methotrexate resistant human colon cancer cells[J]. Biochem Pharmacol, 2011, 82(11): 1572 - 1582.
[12] Wu H, Haag D, Muley T, *et al*. Tumor-microenvironment interactions studied by zonal transcriptional profiling of squamous cell lung carcinoma [J]. Genes Chromosomes Cancer, 2013, 52(3): 250 - 264.
[13] Yao G, Yin M, Lian J, *et al*. MicroRNA-224 is involved in transforming growth factor-beta-mediated mouse granulosa cell proliferation and granulosa cell function by targeting Smad4[J]. Mol Endocrinol, 2010, 24(3): 540 - 551.
[14] Pineau P, Volinia S, McJunkin K, *et al*. miR-221 overexpression contributes to liver tumorigenesis[J]. Proc Natl Acad Sci U S A, 2010, 107(1): 264 - 269.
[15] Myers C, Charboneau A, Cheung I, *et al*. Sustained expression of homeobox D10 inhibits angiogenesis[J]. Am J Pathol, 2002, 161(6): 2099 - 2109.
[16] Botas J. Control of morphogenesis and differentiation by HOM/Hox genes[J]. Curr Opin Cell Biol, 1993, 5(6): 1015 - 1022.
[17] Boudreau N, Bissell M J. Extracellular matrix signaling: integration of form and function in normal and malignant cells[J]. Curr Opin Cell Biol, 1998, 10(5): 640 - 646.
[18] Edelman G M, Jones F S. Outside and downstream of the homeobox [J]. J Biol Chem, 1993, 268(28): 20683 - 20686.
[19] Cho M A, Yashar P, Kim S K, *et al*. HoxD10 gene delivery using adenovirus/adeno-associate hybrid virus inhibits the proliferation and tumorigenicity of GH4 pituitary lactotrope tumor cells[J]. Biochem Biophys Res Commun, 2008, 371(3): 371 - 374.
[20] Sasayama T, Nishihara M, Kondoh T, *et al*. MicroRNA-10b is overexpressed in malignant glioma and associated with tumor invasive factors, uPAR and RhoC[J]. Int J Cancer, 2009, 125(6): 1407 - 1413.

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个案与短篇

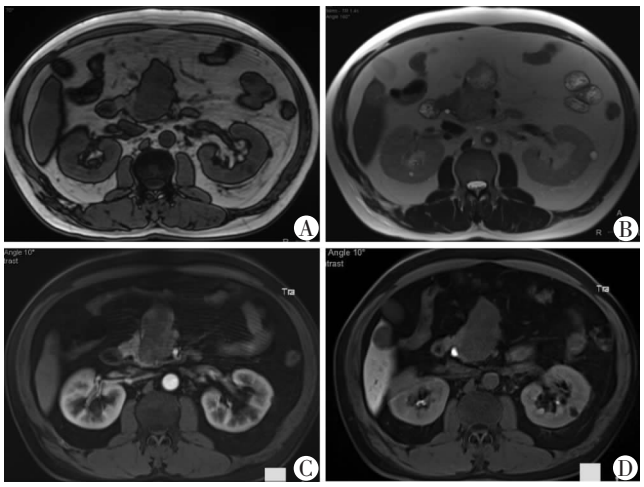
胰头低度恶性肌纤维母细胞肉瘤 1 例

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［**关键词**］　胰头;肌纤维母细胞肉瘤;MRI;CT;病理
［**中图法分类号**］　R816.5　　［**文献标志码**］　B

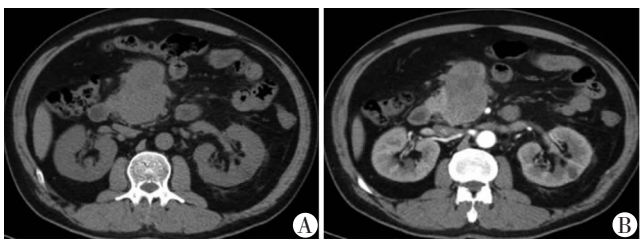
1　临床资料

患者,男性,50 岁,因“反复腹痛、腹胀 6 个月,加重伴消瘦 3 个月”于 2013 年 4 月 21 日入我院胃结直肠肛门外科就诊。体检:腹部平坦,未见异常搏动。剑突下扪及一包块,大小约 10 cm×9 cm×5 cm,边界不清,活动度欠佳,压痛,余腹无压痛,无反跳痛及肌紧张。叩诊呈鼓音,无移动性浊音,肠鸣音 4/min。4 月 26 日行上腹部 MRI 检查示:胰头部一类椭圆形软组织肿块,大小约 7.81 cm×4.58 cm,边缘不规则,边界尚清晰,其内呈混杂长 T_1 混杂长 T_2 信号,并可见小片状短 T_1 信号(图 1A、B),增强扫描病变不均匀强化(图 1C、D)。7 月 15 日 CT 定位检查示:平扫胰头部见一软组织肿块,大小约 8.6 cm×6.2 cm,密度欠均匀,其中心见较低密度区,CT 值 8~35 HU,肿块边缘不规则,分界尚清(图 2A);增强后肿块中度强化,其内低密度区未见明显强化(图 2B)。该患者在 CT 引导下行胰头包块穿刺活检,穿刺标本肉眼呈灰白灰褐色碎米样组织。病理观察示:镜下见肿瘤细胞呈梭形,核大,深染,细胞排列呈编织状、束状,有一定异型性,核分裂象为 2/10HPF。免疫组化:SMA(++)、Desmin(-)、Calponin(+)、S-100 单(-)、CD117(-)、CD21(-)、CD23(-)、CD56(-)、S-100 多(+)、Syn(+)、Ki-67(50%)、CK(-)、Dog-1(-)。病理诊断:胰头低度恶性肌纤维母细胞肉瘤(图 3)。



A:T_1WI 呈低信号;B:T_2WI 呈混杂高信号;C:MR 增强(动脉期)不均匀强化;D:MR 增强(延迟期)强化减退

图 1　胰头低度恶性肌纤维母细胞肉瘤患者上腹部 MRI 表现



A:平扫;B:增强扫描

图 2　胰头低度恶性肌纤维母细胞肉瘤患者腹部 CT 表现

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［**优先出版**］　http://www.cnki.net/kcms/detail/51.1095.R.20140618.1744.010.html(2014-06-18)

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病理重构具有重要的保护作用,可能为心血管疾病提供新的药物靶点。

参考文献:

[1] Mercure C, Yogi A, Callera G E, *et al*. Angiotensin(1-7) blunts hypertensive cardiac remodeling by a direct effect on the heart[J]. Circ Res, 2008, 103(11): 1319 – 1326.

[2] Giani J F, Munoz M C, Mayer M A, *et al*. Angiotensin-(1-7) improves cardiac remodeling and inhibits growth-promoting pathways in the heart of fructose-fed rats[J]. Am J Physiol Heart Circ Physiol, 2010, 298(3): H1003 – H1013.

[3] Ferreira A J, Castro C H, Guatimosim S, *et al*. Attenuation of isoproterenol-induced cardiac fibrosis in transgenic rats harboring an angiotensin-(1-7)-producing fusion protein in the heart[J]. Ther Adv Cardiovasc Dis, 2010, 4(2): 83 – 96.

[4] Pei Z, Meng R, Li G, *et al*. Angiotensin-(1-7) ameliorates myocardial remodeling and interstitial fibrosis in spontaneous hypertension: role of MMPs/TIMPs[J]. Toxicol Lett, 2010, 199(2): 173 – 181.

[5] Olivetti G, Capasso J M, Meggs L G, *et al*. Cellular basis of chronic ventricular remodeling after myocardial infarction in rats[J]. Circ Res, 1991, 68(3): 856 – 869.

[6] Fraccarollo D, Hu K, Galuppo P, *et al*. Chronic endothelin receptor blockade attenuates progressive ventricular dilation and improves cardiac function in rats with myocardial infarction: possible involvement of myocardial endothelin system in ventricular remodeling[J]. Circulation, 1997, 96(11): 3963 – 3973.

[7] Wilson E M, Moainie S L, Baskin J M, *et al*. Region- and type-specific induction of matrix metalloproteinases in post-myocardial infarction remodeling[J]. Circulation, 2003, 107(22): 2857 – 2863.

[8] Peterson J T, Li H, Dillon L, *et al*. Evolution of matrix metalloprotease and tissue inhibitor expression during heart failure progression in the infarcted rat[J]. Cardiovasc Res, 2000, 46(2): 307 – 315.

[9] Li Y Y, Feng Y Q, Kadokami T, *et al*. Myocardial extracellular matrix remodeling in transgenic mice overexpressing tumor necrosis factor alpha can be modulated by anti-tumor necrosis factor alpha therapy[J]. Proc Natl Acad Sci U S A, 2000, 97(23): 12746 – 12751.

[10] Cadete V J, Arcand S A, Chaharyn B M, *et al*. Matrix metalloproteinase-2 is activated during ischemia/reperfusion in a model of myocardial infarction[J]. Can J Cardiol, 2013, 29(11): 1495 – 1503.

[11] Zhong J, Basu R, Guo D, *et al*. Angiotensin-converting enzyme 2 suppresses pathological hypertrophy, myocardial fibrosis, and cardiac dysfunction[J]. Circulation, 2010, 122(7): 717 – 728.

[12] Grobe J L, Mecca A P, Mao H, *et al*. Chronic angiotensin-(1-7) prevents cardiac fibrosis in DOCA-salt model of hypertension[J]. Am J Physiol Heart Circ Physiol, 2006, 290(6): H2417 – H2423.

[13] Iwata M, Cowling R T, Gurantz D, *et al*. Angiotensin-(1-7) binds to specific receptors on cardiac fibroblasts to initiate antifibrotic and antitrophic effects[J]. Am J Physiol Heart Circ Physiol, 2005, 289(6): H2356 – H2363.

[14] 孟莹, 余常辉, 蔡绍曦, 等. 血管紧张素 1-7 对博来霉素诱导大鼠肺纤维化的抑制作用[J]. 中华医学杂志, 2013, 93(20): 1585 – 1589.

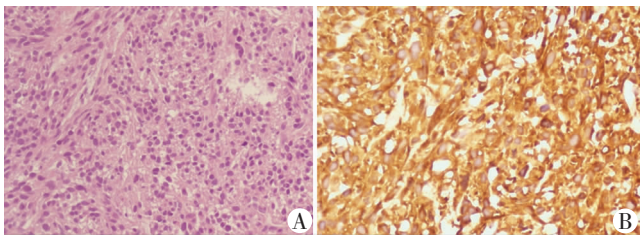
[15] Iusuf D, Henning R H, van-Gilst W H, *et al*. Angiotensin-(1-7): pharmacological properties and pharmacotherapeutic perspectives[J]. Eur J Pharmacol, 2008, 585(2/3): 303 – 312.

[16] Rosenzweig A. Vectors for cardiovascular gene therapy[J]. J Mol Cell Cardiol, 2003, 35(7): 731 – 733.

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A:光镜观察(HE ×200);B:免疫组化染色(SMA ×400)

图 3 患者胰头包块穿刺标本病理学观察

2 讨论

低度恶性肌纤维母细胞肉瘤(low-grade myofibroblastic sarcoma,LGMS)临床罕见,不同于以前报道的炎性肌纤维母细胞瘤,没有许多淋巴细胞及浆细胞,且核不呈组织细胞样特征[1]。LGMS 组成成分主要为分化程度不同的肌纤维母细胞,在 2002 年 WHO 软组织肿瘤分类中被列为独立的疾病类型[2]。

LGMS 发生部位以头颈部最常见,尤其是口腔及舌,其次为躯干、四肢等,腹腔最为少见[3]。本例患者发生于胰头确属罕见。由于其临床上、影像学上均无特异性表现,确诊主要依靠病理诊断。LGMS 在光镜下可见大量的梭形细胞呈束状、花纹状排列,并呈浸润性生长,瘤细胞常弥漫浸润于周围软组织,特别是横纹肌和脂肪,罕见核分裂数及炎症细胞,可与炎性肌纤维母细胞瘤鉴别。Coffin 等[4]报道 LGMS 免疫组织化学常表现为:Vimentin 强阳性,SMA 阳性,肌特异性肌动蛋白(MSA)阳性,Desmin 呈灶性阳性,CK、CD68(KP21)、CD30 偶见灶性阳性。本例标本病理学表现与文献报道基本相符。

目前 LGMS 的治疗主要依靠手术切除,术后少数患者出现局部复发,个别患者可出现远处转移[3]。本例患者行上腹部 CT 影像查见肝脏多发转移及门静脉瘤栓,考虑可能是病变发生于胰头部,肿瘤细胞随血循环经门静脉达到肝脏所致。LGMS 虽属于低度恶性肿瘤,但经本例患者病情进展可提示,不同部位的 LGMS 预后差异可能较大。因此,临床医生能否尽早发现并明确诊断,选择适宜的手术时机,并争取最佳手术机会,将直接影响患者的生存及预后。

参考文献:

[1] Mentzel T. Myofibroblastic sarcomas: a brief review of sarcomas showing a myofibroblastic line of differentiation and discussion of the differential diagnosis[J]. Curr Diagn Pathol, 2001, 7(1): 17 – 24.

[2] Fletcher C D M, Unni K K, Mertens F. World Health Organization Classification of Tumours. Pathology and Genetics of Tumours of Soft Tissue and Bone[M]. Lyon: IARC Press, 2002: 94.

[3] Miyazawa M, Naritaka Y, Miyaki A, *et al*. A low-grade myofibroblastic sarcoma in the abdominal cavity[J]. Anticancer Res, 2011, 31(9): 2989 – 2994.

[4] Coffin C M, Humphrey P A, Dehner L P. Extrapulmonary inflammatory myofibroblastic tumor: a clinical and pathological survey[J]. Semin Diagn Pathol, 1998, 15(2): 85 – 101.

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