

Conclusions Anaerobic infection occurs in ~33% of cases of pleural infection and is typically polymicrobial. Sequencing revealed many anaerobic bacteria never previously isolated in the pleural space. These bacteria have a strong association with the oropharynx, particularly the gingival crevices. Such findings add to our understanding of the mechanism of development of pleural infection.

S115 PLEURAL FLUID ADENOSINE DEAMINASE (ADA) IN THE DIAGNOSIS OF TUBERCULOUS PLEURAL EFFUSIONS IN A LOW INCIDENCE POPULATION

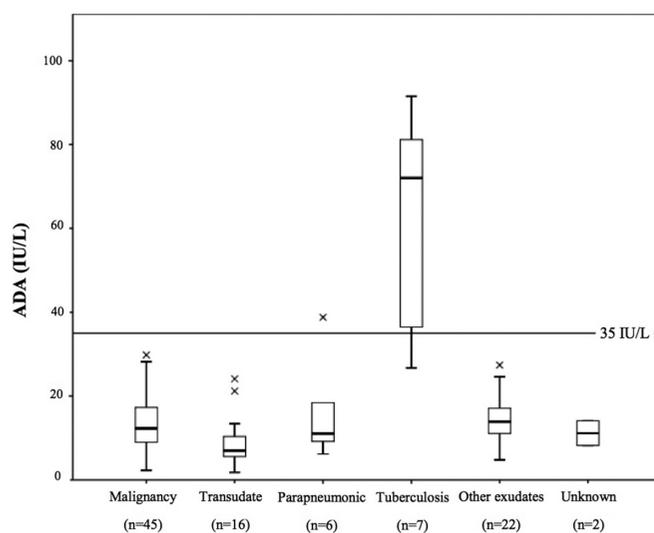
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Introduction Numerous studies have assessed the diagnostic ability of pleural adenosine deaminase (ADA) in detecting tuberculous pleural effusions, with good specificity and sensitivity reported. However, in the UK (UK) ADA is not routinely used in the investigation of a patient with a pleural effusion, mainly due to a lack of evidence as to its utility in areas where tuberculosis (TB) incidence is low.

Methods Patients presenting with an undiagnosed pleural effusion to a tertiary pleural centre in South-West England over a 3 year period, were prospectively recruited to a pleural biomarker study, in which baseline pleural fluid samples were collected and stored. Samples from consecutive patients with robust 12-month follow up data and confirmed diagnoses were sent for ADA analysis.

Results Of 338 patients enrolled, 7 had confirmed tuberculous pleural effusion (2%). All 7 TB effusions were lymphocyte predominant with a median ADA of 72.0 IU/L (range- 26.7 to 91.5) compared to a population median of 12.0 IU/L (range- 0.3 to 568.4). Using the established cut off of 35 IU/L, ADA was shown to have a negative predictive value (NPV) of 99.7% (95% CI; 98.2–99.9%) for the exclusion of TB, and sensitivity of 85.7% (95% CI; 42.2–97.6%) with an area under the curve of 0.88 (95% CI; 0.732–1.000). In the context of a lymphocytic effusion an ADA over 35 IU/L had a sensitivity and positive predictive value of 85.7% (95% CI; 42.2–97.6%), see figure. Bacterial pleural infection was the main alternative cause of raised ADA in our cohort.



Abstract S115 Figure 1 Boxplot of ADA levels by diagnostic category in lymphocyte predominant effusions

Discussion This is the first study examining the diagnostic utility of pleural fluid ADA in a low TB incidence area. The chance of an effusion with an ADA under 35 IU/L being of tuberculous aetiology was negligible and empirical anti-TB therapy could be avoided in such cases. A pleural ADA of over 35 IU/L in lymphocyte-predominant pleural fluid gives a strong suspicion of tuberculous aetiology. In patients who are unsuitable for more invasive procedures this could be used as an indication to start therapy.

S116 SYSTEMIC CHEMOTHERAPY AND THE RISK OF PLEURAL INFECTION WITH INDWELLING PLEURAL CATHETERS (IPCS)

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Introduction Indwelling pleural catheters (IPCs) are an alternative method for controlling recurrent, symptomatic malignant pleural effusions, currently reserved for cases of known trapped lung, or after an initial unsuccessful talc pleurodesis.¹ A 2.8% pleural infection rate has been observed in patients with an IPC² leading to the concern that this risk of infection may increase further with systemic chemotherapy.

Objectives

- Determine whether systemic chemotherapy increased the number of IPC related pleural infections.
- Whether the risk was related to the interval between chemotherapy and IPC insertion.

Methods A detailed retrospective review was performed of the first 100 patients treated with an IPC from February 2011 to December 2013 at Queen Alexandra Hospital, Portsmouth.

Results 11 of the 100 IPC insertions developed pleural infection (11%), four of whom had received chemotherapy (see table). Overall 15 patients received chemotherapy after IPC insertion with an interval varying from 0 days (same day) to 43 days, with a median of 9 days. The 2 × 2 table compared the proportion of patients developing pleural infection with and without chemotherapy.

Further analyses assessed the interval from chemotherapy to IPC insertion and whether it influenced the numbers of pleural infections, using cut-off points at 7, 14, 21 and 28 days. There was no significant difference at any of these time-points (p=NS).

Conclusions The pleural infection rate with an IPC was slightly higher than previous published series. There was however no significant increase in pleural infection in those receiving chemotherapy, although these analyses did not account for variability in tumour type, tumour stage, performance, co-morbidity and leucopenia at time of insertion. While the analyses were not significant, this needs to be retested in larger or combined registries of patients treated with IPCs for pleural malignancy.

Abstract S116 Table 1 A 2x2 contingency table assessing numbers of pleural infection with systemic chemotherapy in patients with an IPC

	Pleural infection	No pleural infection	
Chemo	4	11	15
No chemo	7	78	85
	11	89	

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S117 SURVIVAL IN PATIENTS WITH MALIGNANT PLEURAL EFFUSIONS WHO DEVELOPED PLEURAL INFECTION: A RETROSPECTIVE CASE REVIEW FROM 6 UK CENTRES

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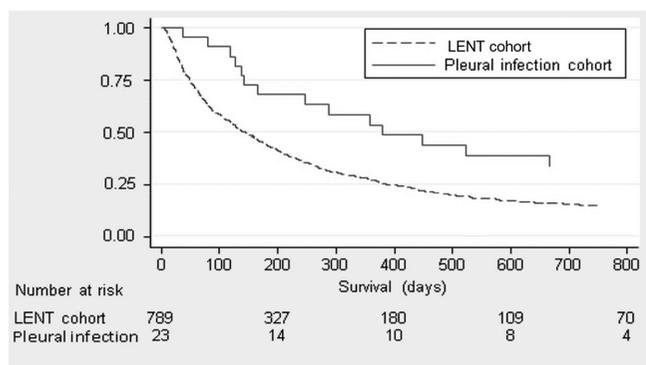
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Background The incidence of malignant pleural effusions (MPE) is increasing and overall prognosis remains poor. In-dwelling pleural catheters (IPCs) relieve symptoms, but increase the risk of pleural infection. We reviewed survival times of cases of pleural infection in patients with IPCs for MPE from 6 UK centres.

Methods Baseline data were collected for all IPC insertions from 1/1/05 to 31/1/14. Survival times were analysed by underlying tumour. Results were compared with national data, and with data from a cohort of 789 patients with MPE (the LENT cohort). LENT scores were used to calculate individual predicted life expectancy, which was compared with actual survival.

Results Of 672 IPCs inserted across 6 centres during the study period, 25 patients (3.6%) experienced pleural infection. 19/25 were male, median age 69 (range 35–79). 12/25 had mesothelioma, 8/25 lung cancer, 3/25 breast cancer, 1/25 lymphoma and 1/25 thyroid cancer. 18/25 had a performance status of 0–1, and 19/25 received oncological treatment.

Survival with MPE and pleural infection compared favourably with the LENT cohort (see figure 1). Median survival with mesothelioma and pleural infection was 753 days (95% confidence interval 446–1089) compared with 339 days in the LENT cohort (95% CI 267–442) and less than 365 days in nationally reported data. Patients with lung cancer and pleural infection also outlived their LENT counterparts; median survival of 138 days (95% CI 62–479) versus 74 days (95% CI 60–90). Patients with breast cancer had similar survival times (167 vs 192 days).



Abstract S117 Figure 1 Survival in patients with malignant pleural effusions who developed pleural infection: a retrospective case review from six UK Centres

LENT scores were calculated where possible. 9/13 (69%) outlived their predicted life expectancy. 16/25 (64%) developed infection within 90 days of IPC insertion. There was no difference in survival times between patients with early and late infection ($p = 0.6$).

Discussion In this series of patients with IPCs, pleural infection was associated with longer survival with mesothelioma and lung cancer, but not breast cancer. Most patients experienced early infection, suggesting this result isn't simply a result of higher infection rates in patients who survive longer with an IPC *in situ*. We propose that pleural infection stimulates a local immune response, which acts against tumour. Further studies are planned to investigate this hypothesis further.

Clinical investigations and outcomes in pulmonary vascular disease

S118 INCIDENCE AND SEVERITY OF CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION FOLLOWING THE INTRODUCTION OF A ONE-STOP CLINIC FOR ACUTE PULMONARY EMBOLISM

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Introduction The management and follow-up of pulmonary embolism (PE) is delivered by various specialities resulting in both under and over investigation for suspected chronic thromboembolic pulmonary hypertension (CTEPH). To standardise our approach to long-term PE management a “one-stop” clinic was established in Sheffield in March 2010 to review all patients approximately 3 months after their presentation with acute PE. The aim of this study was to evaluate the incidence and severity of CTEPH identified from a one-stop clinic using an investigative strategy based on careful clinical assessment

Methods Consecutive patients attending the one-stop PE clinic following hospital admission with acute PE were identified. During the one-stop consultation a haematologist and respiratory physician reviewed the patient jointly. The need for further investigation was based on clinical assessment. CTEPH was defined as mean pulmonary artery pressure (mPAP) at right heart catheterisation ≥ 25 mmHg and required multimodality imaging (isotope perfusion scanning, CT pulmonary angiography and MR imaging including MRA and MR perfusion mapping) demonstrating classical features of CTEPH.

Results Over a 3-year period between March 2010 and March 2013, 616 patients (mean age 67.7 years, 50% male) attended the one-stop PE clinic approximately 3 months following their acute presentation. 16 patients were diagnosed with CTEPH. An overall diagnostic rate of CTEPH of 2.6% for patients seen at the clinic and an annual incidence of 8.9/million/year was observed based on a referral population of 600,000. This compares to an annual incidence of CTEPH of 4.8/million/year in patients referred to the SPVDU over the same time period, based on a referral population of 15 million. The 16 patients with CTEPH had mPAP 37 ± 11 mmHg, pulmonary vascular resistance (PVR) 362 ± 240 dynes, significantly lower than patients with CTEPH diagnosed at the SPVDU until 2010 ($n = 242$) mPAP 48 ± 11 mmHg and PVR 735 ± 389 dynes (Hurdman *et al* *Eur Respir J* 2012;39(4):945–955).

Conclusion Introduction of a one-stop PE clinic for routine follow-up of patients with acute pulmonary embolism identifies