

7. Kongres Hrvatskog  
torakalnog društva

7<sup>th</sup> Congress of Croatian  
Thoracic Society

# TORAKS

## 2017

Hotel Westin Zagreb

**26. - 29. TRAVANJ / APRIL**



## BACK TO BASICS - UNUSUAL SIDE EFFECT OF MUSCARINIC ANTAGONIST

Grubić Rotkvić P.<sup>1</sup>, HULJEV ŠIPOŠ I.<sup>2</sup>, Lukenda J.<sup>1</sup>, Jurić I.<sup>3</sup>, Labor M.<sup>4, 5</sup>

<sup>1</sup> KB "Sveti Duh", Zagreb, Croatia

*Zavod za bolesti srca i krvnih žila*

<sup>2</sup> Opća bolnica Šibenik, Šibenik, Croatia

*Odjel interne medicine*

<sup>3</sup> Klinički bolnički centar Osijek, Osijek, Croatia

*Zavod za bolesti srca i krvnih žila*

<sup>4</sup> Klinički bolnički centar Osijek, Osijek, Croatia

*Zavod za pulmologiju*

<sup>5</sup> Sveučilište J.J. Strossmayera, Osijek, Croatia

*Medicinski fakultet*

Background: Anticholinergic effects are produced by the inhibition of cholinergic neurotransmission at muscarinic receptors sites. Substances with anticholinergic properties competitively antagonize acetylcholine muscarinic receptors. There are five types of muscarinic receptors based on their pharmacological activity (M1-M5). Anticholinergic agents have proved to be of particular value in the treatment of COPD. Anticholinergics block muscarinic receptors on airway smooth muscle and submucosal gland cells. Three subtypes of muscarinic receptors have been demonstrated in human airways. M1 in parasympathetic ganglia facilitate cholinergic neurotransmission. M3 on airway smooth muscle cells and glands mediate bronchoconstriction and mucous

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secretion. M2 at cholinergic nerve endings inhibit the release of acetylcholine (act as feedback inhibitory receptors). Furthermore, several lines of evidence have shown that both M2 and M3 receptors are postjunctionally expressed in many smooth muscles, including the gastrointestinal tract and urinary bladder. Aclidinium bromide is a novel potent long-acting inhaled muscarinic antagonist that has affinity for the five muscarinic receptors and demonstrated kinetic selectivity for M3.

Case report: We are presenting a case of an unusual reaction to an inhaled anticholinergic agent. A 63-year old patient was admitted to hospital for coronary angiography. He has been receiving bronchodilators and inhaled corticosteroids (salmeterol, fluticasone, ipratropium) since 2015 due to COPD with known emphysema (FEV1 50%, FEV1/FVC 48%, DLCO 33%). One week before admission, ipratropium was substituted with aclidinium bromide. A day after coronary angiography was done, he developed paralytic ileus and urine retention. Urine catheterization and nasogastric intubation were performed. The patient received neostigmine and aclidinium intake was stopped. On day after, the patient had the first stool, a bowel distension was still present, but he felt better and normal urination was restored. Subsequently, an extensive gastrointestinal tract examination was performed (CT scan, Barium studies, gastroscopy), but no pathological findings were detected.

Conclusion: Common delayed reaction to iodinated contrast agents are rash, nausea, vomiting, diarrhea, hypotension and contrast-induced nephropathy. There have not been found reactions such as ileus in literature. Moreover, iodinated contrast agents given orally can induce hyperperistalsis. Considering all that above, the reaction was probably induced by anticholinergic effect of aclidinium bromide. Systemic effects should always be considered with such agents.