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Misuse of the Early Warning Score charts "Chronic Hypoxia" modifier

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Forth Valley Royal Hospital (FVRH) employs 'Chronic Hypoxia' modification to the National Early Warning Score (EWS) chart. This aims to reduce development of type 2 respiratory failure (T2RF) in at risk patients by maintaining peripheral oxygen saturations between 88 and 92%. However anecdotal reports suggested that this modification had been misunderstood, resulting in patients admitted to the medical admissions unit being inappropriately exposed to hypoxaemia. We conducted this survey to explore the validity of these concerns prior to deployment of the next local version of the EWS chart. The British Thoracic Society (BTS) guideline on oxygen prescribing (1) was employed as a standard. To summarise, initial target saturations of 88-92% are recommended in patients with conditions placing them at risk of developing T2RF until ABG results are available. If hypercapnia is present then target saturations of 88-92% should be continued. However if the result demonstrates eucapnia, an oxygen saturation target of ≥ 94% should be employed, with a repeat ABG 30 minutes later to allow adjustment of treatment.

Methods: Data was collected from patients in whom the 'Chronic Hypoxia' option was selected over a 2 week period in June 2015 in the Medical Admissions Units at FVRH. A retrospective analysis of notes was performed, assessing risk factors for T2RF, arterial blood gas (ABG) result and interpretation and adherence to BTS guidance. 28 patients were included in this study (11 male and 17 female patients). 27 patients were at risk of T2RF due to COPD. One patient had target saturations inappropriately adjusted (background of pulmonary hypertension due to left heart disease).

Results: ABGs were performed in 50% of cases. Within this group five patients were appropriately continued on a permissive hypoxaemia regime. However the remainder (n=9) continued with a target saturation of 88-92% despite the absence of hypercapnia Although BTS guidelines were initially followed, the majority of these patients were inappropriately maintained on a permissive hypoxaemia regime. Education of nursing and medical staff on the appropriate employment of permissive hypoxaemia is required to improve patient safety and adherence to national guidelines on oxygen prescribing.

Conclusion: This audit provides evidence supporting changing the "Chronic Hypoxia" box to a "Chronic Hypercapnia" box on the local EWS chart. This change was planned for early 2016. We plan to assess medical and nursing staff understanding of the criteria for employment and a further survey will be performed to assess the impact of the planned interventions

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Molecular insights into lung cancer subtypes and therapeutic targeting approaches involving HDACi

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The Janus tyrosine kinases JAK1-3 and tyrosine kinase-2 (TYK2) are frequently hyperactivated in tumors. In lung cancers JAK1 and JAK2 induce oncogenic signaling through STAT3. A putative role of TYK2 in these tumors has not been reported. We found a previously not recognized TYK2-STAT3 signaling node in lung cancer cells. We reveal that the E3 ubiquitin ligase seven-in-absentia-2 (SIAH2) accelerates the proteasomal degradation of TYK2. This mechanism consequently suppresses the activation of STAT3. In agreement with these data the analysis of primary non-small-cell lung cancer (NSCLC) samples from three patient cohorts revealed that compared to lung adenocarcinoma (ADC), lung squamous cell carcinoma (SCC) show significantly higher levels of SIAH2 and reduced STAT3 phosphorylation levels. Thus, SIAH2 is a novel molecular marker for SCC. We further demonstrate that an activation of the oncologically relevant transcription factor p53 in lung cancer cells induces SIAH2, depletes TYK2, and abrogates the tyrosine phosphorylation of STAT1 and STAT3. Moreover, we demonstrate that epigenetic modulators belonging to the class of histone deacetylase inhibitors (HDACi) modulate pro- and anti-metastatic gene expression patterns in tumor cells. These involve among other key molecules the surface molecules CD44 and E-Cadherin as well as the tyrosine kinase ACK1. Our studies may help to identify molecular mechanisms affecting lung carcinogenesis and potential therapeutic targets and strategies.

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