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[i]. 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.

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P156 NEUTROPHIL AND REDOX DEPENDENT PROTEOLYSIS OF BONE MORPHOGENETIC PROTEIN 9: POTENTIAL ROLE IN THE PATHOGENESIS OF PULMONARY ARTERIAL HYPERTENSION

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Introduction A critical reduction of bone morphogenetic protein type II receptor (BMPRII) in the pulmonary circulation, either due to the genetic loss-of-function mutations, heightened inflammation or prolonged hypoxia, is one of the major causes behind pulmonary arterial hypertension (PAH), a fatal disease with poor prognosis. BMPRII is highly expressed in the vascular endothelium and undergoes rapid turnover. Bone morphogenetic protein 9 (BMP9), the only active circulating BMP, signals via endothelial BMPRII, inducing BMPRII expression and maintaining endothelial homeostasis. Although BMPRII function has been studied extensively, factors that regulate BMP9 stability and activity remain unclear.
Objective To investigate how BMP9 activity and stability are regulated and whether this regulation plays a role in pulmonary arterial hypertension.
Results Two forms of BMP9 dimer could be co-purified, with (D-form) or without (M-form) intermolecular disulphide bond. M- and D-forms BMP9 are interchangeable with redox potential, but have different stability. While the M-form is more susceptible to redox-dependent cleavage and proteases present in serum, the D-form is a preferred substrate for neutrophil elastase. Freshly isolated human peripheral blood neutrophils, when activated by hypoxia or inflammatory stimuli, released elastase that cleaved BMP9 effectively.
Conclusions and Discussions This study demonstrates a novel proteolytic regulation of BMP9 under physiological and pathological conditions, suggesting neutrophil elastase could be a potential link between inflammation/hypoxia and BMPRII signalling, and the recognised benefits of elastase inhibition in rodent models of PAH may be due in part to reduced degradation of BMP9 and preservation of endothelial BMPR-II signalling.

P157 HEPATOCYTE GROWTH FACTOR CONCENTRATION CORRELATES WITH HAEMODYNAMIC SEVERITY IN CONNECTIVE TISSUE DISEASE-ASSOCIATED PULMONARY ARTERIAL HYPERTENSION

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Introduction Hepatocyte growth factor (HGF) acts via the tyrosine kinase receptor, c-MET, on endothelial and epithelial cells. It has angiogenic, mitogenic, motogenic and anti-apoptotic effects. Administration of HGF has been shown to ameliorate pulmonary arterial hypertension (PAH) in the monocrotaline rat model.[1,2] Little is known regarding circulating HGF levels in human disease.
Methods 47 incident, treatment naive patients with PAH in association with connective tissue disease (CTD-PAH) had blood sampling at or within 1 day of diagnostic right heart catheterisation. Plasma HGF concentrations were measured using Bio-Plex bead array. A proportion of patients also had NT-proBNP measured and underwent cardiac MRI.
Results Baseline characteristics were (mean, sd): Age 64(10)yrs, mean right atrial pressure (mRAP) 9.6(11.7)mmHg, mean pulmonary arterial pressure (mPAP) 40.6(13)mmHg, pulmonary arterial wedge pressure 10.5(4.5)mmHg, cardiac index (CI) 2.97 (0.7)L/min, pulmonary vascular resistance (PVR) 531(350)dyns. HGF levels correlated positively with mRAP (0.6, $r < 0.001$), mPAP ($r = 0.68$, $p < 0.001$: fig 1), PVR ($r = 0.51$, $p = 0.001$) and negatively with CI ($r = -0.43$, $p = 0.008$) and right ventricular ejection fraction measured by MRI ($r = -0.53$, $p = 0.034$). N-terminal pro B-type natriuretic peptide (NT-proBNP) measured in approximately 50% of patients correlated more strongly with CI ($r = -0.72$, $p < 0.001$) and PVR ($r = 0.61$, $p = 0.003$) but did not correlate with mPAP. A small proportion (7) of patients underwent repeat right heart catheterisation (RHC)