慢性氣喘病人在運動(exercise)誘發性或醯丑甲基 膽鹼 (methacholine) 誘發性氣道收縮下,有不 同的氣道過度反應機制

Different mechanisms of airway
hyperresponsiveness between different
mechanisms of airway
hyperresponsiveness between exercise
and methacholine-induced
bronchoconstriction in chronic asthma.

177-11-11

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摘要

此次研究目的主要是探索慢性氣喘病人,在運動及醯丑甲基膽鹼(methacholine)誘發性氣道收縮下,有不同的氣道過度反應機制。選取 41個慢性氣喘病人接受運動誘發性氣道收縮,及膽鹼誘發性氣道收縮測試。在測試前及測試後0、5、 10、 15、 30、 45、60、75 分鐘內,我們記錄用力吐氣第一秒量(FEV1);並計算其在六十分鐘內,用力吐氣第一秒量之減少量百分比與時間曲線下的面積(AUC 0-60 min),以及計算由最低用力吐氣第一秒量(maximum FEV1 decrease)開始,到回復95% 用力吐氣第一秒量基準量所需要的時間(Recovery time)。在41個受試者中,有12個有運動誘發性氣道收縮,29個沒有運動誘發性氣道收縮。結果發現,有運動誘發性氣道收縮的受試者,其AUC 0-60 min以及recovery time皆比沒有運動誘發性氣道收縮的受試者來的高(1201.0 ± 70.0, n=12 versus 328.0 ± 28.0 % min,n=29,p<0.0001;109.2 ±26.5, n=11 versus 36.9 ± 5.9 min,n=28,p<0.0001)。這兩群 [EIB(+) and EIB(-)] 在接受膽鹼誘發性氣道收縮測試後,AUC 0-60 min以及recovery time皆無顯著差別(1136.0 ± 115.8 versus 1121.0 ± 122.7 %・min,p=0.936;111.5 ± 14.2 versus 106.0 ± 14.3 min,p=0.757)。此外,比較運動誘發

性或醯丑甲基膽鹼誘發性氣道收縮AUC 0-60 min,發現並無明顯的正相關 (r=024)。因此,藉由比較運動或醯丑甲基膽鹼誘發性氣道收縮後,兩者用力 吐氣第一秒量之減少量百分比與氣道擴張回復時間的差異性,推測兩者應該有不 同的氣道過度反應機制。

Abstract

Purpose: The aim of this study was to explore the different mechanisms of exercise-(EIB) and methacholine-(MIB) induced bronchoconstriction in patients with chronic asthma. Methods: We measured the FEV1, recovery time (RT), and AUC 0-60 min (area under the curve from 0 to 60 min after exercise in FEV1) in 41 asthmatics, who received exercise and methacholine challenge tests. Results: Among these asthmatics, 12 asthmatics had EIB and 29 had no EIB. The recovery time was prolonged (109.2 \pm 26.5 min, n= 11, p<0.001) and AUC 0-60 min was larger (1201.0 \pm 70.0 % • min, p<0.0001) in the EIB group, compared to the non-EIB group (RT: 36.9 ± 5.9 min; AUC: 328.0 ± 28.0 %·min, respectively, n=28). There was no difference in AUC and RT after methacholine-induced bronchoconstriction between the EIB and non-EIB groups (1136.0 \pm 115.8 versus 1121.0 \pm 122.7 % · min, p= 0.936; 111.5 \pm 14.2 versus 106.0 \pm 14.3 min, p=0.757). There was no significant correlation between the magnitude of AUC induced by exercise test or methacholine challenge (r= 0.24) Conclusion: We suggested that there were different mechanisms between EIB and MIB. The delayed bronchodilation in the EIB asthmatic subjects was probably related to presence of bronchoconstrictors.