The Dynamic study of the Pulmonary Function with the Static Image of 99m-Tc-MAA Pulmonary Scan

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99m-Tc-MAA pulmonary scan might be thought out of date, because 133-Xe perfusion scan is able to demonstrate the active respiratory function and is used for the analysis of the real pulmonary function. But I had some experiences of the fact that the static pulmonary scan with 99m-Tc-MAA is still able to diagnose the pulmonary function of the respiratory distress symdrome.

The defect negative image of the 99m-Tc-MAA pulmonary scan is meant that those lesions are the areas of poor blood circulation. The differential diagnosis of those cases are considered as the pulmonary tumor, inflammatory disease of exsudation, lymph nodal swelling, pulmonary embolism and respiratory distress symdrome.

Our first case presented here is 8 year old girl, who suffered dyspnea and had tracheotomy. The cause of dyspnea is not clear, and she had caught cold three months before admission.

Her chest radiogram revealed only pleural adhesion scars at the right lower lobe. But P_{co2} was more than 70 mmHg and 99m-Tc-MAA pulmonary scan revealed the large defects at the right lobe and moth eaten appearances of the left lobe. Six months later, her dyspnea is slightly improved. And 99m-Tc-MAA scan view is also improved.

The second case is 46 year old man, and he was suffered the severe interstitial pulmonary fibrosis. But the 99m-Tc-MAA scan is revealed almost normal, and P_{co2} is within normal. Those facts demonstrate the 99m-Tc-MAA scan is usefull to evaluate the pulmonary function, and might be made clear its prognosis.

Studies on Plasma Prostaglandin Levels in Patients with Bronchial Asthma Measuring by Radioimmunoassay Method

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The plasma prostaglandin (PGE_1, $PGF_{2\alpha}$) levels and prostaglanidn $F_{2\alpha}$ MUM of one hundred and ten patients with bronchial asthma, thirty one patients with pulmonary tuberculosis and other respiratory diseases and thirty seven healthy individuals were measured by radioimmunoassay method.

The results were obtained as follows:

1. The mean of PGE_1 levels were 1.63 ± 1.76 ng/m*l* in normals, 5.06 ± 10.69 ng/m*l* in attack free interval, 2.02 ± 2.90 ng/m*l* in athma attack and 0.55 ± 10.28 ng/m*l* in pulmonary tuberculosis.

The plasma PGE_1 levels of the patients with pulmonary tuberculosis were decreased significantly compared with normal subjects ($0.02 < p < 0.05$).

The mean of plasma $PGF_{2\alpha}$ levels were 0.63 ± 0.86 ng/m*l* in normal individuals, 0.43 ± 0.31 ng/m*l* in attack free interval, 2.54 ± 3.41 ng/m*l* in asthma attack and 0.24 ± 0.07 ng/m*l* in pulmonary tuberculosis.

$PGF_{2\alpha}$ levels in asthma attack were most increased of them ($0.02 > p > 0.05$).

2. The mean $PGF_{2\alpha}/E_1$ ratios were 0.47 ± 0.36 ng/m*l* in normal subjects, 0.50 ± 0.44 ng/m*l* in attack free interval, 1.46 ± 1.87 ng/m*l* in asthma attack and 0.57 ± 0.14 ng/m*l* in pulmonary tuberculosis; $PGF_{1\alpha}/Et$ ratios in asthma attack were the higher of other three groups ($0.02 > p > 0.05$).

3. The mean of $PGF_{2\alpha}$ -MUM levels in 24 hours were 11.32 ± 3.78 μg/day in normal subjects, 10.58 ± 7.13 μg/day in attack free interval 14.65 ± 7.55 μg/day in asthma attack and 4.29 ± 2.34 μg/day; $PGF_{2\alpha}$-MUM levels in asthma attack