Background and Aims: Alcohol consumption in China has substantially increased over the last three decades and the number of patients with alcoholic liver disease (ALD) is rising at an alarming rate. However, accurate and representative data on time trends in its hospitalization rates are not available. The aim of this study is to assess the current status and burden of ALD in China by analyzing the data from a large tertiary referral hospital, Beijing 302 Hospital.

Methods: Data were retrospectively recorded from patients diagnosed as ALD in Beijing 302 Hospital from 2002 to 2013. The disease spectrum and biochemical parameters of each patient were collected.

Results: The patients with ALD accounted for 3.93% (7,422) of all patients (188,902) with liver diseases between 2002 and 2013. The number of patients hospitalized with ALD increased from 110 in 2002 to 1,672 in 2013. The ratio of patients hospitalized with ALD to all patients hospitalized with liver diseases was rising almost continuously and increased from 1.68% in 2002 to 4.53% in 2013. Most patients with ALD were male. Age distribution of ALD hospitalization showed that the highest rate was in 40- to 49-year-old group in subjects. Notably, the annual proportion of severe alcoholic hepatitis (SAH) increased 2.43 times from 2002 to 2013. We found the highest levels of MCV, the AST/AI ratio, TBL, INR, and ALP in SAH patients, while serum levels of HGB, ALB, and CHE were significantly decreased in SAH group. Among these ALD, the SAH patient population has the worst prognosis. Alcoholic cirrhosis (ALC) is the most common ALD, and annual admissions for ALC increased significantly during the analyzed period.

Conclusions: The number of hospitalized patients with ALD and the annual hospitalization rate of ALD were increasing continuously in Beijing 302 hospital from 2002 to 2013. More attention should be paid to develop population-based effective strategy to control ALD.

Disclosure of Interest: None Declared.
Background and Aims: Corticosteroids remain the most popular pharmacological therapy for SAH. However, there is wide variation in practice in the UK regarding length of treatment course and discontinuation rules reflecting the uncertainty of how to measure clinical corticosteroid response. There is a need to improve methods of patient stratification to enable accurate selection of patients who derive the most benefit from corticosteroids.

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FRI-303
Intermediate (CD14++CD16+) monocytes from patients with acute severe alcoholic hepatitis are activated and functionally similar to classical (CD14+CD16−) monocytes
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Background and Aims: Monocytes respond to pathogenic and inflammatory signals to coordinate the immune response. They are important in acute severe alcoholic hepatitis (AAH) in which infection often contributes to severe liver inflammation. Three phenotypically distinct monocyte subsets have been described: classical (CD14+CD16−), intermediate (CD14++CD16+) and non-classical (CD14−CD16+). Enrichment of intermediate monocytes has been demonstrated in many inflammatory conditions which we have previously confirmed in AAH. However, their functional phenotype has not been fully characterised. We aimed to investigate monocyte subset function in terms of phagocytosis, monocyte oxidative burst (MOB) and T cell interaction in patients with AAH in comparison to healthy controls (HC).

Methods: Peripheral blood mononuclear cells were isolated from 10 patients with AAH (recent onset jaundice in a heavy alcohol drinker; DF > 32; AST:ALT > 1.5) and 6 HCs. Monocyte subsets and memory CD4+ T cells were isolated by flow cytometry. MOB was determined by oxidation of dihydrorhodamine to rhodamine123 over 10 minutes which was quantified in each subset by flow cytometry. Phagocytosis was determined by cellular uptake of fluorescent latex beads. Memory CD4+ T cells were co-cultured for 5 days with each monocyte subset and T cell phenotype determined by flow cytometry.

Results: Intermediate monocytes are expanded in patients with AAH compared to HCs (24% v 9%; p < 0.001). Phagocytic capacity of classical and intermediate monocytes from patients with AAH was similar and significantly greater than HCs (62% v 44% for classical; p = 0.02 and 39% v 20% for intermediate; p < 0.001). MOB was similar between classical and intermediate monocytes from AAH patients (62% and 96% rhodamine123 positive respectively). Memory T cell proliferation was greater in co-cultures with intermediate monocytes from AAH patients than HCs (73% v 50%; p = 0.03) but proliferation, IL-17 and IFNγ expression were similar in co-cultures with each monocyte subset in patients with AAH.

Conclusions: Intermediate monocytes are functionally activated with higher capacity for phagocytosis and ability to drive T cell proliferation in AAH patients than in HCs. In patients with AAH classical and intermediate monocyte subsets have a similar functional phenotype. These in vitro surrogates of monocyte function suggest that in the context of severe inflammation the expanded pool of circulating intermediate monocytes is primed to clear pathogens and perpetuate the immune response.