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暴发性1型糖尿病合并急性肾功能衰竭1例

刘 娇,桂灵升,张 群,胡 蕖 (410220 重庆,重庆市长寿区人民医院内分泌内科)

[**关键词**] 暴发性1型糖尿病;肾功能衰竭
[**中图法分类号**] R587.1 [**文献标志码**] A

1 临床资料

患者,男性,29岁,因上腹部不适伴恶心、呕吐、腹泻2 d入院。入院前2 d患者饮酒后出现上腹部不适、恶心、呕吐,伴腹泻、乏力、纳差、口干、多饮,入我院急诊科,急诊以"急性胃肠炎"收入消化内科。查体:体温36.8 ℃,脉搏84 /min,呼吸21 /min,血压84/44 mmHg,BMI 18 kg/m^2,表情痛苦,意识清楚,双肺呼吸音清,心率84 /min,律齐,全腹散在压痛,无反跳痛及肌紧张。入科后予吸氧、抑酸、抗感染等治疗,完善肾功能示尿素19.5 mmol/L、肌酐188.7 μmol/L、尿酸637.8 μmol/L。电解质示二氧化碳结合力5.6 mmol/L、钾6.73 mmol/L、钠106.7 mmol/L、氯71.8 mmol/L、钙1.81 mmol/L。血气分析:pH 7.02、氧分压65 mmHg、红细胞剩余碱-26.6 mmol/L、血气血糖27.8 mmol/L。上腹部CT:双下肺肺炎,双侧胸腔积液,脂肪肝。立即转入肾内科予血液净化、胰岛素降糖、补液、抗感染等治疗后病情好转,入院第2天完善尿常规示酮体(+)、葡萄糖(++)。复查肾功、电解质恢复正常。入院后第4天因血糖控制欠佳转入我科治疗,测定糖化血红蛋白6.1%,糖尿病相关抗体阴性。空腹C肽<0.1 ng/mL,75 g葡萄糖刺激后2 h C肽<0.1 ng/mL。确诊为暴发性1型糖尿病合并急性肾功能衰竭、电解质代谢紊乱。予胰岛素泵降糖治疗后血糖下降。最终降糖方案为3餐前皮下注射诺和锐各7 U、晚上10点注射甘精胰岛素10 U,血糖控制平稳。出院3个月后随访,空腹血糖为5.2 mmol/L,糖化血红蛋白6.3%,空腹C肽仍<0.1 ng/mL。

2 讨论

暴发性1型糖尿病(fulminant type 1 diabetes mellitus F1D)由日本学者Imagawa等[1]于2000年首次报道。符合以下3条即可确诊:(1)高血糖症状出现7 d内发生酮症或酮症酸中毒;(2)血糖≥16.0 mmol/L且

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[**通信作者**] 胡 蕖,E-mail:270975914@qq.com
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细胞化生,胶原纤维增生。而相对更重要的气道壁增厚包括气道下纤维沉积,平滑肌肥大增生则必须经过较长时间的持续激发,本实验条件下至少需要激发8周。本研究结果阐明了气道重塑病理特征的形成需要大约8周的时间激发,且长时间持续激发并未诱导免疫耐受而导致气道炎症的减轻甚至消失,为哮喘气道重塑模型的构建提供了重要参考。但停止激发以后气道炎症和气道重塑改变能否继续维持,且本研究中缺乏气道高反应性的检测,因此仍需进一步研究,为今后气道重塑的研究提供更多的依据。

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初诊时HbA1c <8.5%;(3)发病时胰岛功能满足二者之一:①24 h尿C肽<10 μg/d;②空腹C肽<0.3 ng/mL和餐后C肽(或)胰高糖素刺激试验<0.5 ng/mL。除此之外,还可能有以下临床特点:胰岛相关抗体阴性;常常在1周内起病;98%的患者血清胰酶增高;70%的患者起病前有类似感冒样症状或胃肠道症状;可发生在妊娠或分娩后[2]。本例患者为急性起病,起病前有上腹部不适、恶心、呕吐等胃肠道症状,2 d即出现糖尿病酮症酸中毒。入院时血糖>16 mmol/L,糖化血红蛋白水平仅为6.1%。胰岛相关抗体测定均为阴性,空腹C肽<0.1 ng/mL,75 g葡萄糖刺激后C肽<0.1 ng/mL。据以上临床特点及检查结果本例患者可确诊为F1D。

F1D的具体发病机制尚不明确,大多数学者认为遗传易感性、病毒感染、自身免疫及妊娠可能与本病的发生有关[3]。国内许多报道F1D大多发生在妊娠期间或分娩后[4],对于青年男性发生FID合并急性肾功能衰竭却鲜有报道。在临床工作中,对疑诊为F1D的患者应及时完善血糖、肾功能等检查,以免漏诊或误诊。

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