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·病例报道·

Ultrasonic manifestations of Von Hippel-Lindau syndrome with polycythemia: a case report

VHL综合征合并红细胞增多症超声表现1例

吴月双

[中图法分类号]R445.1 [文献标识码]B

患者女，29岁，已婚未育。自诉口渴多饮，近1个月体质量减轻3 kg。体格检查：精神状态好，体质量指数19.0 kg/m^2，血压、呼吸、心率正常，无头晕头痛，无恶心呕吐，无腹痛腹泻，无既往疾病史，无家族遗传病史。实验室检查：空腹血糖12.55 mmol/L，丙氨酸氨基转移酶55.1 U/L，余未见异常。超声检查：胰腺形态大小失常，胰头4.4 cm，胰体3.6 cm，胰尾3.8 cm，实质内可见多个弥漫分布的囊性回声，最大者2.1 cm×1.6 cm，内透声差（图1），主胰管显示不清；右肾大小12.5 cm×6.5 cm，左肾大小14.2 cm×7.6 cm，实质内均可见多个大小不等的囊性回声，右肾最大者4.1 cm×3.8 cm，左肾最大者5.9 cm×5.1 cm，内透声差（图2）。余部肾实质回声增强。左肾窦区靠近下盏见一大小约0.5 cm×0.3 cm强回声，后方伴声影。CDFI示未见明显异常。超声诊断：①胰腺多囊性改变（考虑多囊胰腺）；②双肾多囊性改变（考虑多囊肾）；③左肾结石。经临床行全身多系统检查确诊为VHL综合征，并行对症治疗。出院后随访3年，超声均提示胰腺、双肾均无变化；血常规检查提示红细胞计数升高，数次检查范围（5.18~5.24）×10^{12}/L；实验室生化检查提示空腹血糖升高，数次检查范围为11.41~16.39 mmol/L，丙氨酸氨基转移酶升高，数次检查范围为47.4~50.3 U/L。

讨论：VHL综合征是一种罕见的常染色体显性遗传性疾病，发病率约0.25%，具有家族性、多系统肿瘤的特征，为位于3p25-26染色体肿瘤抑制基因突变所致[1]。其病程长，发病缓慢，病变累及多个器官，临床表现多种多样，如表现为中枢神经系统血管母细胞瘤、视网膜血管瘤、胰腺囊肿、肾透明细胞癌和（或）多发性肾囊肿的多器官肿瘤综合征，部分合并肾上腺嗜铬细胞瘤。临床上根据是否有嗜铬细胞瘤将VHL综合征分为两型：VHL-1型不伴有嗜铬细胞瘤；VHL-2型伴有嗜铬细胞瘤，并进一步分为3种亚型，分别有不同的临床表现特点及突变类

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型[2], 本例为VHL-1型。本病诊断主要依据病史、影像学检查和眼底等检查, 以及针对VHL的基因进行检测。本例第一次随访起开始发现红细胞计数升高, 可能与VHL基因的突变导致红细胞增多密切相关。VHL综合征目前尚无有效的预防及治疗手段, 主要是对症治疗及密切随访。基于VHL综合征临床表现的多异性, 超声对其的诊断有重要意义: 早期可发现视网膜、腹部器官和泌尿、生殖系统等病变, 为临床准确诊断提供可靠依据; 后期随访中超声可清晰显示病变的大小、变化、血供情况及其与周围组织的关系, 直观了解病程进展, 有助于及时调整治疗方案, 且超声具有安全无辐射、可重复检查的优势, 是临床检查的首选。

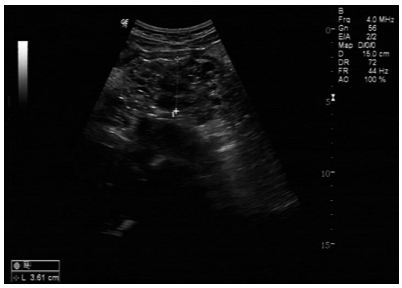

图1 胰腺多囊样改变声像图示胰腺肿大

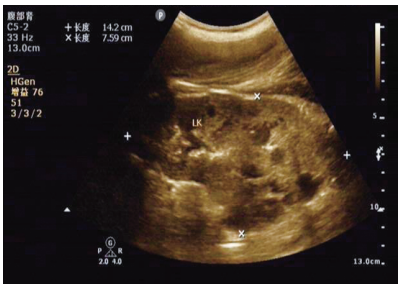

图2 肾脏多囊样改变声像图示肾脏肿大

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