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New treatment options for lung viral diseases: the pharmacology of the non-classical renin-angiotensin system. A review

SARS-CoV-2, SARS-CoV and MERS-CoV bind to ACE2, cell membrane receptor of these β-coronaviruses. Monocarboxypeptidase ACE2 activity stops upon viral entry into cells, leading to inadequate tissue production of angiotensin 1-7 (Ang1-7). Acute lung injury due to human respiratory syncytial virus (hRSV) or avian influenza A H7N9 and H5N1 viruses is also characterized by downregulation of lung ACE2 and increased systemic levels of angiotensin II (Ang II). Restoration of Ang1-7 anti-inflammatory, anti-fibrotic, vasodilating, and natriuretic properties was tried in Covid-19 patients through i.v. infusion or intranasal administration of modified ACE2 protein: it proved useless. Conversely, use of ACE inhibitors (ACEis), which increase ACE2 cell expression, improved the prognosis of hypertensive patients with Covid-19.

To restore Ang1-7 tissue levels in all these viral diseases and avoid the untoward effects related to ACE2 administration, a strategy may be hypothesized. When metallopeptidase inhibitors block ACE2, neprilysin (NEP), highly expressed in higher and lower airways, starts cleaving angiotensin I (Ang I) into Ang1-7. A discerning use of ACEis should be made in patients with the above-mentioned viral pneumonia, to block ACE-dependent Ang II synthesis and Ang1-7 degradation into angiotensin 1-5; concurrently, i.v. infused Ang I, which is not hypertensive provided ACE is inhibited, may become the primary substrate for Ang1-7 synthesis by ubiquitous NEP. That is, NEP could replace ACE2 to produce Ang1-7 in tissues, if Ang I were freely available, in coronavirus diseases and in atypical pneumonia caused by avian influenza viruses and hRSV. Finally, inhibitors of chymase, serine endopeptidase responsible for 80% of Ang II synthesis in tissues and vessel walls, could protect patients with atypical pneumonia from Ang II–mediated microvascular damage without reducing arterial pressure.

Keywords

coronavirus, Covid-19, renin angiotensin system, cytokine storm, metallopeptidases, neprilysin.

Biography

Giovanni Sansoe', MD. Assistant professor, Division of Gastroenterology, Humanitas Institute, Gradenigo Hospital, Torino, Italy, from 1998 to 2025. 2011-2014. Scientific Consultant to Shire (pharmaceuticals). June 2008-November 2008. Clinical Associate Professor in Hepatology (University of Calgary, Alberta, Canada). July 2004-today. Clinical Fellowship (University of Toronto, Ontario, Canada). Internationally recognized expert in the pharmacology of classical and non classical renin-angiotensin system (RAS) in hepatic, renal and viral diseases.

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