vitro. Thus, we provide important insights into the changes occurring after prolonged in vitro expansion, underlining the importance of using low passage MSCs in clinical trials for ARDS. In agreement with published data, we also found that MSCs do not induce cellular proliferation in the absence of stimulation.

REFERENCES

PLASMA SYNDECAN-1 LEVEL AS A PREDICTIVE MARKER OF VASOPLAGIA ASSOCIATED WITH SURGERY REQUIRING CARDIOPULMONARY BYPASS AND POSSIBLE INVOLVEMENT OF OXIDATIVE STRESS

Background Vasoplegic syndrome (severe refractory hypotension) is associated with oxidative stress leading to endothelial dysfunction and complications 10 to 40% of surgery requiring cardiopulmonary bypass (CPB). Whilst operative mortality is low, recovery is often prolonged in patients developing vasoplegia. There are, as yet, no validated biomarkers for vasoplegia that could be used to identify ‘at risk’ patients. We hypothesised that plasma levels of the endothelial surface layer (glycocalyx) protein, syndecan-1, shed during CPB, will be higher in patients who develop vasoplegia and that leukocyte responses to oxidative stress will be altered.

Methods Patients (n = 48) undergoing cardiac surgery requiring CPB were, prospectively, enrolled; blood collected and indices of the cohort (p = 0.0061 and p = 0.0148, respectively). NQO1 relative expression was significantly higher (p = 0.022) in vasoplegic patients (3.779 ± 1.036) than non-vasoplegic patients (1.3 ± 0.302); whereas, neither SOD2 nor TNFα expression were significantly altered.

Conclusion Plasma syndecan-1 measured immediately post-CPB had good predictive power for patients at risk of vasoplegia. Greater relative expression of leukocyte NQO1 in vasoplegic patients indicates activation of antioxidant defence mechanisms in response to oxidative stress, which could contribute to syndecan-1 shedding.

PHARMACOKINETICS AND PHARMACODYNAMICS OF ANTIMICROSURICIY IN CRITICALLY ILL PATIENTS WITH LOWER RESPIRATORY TRACT INFECTIONS. ARE ‘ONE SIZE FITS ALL’ DOSES APPROPRIATE?

Introduction Respiratory infection is a common cause of severe sepsis. Current therapeutic guidelines emphasise the importance of early initiation of antibiotic therapy, but make no