Introduction CT pulmonary angiography (CTPA) is the recommended imaging modality for suspected pulmonary embolism (PE). Current NICE guidelines recommend using clinical prediction scoring systems to estimate the probability of PE and guide further investigation[1]. A low or intermediate probability score, coupled with a negative D-dimer, reliably excludes PE, thereby avoiding the need for CTPA.

Objectives We undertook a retrospective audit to examine adherence to NICE guidelines for diagnosis of suspected PE in patients admitted to a district general hospital, and identify patients who may have undergone unnecessary CTPA.

Methods We obtained a list of all CTPAs undertaken in our hospital between December 2012 and February 2013. D-dimer tests are poorly specific within hospitalised patients; therefore, we excluded post-surgical and obstetric patients, and pre-existing inpatients where primary admission was not for suspected PE. We also excluded outpatient CTPAs. We searched the records for contemporaneous PE probability scores and D-dimer results. For patients without a probability score result, we reviewed the clinical notes and calculated a probability score retrospectively using a local scoring system adapted from BTS guidelines.

Results There were 115 CTPAs during the study period – 36 were excluded and 4 patients’ case notes were unavailable. 75 patients fulfilled the inclusion criteria (mean age 68.2 years), and PE was confirmed in 20%. 50 patients (66.7%) had a contemporaneous documented clinical probability score. There were 5 patients (6.7%) with a low/intermediate probability score and negative D-dimer, who underwent unnecessary CTPA (PE excluded in each case). There were 9 patients (12%) with retrospectively calculated low/intermediate clinical probability scores and no D-dimer result, who may have avoided CTPA had D-dimer been undertaken (CTPA excluded PE in each case).

Conclusions In our district general hospital, the underuse of clinical probability scoring and D-dimer testing in patients with suspected PE is contributing to unnecessary CTPAs. Introducing mandatory documentation of PE clinical probability score on CTPA request forms may reduce the number of unnecessary CTPAs.

REFERENCES
following therapy with Bosentan (n = 6) or sildenafil (n = 1). Interestingly, improvement in CI at repeat RHC correlated positively with a further increase in HGF levels (r = 0.8, p = 0.03).

Results EPCs form a biological exclusion barrier with similar capabilities as mature hPAECs. TNFα significantly enhanced the permeability of EPCs (P < 0.05) and hPAECs (P < 0.05) monolayers. There is no difference in EPC colony formation between HC and SSc EPCs.

Discussion We developed a robust method for isolating EPCs from PBMCs. We have demonstrated that EPCs can maintain an endothelial barrier consistent with that observed by mature hPAECs in vitro. We have established that EPCs respond to TNFα in a similar manner to mature PAECs. We have shown no significant difference in the capacity of PBMCs from SSc patients to form EPC colonies compared to HCs.

Abstract P157 Figure 1.

Conclusion Plasma HGF concentrations correlate with haemodynamic severity in CTD-PAH. Further work to determine whether high levels of HGF are involved in the pathogenesis of PAH or are instead elevated in a reparative response to underlying pulmonary vascular remodelling is required.

REFERENCES

P158 ENDOTHELIAL PROGENITOR CELLS FORM BIOLOGICAL EXCLUSION BARRIERS SIMILAR TO THAT OF MATURE ENDOTHELIAL CELLS- A THERAPEUTIC POTENTIAL IN SYSTEMIC SCLEROSIS?

Abstract Vascular complications associated with systemic sclerosis (SSc) including pulmonary arterial hypertension (PAH-SSc), result from endothelial damage and loss of barrier function. Endothelial progenitor cells (EPCs) express endothelial (VEGFR2+, CD31+) and haematopoietic (CD133+) markers. They home to sites of vascular injury and differentiate into endothelial cells restoring the endothelium. In SSc patients circulating levels of EPCs are reduced. This study aimed to develop a robust method to grow EPCs from peripheral blood mononuclear cells (PBMCs) and to compare cellular functions to mature endothelial cells.

Methods EPCs and human pulmonary artery endothelial cells (hPAECs) were seeded into transwell inserts and grown to confluence. Cells were incubated with TNFα (50ng/ml) for 24h at 37°C. Following centrifugation, plasma was aspirated and samples frozen at -20°C, and IL-6 and CXCL8 ELISAs later performed. Data are mean ± SEM.

Results IL-6 and CXCL8 release was higher from PBMCs of patients compared to controls. For example for LPS-induced IL-6 release from PBMC, both the maximal effect (Emax) was lower in PAH vs. controls (1048 ± 288.4 p < 0.05) and the log EC50 (half maximal effective concentration) was higher in PAH vs. controls (5148 ± 1320 vs. 9154 ± 2510 pg/ml, p < 0.05) and the log EC50 (half maximal effective concentration) was higher in PAH vs. controls (5148 ± 1320 vs. 9154 ± 2510 pg/ml, p < 0.05) and the log EC50 (half maximal effective concentration) was higher in PAH vs. controls (5148 ± 1320 vs. 9154 ± 2510 pg/ml, p < 0.05).

Conclusion The main findings of this study are that: (1) baseline CXCL8 and IL6 release was higher from PBMCs of patients with PAH compared to control donors; (2) PBMCs and WB from patients were hyposensitive to LPS (and TNFα),