group the mean OCS dose is equivalent to 11.5 mg (± 7.6) of prednisolone. The mean of the highest historical eosinophil level was 700 cells/ microlitre (± 360).

Conclusions The following barriers to mepolizumab therapy have been identified:

- Poor adherence to ICS therapy in patients believed to be on optimal therapy.
- Long-term OCS treatment is suppressing the eosinophil count below 300 cells/microlitre

Firstly, this highlight the need for service improvement to ensure that adherence is regularly monitored. Those who are non-adherent can be referred to the RASP study or for inhaled nitric oxide (FeNO) monitoring. Secondly, patients on long-term OCS may be prevented from benefiting from the steroid sparing effects of mepolizumab. To achieve the necessary eosinophil counts steroids must be progressively reduced risking destabilisation of asthma control.

REFERENCE

1. National Institute for Health and Clinical Excellence. Mepolizumab for treating severe refractory eosinophilic asthma. *Nice Technology Appraisal Guidance* 2017;431.

P19

IMPACT OF MONTH OF INITIATION OF OMALIZUMAB ON TREATMENT OF SEVERE ALLERGIC ASTHMA, A SUB-ANALYSIS OF THE APEX II STUDY

¹R Niven, ²D Saralaya, ³R Chaudhuri, ⁴M Masoli, ⁵I Clifton, ⁶AH Mansur, ⁷S Hollywood, ⁸C McLain-Smith, ¹A Menzies-Gow. ¹University Hospital of South Manchester, Manchester, UK; ²Bradford Teaching Hospitals NHS, Bradford, UK; ³Gartnavel General Hospital, Glasgow, UK; ⁴Plymouth Hospitals NHS Trust, Plymouth, UK; ⁵St James's University Hospital, Leeds, UK; ⁶Birmingham Heartlands Hospital, Birmingham, UK; ⁷Novartis Pharmaceuticals UK Ltd, Frimley, UK; ⁸P H Associates Ltd, Marlow, UK

10.1136/thoraxjnl-2017-210983.161

Introduction and Objectives In asthma, seasonal variation in outcomes is known with seasonal peaks in exacerbation. Allergic inflammation is associated with greater susceptibility to viral infections. The UK real world studies, APEX I and II, demonstrated omalizumab reduced exacerbation frequency, healthcare utilisation, OCS burden and improved lung function in severe allergic asthma patients, but initial analyses excluded an assessment of seasonal impact. Patients initiated during their usual symptom season, may have inadequate time to suppress basophils and mast cells prior to exacerbating and consequently fail their 16 week treatment assessment assessing response. The objective was to determine if season/month of initiation had any impact on response (16 week clinical assessment according to usual clinical practice at each centre), hospital (A and E attendance and/or admission) and 'dose exacerbation', (OCS dose increase ≥ 10 mg for ≥ 3 days) rates. **Methods** The APEX II data was reanalysed, directly comparing response rate and frequency of exacerbations with time of initiation. We also looked at the pattern of seasonal exacerbations pre- and post-omalizumab initiation.

Results In the 258 cases included, response rate at 16 weeks where response was known was 82.4%. Highest response rates were in those initiated on treatment in December (90%) and July (89%) and lowest were January (62%) and August (69%). The total number of 'hospital exacerbations', over the 12 month period pre- and post-initiation was reduced from

362 to 151. Pre-initiation, there was a seasonal peak of hospital exacerbations (figure 1) from August to October. This was suppressed by omalizumab, with greatest reduction observed in September (76%) and lowest in March (29%). The total number of 'dose exacerbations', over the 12 month period pre- and post-initiation was reduced from 948 to 522. The seasonal pattern was different than for hospital admissions, with a relatively consistent reduction of dose exacerbations across the year. The season/month of initiation was not statistically different for response, hospital and dose exacerbation rates.

Conclusions Regardless of the timing of initiation, the response rate to omalizumab is consistent through the year, the biggest observable seasonal effect, was the diminishing of the seasonal peak of hospital exacerbations around early autumn.

Please refer to page A257 for declarations of interest in relation to abstract P19.

Abstract P19 Figure 1 Impact of omalizumab on hospital exacerbations. 2 point moving average is the average of the previous data point and the current data point.

P20

LATENT HELMINTH DISEASE AS A CAUSE OF EOSINOPHILIA IN RESPIRATORY PATIENTS

¹AM Singer, ²J Barrett, ¹FR Ali, ²J Lambourne, ¹PE Pfeffer. ¹North Central and East London Severe Asthma Service, St Bartholomew's Hospital, Barts Health NHS Trust, London, UK; ²Department of Microbiology, Barts Health NHS Trust, London, UK

10.1136/thoraxjnl-2017-210983.162

Introduction Within the UK, eosinophilia is most commonly associated with allergy and respiratory diseases such as asthma. Increasingly raised blood eosinophil counts are seen as a biomarker of heightened Th2 inflammation indicating a need to prescribe steroids in airways diseases. New treatments for severe Th2-high asthma aim to inhibit the eosinophilic pathway to reduce pathological inflammation. However by inhibiting this pathway we risk suppressing the body's natural defences against parasitic helminth disease – in patients with asymptomatic latent *Strongyloides stercoralis* there is the risk of catastrophic hyper-infection. Our particular patient cohort in East London is a diverse international community who travel frequently. We therefore sought to evaluate the prevalence of asymptomatic helminth disease in respiratory, and particularly asthma patients, within our Trust, which includes a severe asthma service where we prescribe biologics that inhibit the Th2 pathway.

Methods We prospectively tested eosinophilic patients reviewed in respiratory clinic for helminth infection using serological screening as part of a Service Evaluation. Inclusion criteria were an eosinophilia (≥ 0.3) or those being considered for