**P056 RECURRENT ACE-INHIBITOR INDUCED ANGIOEDEMA IN A PATIENT WITH LONG COVID-19 SYNDROME**

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**Background/Aims**

Here we present the case of a 47-year-old female with long COVID-19 syndrome presenting with four episodes of acute onset angioedema in the absence of urticaria or pruritis affecting the mucosal layer of the lips and tongue.

**Methods**

She had been taking lisinopril for the last 18 months. She was diagnosed with COVID-19 in March 2020 presenting with fever, cough, shortness of breath and anosmia. She did not require hospital admission initially however has been readmitted twice with profound breathlessness and fever. She has subsequently developed a long COVID-19 syndrome with a daily, fluctuating fever of up to 39 °C for the last six months, profound fatigue, persistent shortness of breath with bilateral ground glass changes seen on high resolution CT chest imaging and mild anaemia. During this time, she subsequently reported four episodes of acute onset of severe angioedema affecting the lips and tongue for which she self-medicated with anti-histamines.

**Results**

Following disclosure of these episodes her lisinopril was ceased and there have been no further episodes to date. There are two other case reports in the literature that propose a potential relationship between angiotensin-converting enzyme (ACE) inhibitor induced angioedema and COVID-19. It is known that SARS-CoV-2 binds with high affinity to angiotensin converting enzyme 2 receptors which are found in oral tissues, heart, kidneys and the lung. ACE inhibitors can be associated with angioedema in approximately 0.5% of patients prescribed an ACE inhibitor. It occurs most commonly in the first year following instigation of treatment but can occur years later. The proposed mechanism of action is via the degradation of bradykinin. Bradykinin is converted from Kallikrein by C1 esterase, and ACE in turn metabolises bradykinin.

**Conclusion**

Bradykinin affects vascular permeability and results in vasodilatation, when patients take an ACE inhibitor there is a slower rate of bradykinin degradation and therefore this accumulation results in ACE inhibitor induced angioedema. SARS-CoV-2 binds to the ACE2 receptors. ACE2 is thought to counterbalance ACE in the renin-angiotensin system. It is theorised that the potential downregulation of ACE2 by the presence of bound SARS-CoV-2 may therefore potenciate angioedema in susceptible individuals. At present we cannot confirm this proposed relationship and global current consensus supports the ongoing use of ACE inhibitors during the pandemic but clinicians should continue to be aware of emergent pathologies related to the COVID-19 pandemic.

**Disclosure**

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