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Letter to the Editor

Thyroid storm, clouds and silver linings

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Dear Editor,

In addition to the use of personal protective equipment (PPE), significant interest has developed recommending topical application of Povidone Iodine (PVP-I) for pre-exposure prophylaxis (PEP) against COVID-19 transmission. PVP-I is safe, demonstrating a 99.9% reduction in MERS and SARS activity, far greater than chlorhexidine or benzalkonium-chloride [1].

As oral and nasal fluids contain greater than 10^7 COVID-19 viruses per mL, PVP-I is a theoretically useful PEP adjunct with PPE.

However, the evidence for using PVP-I as PEP, is neither clear nor clinical, extrapolations were made assuming Covid-19 will be just as susceptible to PVP-I in-vivo, as MERS and SARS were in-vitro [1].

Placing those concerns aside, 15% of the population with Thyroid disease are excluded from PVP-I use, but a similar proportion, who are as yet undiagnosed for thyroid disease remain at physiological risk from inadvertent exposure to iodine in PVP-I [2].

In healthy patients, iodine exposure initiates the Wolff-Chaikoff effect; transiently reducing thyroid hormone levels and this is a valuable response to control hypermetabolic morbidity and mortality from the thyroid storm [3].

However, for patients with unrecognised thyroid dysfunction, iodine exposure can either lead to chronic hypothyroidism with no adaptation to Wolff-Chaikoff down-regulation, or it can lead to the Jos-Basedow phenomenon and critical thyroid storm initiation from a loss of negative feedback [3].

Therefore the 15% of patients excluded from PVP-I use are not placed at risk but those patients undiagnosed as yet not recognised as having a thyroid dysfunction will be placed at risk if exposed to PVP-I.

If the bidirectional relationship between thyroid disease and kidney function is considered with regard to renal drug excretion; the association of cardiovascular and psychiatric illness with underlying and undiagnosed thyroid disease is problematic. With such patients, the risk of adverse pharmacokinetic drug interactions demands even closer attention if we are to use PVP-I for any patient.

Given the positive relationship of thyroid function with RAAS (renin-angiotensin-aldosterone-system), the potential to amplify ACE 2 expression with PVP-Iodine exposure could increase COVID-19 infection risk as more ACE 2 receptors become activated and the potential for viral cellular entry increases [4].

Thyroid disease exhibits a 10:1 female to male predisposition [2], whereas the morbidity and mortality from COVID-19, with equal infectivity is predominantly male. Using PVP-I as PEP may be intuitive, but paradoxically counter-productive; as a greater risk of infection arises in patients more at risk from the effects of COVID-19 by using PVP-I.

With 37 isotopes and as many molecular formulations, iodine has many benefits, for example; Silver-Iodide can precipitate rainfall and disinfect surfaces, but there are environmental and medical risks too.

To realise the full PEP potential of PVP-I against COVID-19, the theoretical concerns outlined above require further analysis.

Thyroid patients are not at increased risk of morbidity and mortality from COVID-19 [5] and this silver lining in a nebulous cloud of PEP should be recognised when considering measures that could potentially do more harm than good.

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