕，其中7例患者受累支气管血管束周围肺组织见片状实变/磨玻璃影，结果如表1所示。

首诊CT后30 d内61.1%（11/18）患者死亡，38.9%（7/18）患者存活，存活者CT随访HPS吸收。

Ⅱ型HPS在死亡组中7例（63.6%），存活组中2例（28.6%），两组比较无统计学差异（P=0.335）；腹部分支血管受累在死亡组中7例（63.6%），存活组中0例，两组比较有统计学差异（P=0.01）；两组患者间升主动脉直径，头臂分支血管受累，心包/胸膜腔积液/血差异无统计学意义。

3　讨　论

急性Stanford A型AD并发HPS在既往文献中多为个例报道，且命名各异[4-6]。主动脉根部和中央肺动脉（主肺动脉、左/右肺动脉主干）在解剖上拥有共同的结缔组织鞘膜结构[7-8]。主动脉夹层时假腔侧受损主动脉壁因内压增高而破裂，血液经破裂主动脉壁进入肺动脉鞘所致。由于右侧肺动脉主干与主动脉壁紧邻，理论上HPS更容易累

表1 18例肺动脉鞘血肿患者的临床资料和CT表现

Table 1 Clinical data and CT manifestations of the 18 patients with hemorrhagic pulmonary sheath

No.	Age/Sex	AA D-max	Branches involved	Stenosis of PA	PA involved	HPS Type	Pericardial hemorrhage /effusion	Hemothorax /pleural effusion	Surgery	outcome
1	48/M	49 mm	None	<50%	Bilateral	Ⅰ	+	−	+	Alive
2	56/M	53 mm	none	<50%	Right	Ⅰ	+	+	+	Alive
3	74/F	68 mm	none	≥50%	Right	Ⅱ	+	+	−	Death
4	55/M	50 mm	visceral	<50%	Right	Ⅰ	+	−	+	Death
5	61/M	44 mm	visceral	<50%	Right	Ⅱ	+	+	+	Death
6	43/M	57 mm	visceral	<50%	Bilateral	Ⅱ	+	−	−	Death
7	70/F	51 mm	visceral	<50%	Right	Ⅰ	+	−	−	Death
8	62/M	47 mm	none	<50%	Bilateral	Ⅰ	+	−	+	Alive
9	70/F	51 mm	BCA	<50%	Right	Ⅰ	+	−	+	Alive
10	72/M	50 mm	BCA	<50%	Left	Ⅰ	+	+	−	Death
11	58/F	68 mm	none	≥50%	Bilateral	Ⅱ	+	+	+	Alive
12	53/M	47 mm	BCA	<50%	Bilateral	Ⅰ	+	−	−	Alive
13	57/M	59 mm	visceral	<50%	Left	Ⅱ	+	+	−	Death
14	34/M	49 mm	visceral	<50%	Right	Ⅰ	−	−	−	Death
15	60/M	53 mm	BCA	<50%	Bilateral	Ⅱ	+	+	−	Death
16	69/F	50 mm	BCA	<50%	Right	Ⅱ	+	+	−	Alive
17	74/M	52 mm	visceral	<50%	Bilateral	Ⅱ	+	+	+	Death
18	60/M	46 mm	BCA	<50%	Right	Ⅰ	+	−	−	Death

PA: pulmonary artery; AA: ascending aorta; D-max: diameter-max; BCA: brachiocephalic carotid artery

及右侧肺动脉鞘,在本组中,右侧肺动脉鞘受累者供16例(88.9%),与既往研究报道相符合[9]。Guilmette等[9]回顾性分析了经尸解和病理证实的3例AD合并HPS患者的病理解剖和组织学表现,证实了上述发生HPS的机制。对该征象的命名,笔者认为肺动脉鞘血肿更为恰当。

Sueyoshi等[3]回顾性分析了232例急性Stanford A型AD患者的CT表现,合并HPS者21例,检出率为9.1%。本研究中188例急性Stanford A型AD患者中18例患者合并HPS,检出率为9.6%。上述两项研究结果提示急性Stanford A型AD合并HPS并不少见,但研究对象都为亚洲人群,目前尚未见有关欧美人群的大宗报道,是否急性Stanford A型AD合并HPS在亚洲人群中较欧美人群更为常见,需要更多的研究结果进一步证实。

发生HPS时血液经破裂主动脉壁进入主动脉根部和中央肺动脉共同的鞘膜内形成血肿,血肿可经肺门沿支气管血管束周围间质蔓延,进入小叶间隔甚至累及邻近肺实质。本研究中笔者参考文献并结合我们的经验,提出了HPS的诊断和分类标准。

急性Stanford A型AD是临床危急重症,文献报道其入院前死亡率为49%,入院后30天内死亡率为47%[10]。既往研究认为合并HPS提示患者预后不良,但均为个例报道,缺乏大样本研究和统计分析[3-5]。Sueyoshi等[3]的研究结果显示,21例合并HPS的患者中8例(38.1%)死亡,经肺门沿支气管血管束周围间质蔓延,进入小叶间隔并累及邻近肺实质是与患者死亡相关的危险因素,作者认为血肿进入小叶间隔并累及邻近肺实质,导致了局部肺循环和肺换气障碍。本组18例患者中11例(61.1%)死亡,Ⅱ型HPS在死亡组中占63.6%,较存活组(28.6%)多,但两组比较无统计学差异(P=0.335),笔者认为造成差异的原因可能与样本量较小有关,是否HPS分型与患者预后不良相关,尚需要进一步研究。

本研究中腹部分支血管受累在死亡组中7例(63.6%),存活组中0例,两组比较有统计学差异(P=0.01)。Di Eusanio等[11]的研究也提示肠系膜上动脉和肾动脉受累所致器官灌注不良是主动脉

夹层患者住院死亡的独立危险因素。因此，腹部分支血管受累，可能导致器官灌注不良，是主动脉夹层患者预后不良的危险因素。

本组中38.9%（7/18）患者存活，存活者CT随访HPS吸收，提示CT随访显示HPS吸收可能提示患者短期预后较好，与文献报道一致[12-13]。

本文为回顾性研究且样本量较小是主要不足之处，研究结果显示死亡组和存活组间升主动脉直径，头臂分支血管受累的差异均无统计学意义，这与既往文献报道并不一致，考虑主要与样本量不足有关。本文仅对患者进行了短期随访，HPS是否与患者长期预后相关的还需要进一步研究。

参考文献

[1] Panicek DM, Ewing DK, Markarian B, et al. Interstitial pulmonary hemorrhage from mediastinal hematoma secondary to aortic rupture[J]. Radiology, 1987, 162(9):165-166.

[2] Cao DB, Yang SR, Tong Q, et al. Interstitial pulmonary hemorrhage along the pulmonary artery secondary to ruptured aortic dissection [J]. Intern Med, 2010, 49(8):1681-1682.

[3] Sueyoshi E, Matsuoka Y, Sakamoto I, et al. CT and clinical features of hemorrhage extending along the pulmonary artery due to ruptured aortic dissection[J]. Eur Radiol, 2009, 19(9):1166-1174.

[4] Makdisi G, Said SM, Schaff HV. Acute aortic dissection extending into the lung [J]. Ann Thorac Surg, 2015, 100(55):315-318.

[5] Reid A, Stride P, Hunter J, et al. Extensive bilateral extraluminal perivascular pulmonary haemorrhage associated with stanford type a aortic dissection[J]. Case Rep Med, 2011, 11(3):681643-681678.

[6] Muthu SK, Copley SJ. Case report: thickening of the peribronchovascular interstitium secondary to acute thoracic aortic dissection-- chest radiograph and CT appearances [J]. Clin Radiol, 2008, 63(12):236-238.

[7] Krizova A, Little D, Pollanen MS. Postmortem CT, gross and microscopic images of hemorrhage along the pulmonary artery sheath due to type A aortic dissection [J]. Forensic Sci Med Pathol, 2015, 11(6):455-459.

[8] Charnsangavej C. Occlusion of the right pulmonary artery by acute dissecting aortic aneurysm[J]. AJR Am J Roentgenol, 1979, 132(19):274-276.

[9] Guilmette J, Semionov A, Dennie C, et al. Hemorrhagic infiltration of the aortopulmonary adventitia: A complication of acute aortic dissection[J]. Eur J Radiol, 2016, 85(12):239-247.

[10] Bonaca MP, O'Gara PT. Diagnosis and management of acute aortic syndromes: dissection, intramural hematoma, and penetrating aortic ulcer [J]. Curr Cardiol Rep, 2014,16(10):536-566.

[11] Di Eusanio M, Trimarchi S, Patel HJ, et al. Clinical presentation, management, and short-term outcome of patients with type A acute dissection complicated by mesenteric malperfusion: observations from the International Registry of Acute Aortic Dissection [J]. J Thorac Cardiovasc Surg, 2013, 145(12):385-390.

[12] Shiau EL, Wu FZ, Huang YL, et al. Aortic intramural hematoma with pulmonary artery extension mimics pulmonary embolism[J]. Am J Emerg Med, 2013, 31(9):1533-1538.

[13] Valle FH, Gib M, Torres FS. Pulmonary artery intramural hematoma: unusual complication of aortic dissection [J]. Clin Biomed Res, 2015, 35(8):167-168.

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