patients). The Abstract P253 Table 1 shows the effect of different variables on mortality. This was not affected by sex, transplant status or laboratory parameters. With logistic regression analysis, we found that haematological malignancies (OR 7.44, 1.96–28.20), long term steroids (OR 7.87, 2.36–26.20) and age >65 years (OR 3.94, 1.36–11.40) were significantly associated with mortality. The relationship with immunosuppressive drugs was less significant.

Abstract P253 Table 1

Variable	Died	Survived	Chi square
Haematological malignancy	21 (70%)	9 (30%)	7.37 (p=0.0006)
Long term steroids	21 (72%)	8 (28%)	8.7 (p=0.0003)
Interstitial lung disease	9 (69%)	4 (31%)	1.79 (p=0.18)
Connective tissue disease	7 (64%)	4 (36%)	0.39 (p=0.44)
Recent immunosuppressants or chemotherapy	30 (59%)	21 (41%)	3.7 (p=0.05)
PCP PCR positive	11 (58%)	8 (42%)	0.47 (p=0.49)
Age >65 years	26 (67%)	12 (33%)	9.58 (p=0.002)

Conclusions HIV negative immunocompromised patients with pulmonary infiltrates are more likely to die if they have a haematological malignancy, are on long term steroid therapy or aged >65 years. The effect of immunosuppressive drugs needs more elucidation. Careful attention needs to be paid to these groups of patients to pick up early signs of deterioration.

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P254 INVESTIGATING HMGB1 AS A POTENTIAL INFLAMMATORY MEDIATOR IN BRAIN DEATH INDUCED LUNG DAMAGE

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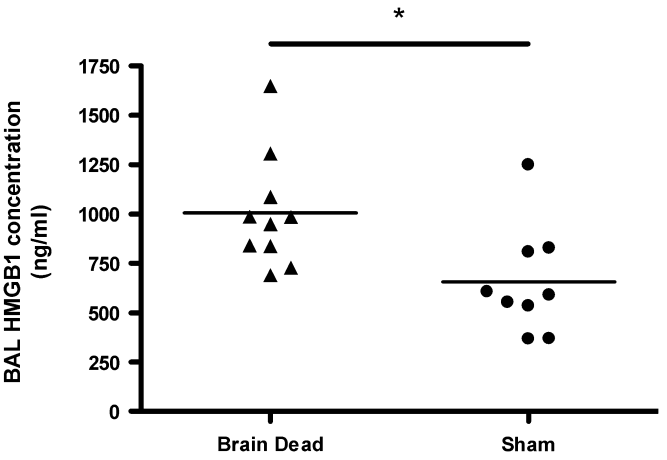
Background Donor lungs are particularly susceptible to the haemodynamic instability and systemic inflammation which occurs following induction of brain death (BD). Increased donor lung inflammation with IL-6 or IL-8 is associated with poor post-transplant outcomes. Donor LPS pre-conditioning significantly ameliorates the lung inflammation after BD in a rat model suggesting that TLR-4 ligation following BD contributes to donor lung inflammation, even in the absence of LPS. We hypothesised that HMGB1, an alarmin released from damaged tissue and immune cells and known TLR-4 ligand, may act as an inflammatory mediator in BD induced lung injury.

Methods BD was induced in a rat model using rapid intra-cranial balloon inflation and bronchoalveolar lavage (BAL), serum and lung tissue were collected and compared with sham operated controls rats. The HMGB1 concentration in the rat serum and BAL was measured by ELISA. Real-time PCR was used to assess HMGB1 mRNA expression in lung tissue following BD. HMGB1 immunolocalisation studies were performed on BD and sham rat lung tissue. Finally HMGB1 staining was assessed in lung tissue from human BD donors and normal controls.

Results The BAL HMGB1 concentration was significantly higher in the BD group (965±302 ng/ml) than in the sham group (655 ± 274 ng/ml) (p=0.0172). There was however no difference in HMGB1 gene expression between the two groups. HMGB1 positive staining was visualised in a nuclear and extra-nuclear location and was dispersed throughout the rat and human lung with a greater density around the bronchioles. There was a significantly higher area of positive staining in BD rat lung tissue than sham tissue (p=0.0345). No difference was seen in the human BD lungs compared to controls (Abstract P254 Figure 1).



Abstract P254 Figure 1

Conclusions HMGB1 is likely to be released passively from lung cells suffering sustained damage during BD. As HMGB1 gene transcription is unchanged up to 5 h following BD, the lung tissue is most likely releasing presynthesised cellular HMGB1. This study provides evidence of the presence of an alarmin which is likely to potentiate inflammation in the donor lung *via* the TLR-4 pathway. If release occurs early following BD, HMGB1 could be an important initiating mediator in donor lung inflammation.

Clinical studies in pulmonary embolism

P255 DEFINING A CIRCADIAN PATTERN OF PRESENTATION OF PULMONARY EMBOLISM ON CT/VQ IMAGING

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Purpose To evaluate whether there is an association between the chronological occurrence of pulmonary emboli, anatomical site, radiological extent and associated clinical risk factors.

Materials and Methods 1410 consecutive CTPA and VQ scans performed between December 2005 and October 2008 were retrospectively reviewed independently by 2 thoracic radiologists (kc=0.78). A third observer was used in equivocal cases. 270 cases of pulmonary emboli were identified. Records were available for 180 patients. 40 were excluded on the basis of incomplete presenting details. All diagnosed cases of pulmonary emboli were risk stratified according to the Wells score, underlying co-morbidities including diabetes, DVT, malignancy and cardiomyopathy. Morbidity, mortality and survival data were recorded. For 140 (Age 67 y SD ±17, 61 male), the occurrence of the pulmonary emboli based on onset of symptoms was defined by four time intervals. Each group was subdivided according to extent of clot. The average Wells score was calculated for each 6 h period.