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Mechanisms of endothelial regeneration and resolution of inflammatory injury

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Acute lung injury (ALI) and its severe form acute respiratory distress syndrome (ARDS) are complex, multi-factorial syndromes which manifest themselves by leaky lung microvessels, protein rich edema and hypoxemia. Despite recent advances on the understanding of the underlying mechanisms, there are currently no effective pharmacological or cell-based treatment of the disease with a mortality rate as high as 40%. Given that recent studies from both human and animal studies has demonstrated the key role of microvascular leakage in determining the outcome of sepsis and associated ARDS, targeting microvascular leakage repair mechanisms represents a novel, effective therapeutic approach for the prevention and treatment of ALI/ARDS. Employing genetic lineage tracing, we have demonstrated that resident endothelial cell proliferation is essential for endothelial regeneration following sepsis challenge. We have defined the crucial role of the Forkhead transcription factor FoxM1 in mediating the intrinsic endothelial regeneration program and delineated the signaling pathway mediated by the p110 gamma isoform of PI3K in mediating endothelial regeneration and vascular repair and resolution of inflammatory injury. We also identified endothelial FoxM1 as the endogenous mediator of the adult stem cell-elicited paracrine effects on protecting from acute lung injury. Our studies suggest that targeting the intrinsic FoxM1-dependent endothelial regeneration program is an effective therapeutic strategy for reversing lung microvessel leakiness and improving survival of sepsis and ARDS patients.

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Precision of blood documentation

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This study of documentation an important and essential component of clinical practice, of blood results within Wales' largest teaching hospital and assess the quality of documentation. The criteria we will work towards have been agreed by at least 50% of a cohort of 50 doctors working in the University hospital of Wales as essential documentation points. Over the space of our audit, we found in general documentation was not adequately adhering to CG2 guidelines with respect to blood results. Our first audit found that only 50% of blood results these were documented. After implementing a small change; a poster presentation and teaching session in junior doctor group teaching, our second audit showed worse documentation, with only 25% of blood results being documented. We then implemented a further change; consultant led teaching session during the foundation group mandatory programme. A questionnaire was distributed with over 50 responses returned. This showed that the over 90% of the cohort of doctors knew how to correctly document bloods and 100% of the doctors knew of at least one reason for which documentation was important. The third audit showed drastic improvement of blood results being correctly documented. In conclusion, our poster and teaching sessions have adequately highlighted to doctors the importance and correct method of documenting blood results. From our questionnaire it was obviously the main reason for poor documentation is time constraints. Electronic signature, to record that results have been actively looking at may be a viable method of saving junior's time.

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