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## Characteristics of immunocompromised patients with influenza A virus admitted to the intensive care unit

C. León Moya<sup>1</sup>, A. Gutiérrez Pizarraya<sup>1</sup>, M.L. Cantón Bulnes<sup>1</sup>, Á. Arenzana Seisdedos<sup>1</sup>, A. Rodríguez<sup>2</sup>, J. Garnacho-Montero<sup>1</sup>

<sup>1</sup>Hospital Universitario Virgen Macarena, Sevilla, Spain; <sup>2</sup>Hospital

Universitari Joan XXIII, Tarragona, Spain

Correspondence: C. León Moya

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**INTRODUCTION.** Information about immunocompromised patients infected with influenza A virus requiring admission to the ICU is lacking. The objective of this study was to know the clinical characteristics of these patients and to identify variables associated with mortality.

**METHODS.** A prospective multicenter observational cohort study was based on data from the GETGAG/SEMICYUC registry (2009–2015) collected by 148 Spanish ICUs. All patients admitted to the ICU with the diagnosis of influenza A virus infection were included. Immunosuppression was defined as any primary immunodeficiency or immunodeficiency secondary to human immunodeficiency virus (HIV) infection, active malignancy, immunodeficiency secondary to radiation treatment or use of cytotoxic drugs or corticosteroids (daily dose >40 mg of prednisolone or the equivalent for >2 weeks), immunological disease, solid organ transplant, and haematological disease. Factors associated with mortality in immunocompromised patients were assessed by logistic regression analysis.

**RESULTS.** Of 2315 patients with influenza A infection, 289 (12.5%) were classified as immunocompromised. Mortality was significantly higher in immunocompromised patients (47.1% vs 20.1%; p < 0.001). Severity of illness and the rates of health-care infection, early use of oseltamivir and administration of corticosteroids were significantly greater in immunocompromised subjects. In this group of patients, factor independently associated with mortality were: APACHE II score (OR = 1.09, 95% CI = 1.04-1.13, p < 0.001), stage 3 of acute renal insufficiency (OR = 2.33, 95% CI = 1.69-4.63, p < 0.05), use of vasopressor drugs (OR = 4.69, 95% CI = 2.44-9.03, p < 0.001) and the use of corticosteroid (OR = 1.89, 95% CI = 1.06-3.37, p < 0.05).

**CONCLUSION.** Immunocompromised individuals with influenza A admitted to the ICU have a poorer outcome than patients without immunocompromise. In this population, we should avoid the use of corticosteroids because it is independently associated with a higher mortality.

### Liver failure: Nutrition and metabolism

#### 1028

Development of a multivariate prediction model for mortality in a cohort of 1177 critically ill patients with hypoxic hepatitis

A. Van den broecke<sup>1</sup>, L. Van Coile<sup>1</sup>, A. Decruyenaere<sup>2</sup>, H. Van

Vlierberghe<sup>3</sup>, K. Colpaert<sup>4</sup>, J. Decruyenaere<sup>4</sup>

<sup>1</sup>Ghent University, Faculty of Medicine and Health Sciences, Ghent, Belgium; <sup>2</sup>Ghent University Hospital, Department of Internal Medicine, Ghent, Belgium; <sup>3</sup>Ghent University Hospital, Department of Hepatology and Gastroenterology, Ghent, Belgium; <sup>4</sup>Ghent University Hospital, Department of Intensive Care Medicine, Ghent, Belgium

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**INTRODUCTION.** Hypoxic hepatitis (HH) ("ischemic hepatitis" or "shock liver") is a common cause of hepatic dysfunction in an ICU, is histologically characterized by centrilobular liver cell necrosis and is caused by an insufficient oxygen delivery to hepatocytes. Typical for HH is the sudden, significant rise (>5x ULN) of aspartate aminotransferase (AST) in response to hemodynamic failure.

**OBJECTIVES.** The aim of this study was to identify mortality risk factors of HH using a multivariate prediction model and to assess the predictive accuracy of 2 existing models (Fuhrmann et al. [2] and Raurich et al. [3]).

**METHODS.** The study cohort consisted of 1177 adults with a first episode of HH (AST >5x ULN) out of 29874 patients admitted to the

ICU of the Ghent University Hospital (UZG) between January 1<sup>st</sup>, 2007 and September 21st, 2015. The incidence of HH was 4.2% and the hospital mortality 42.8%. Further detailed study results are described in another report [1]. 29 parameters were retrospectively collected as potential risk factors based on clinical importance. The association with mortality was studied using logistic regression at the time of maximal AST value. Predictive accuracy of the models was assessed by the area under the receiver operating characteristic curve (AUC), the calibration curve and the Hosmer-Lemeshow (HL) test. As the 2 existing models were developed in patients with AST >20x ULN, only this subgroup (n = 460) was used for their validation. RESULTS. Resp. 8 and 2 risk factors are excluded based on missing values and correlation. The remaining 19 variables have 1153 complete cases. After a backward elimination procedure, the multivariate UZG model indicates that age (OR 1.03 per year), cardiac failure (OR 8.55), septic shock (OR 12.25), hypovolemic shock (OR 7.99), lung embolism (OR 14.04), acute respiratory failure (OR 2.38), acute-on-chronic respiratory failure (OR 6.68) and mechanical ventilation (OR 2.24) are most significantly (all P < 0.001) associated with mortality. Additionally, INR (OR 1.27 per unit, P = 0.002), hyperthermia (OR 14.58, P = 0.019) and AST (OR 1.01 per 100U/L, P = 0.05) are significantly associated. pH (OR 0.56 per 0.1) and trombocytes (OR 0.98 per  $10^4$ /ml) are inversely associated (both P < 0.001). The UZG model has a good discrimination (AUC 0.79) and shows a good fit between the observed and expected mortality (HL: P = 0.67). On the contrary, the models of Raurich et al. and Fuhrmann et al. have a weak discriminative capacity (AUC 0.59 and 0.67, resp.).

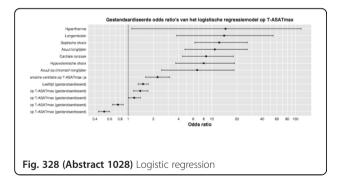
**CONCLUSIONS.** This is by far the largest single-centre cohort study of HH described in literature. 7 mortality risk factors associated with HH are identified. Unlike the 2 existing models, the UZG model has a good discrimination and calibration.

## REFERENCES

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#### 1029

# Epidemiology, cause, evolution and outcome in a cohort of 1177 critically ill patients with hypoxic hepatitis

L. Van Coile<sup>1</sup>, A. Van den broecke<sup>1</sup>, A. Decruyenaere<sup>2</sup>, H. Van Vlierberghe<sup>3</sup>, K. Colpaert<sup>4</sup>, J. Decruyenaere<sup>4</sup>

<sup>1</sup>Ghent University, Faculty of Medicine and Health Sciences, Ghent, Belgium; <sup>2</sup>Ghent University Hospital, Department of Internal Medicine, Ghent, Belgium; <sup>3</sup>Ghent University Hospital, Department of Hepatology and Gastroenterology, Ghent, Belgium; <sup>4</sup>Ghent University Hospital, Department of Intensive Care Medicine, Ghent, Belgium

Correspondence: L. Van Coile Intensive Care Medicine Experimental 2017, 5(Suppl 2):1029

**INTRODUCTION.** Hypoxic hepatitis (HH) ("ischemic hepatitis" or "shock liver") is histologically characterized by centrilobular liver cell necrosis and is caused by an insufficient oxygen delivery to

Correspondence: A. Van den broecke

hepatocytes. Typical for this type of liver cell necrosis is the sudden, significant rise (>5x ULN) of aspartate aminotransferase (AST) in response to cardiac, circulatory or respiratory failure. Other causes of AST rise should be excluded before diagnosing HH. Treatment of HH relies on the prompt correction of the underlying dysfunction.

**OBJECTIVES.** The aim of this study is to describe the epidemiology, cause, evolution and outcome of critically ill patients with HH. More insight may raise more awareness and could result in a faster diagnosis, which may contribute to a better prognosis.

**METHODS.** The screened population consists of all adults (n = 29874) admitted to the ICU at the Ghent University Hospital between January 1<sup>st</sup>, 2007 and September 21<sup>st</sup>, 2015. 4012 patients had at least one episode of AST-levels above 5x ULN. After exclusion of 2835 patients with another cause of elevated AST-levels (e.g. liver surgery, toxic hepatitis, cholangitis,...), a cohort of 1177 first episodes of patients with HH were identified. These patients are classified by underlying cause of HH. The most important outcome variable is ICU and hospital mortality.

RESULTS. The incidence of HH is 4.2%. The male/female ratio is 1.5 and the median age is 66 years. The causes of HH are cardiac failure (40.2%), septic shock (28.3%), post-anesthesia without overt evidence of an acute cardiac or respiratory event (11.6%), hypovolemic shock (8.9%), acute respiratory failure (6.0%), acute-on-chronic respiratory failure (3.1%), pulmonary embolism (1.4%) and hyperthermia (0.5%). The hospital mortality associated with HH is 42.8%, of which 90.4% occurred during ICU stay. Pulmonary embolism and septic shock have the highest (56.2% and 52.9%, resp.) and fastest (median survival 5.4 and 12.3 days, resp.) mortality. Contrary to previous studies, patients with a limited rise of AST (5xULN < AST < 10xULN) were also included. This subgroup contains 439 patients and has a mortality of 26.2%. Another subgroup of 136 patients appeared to develop HH after major surgery with general anesthesia despite having no problem during surgery. This subgroup, with still a considerable mortality of 7.4%, was not described in previous studies.

**CONCLUSIONS.** This is by far the largest single-centre cohort study of HH described in literature. With an incidence of 4.2%, HH is a frequent cause of hepatic dysfunction in critically ill patients and is associated with a high hospital mortality of 42.8%. The principal causes are acute cardiac disease and septic shock, which include more than 2/3 of all episodes. Clinicians should search actively for an undetected underlying hemodynamic or respiratory problem even in patients with moderately elevated AST values or with AST elevations after major uncomplicated surgery.

### 1030

## Thromboelastometry - relation to the severity of liver cirrhosis in patients considered for liver transplantation

G. Dumitrescu<sup>1</sup>, A. Januszkiewicz<sup>1</sup>, A. Ågren<sup>2</sup>, M. Magnusson<sup>3</sup>, S. Wahlin<sup>4</sup>, J. Wernerman<sup>1</sup>

<sup>1</sup>Karolinska Institutet, Anestesia and Intensive Care, Stockholm, Sweden; <sup>2</sup>Karolinska Institutet, Department of Medicine, Division of Hematology, Coagulation Unit, Stockholm, Sweden; <sup>3</sup>Karolinska Institutet, CLINTEC, MMK, Clinical Chemistry and Blood Coagulation Research, Stockholm, Sweden; <sup>4</sup>Karolinska Institutet, Division of Hepatology, Centre for Digestive Diseases, Stockholm, Sweden

**Correspondence:** G. Dumitrescu

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**INTRODUCTION.** The severity of liver disease is assessed by scoring systems which include the conventional coagulation test PT-INR. However PT-INR is not predictive of bleeding in liver disease and thromboelastometry has been suggested to give a better overview of the coagulation system in these patients. It has now been suggested that coagulation as reflected by tromboelastomety, particularly maximum clot firmness (MCF), may also be used for prognostic purposes in patients with stable chronic liver disease.

**OBJECTIVES.** The objective of our study was to investigate if thrombelastometry may discriminate the degree of liver insufficiency

according to the scoring systems Child Pugh and Model for Endstage Liver Disease (MELD).

**METHODS.** Forty adult patients with stable chronic liver disease of different etiologies and stages were included in this observational cross-sectional study. The severity of liver disease was evaluated using the Child-Pugh score and the MELD score, and blood samples for biochemistry, conventional coagulation tests and thromboelastometry were collected at the time of the final assessment for liver transplantation. Statistical comparisons for the studied parameters with scores of severity were made using Spearman's correlation test and receiver operating characteristic (ROC) curves.

	Child-Pugh score A + B	Child-Pugh score C
No.	22	18
Child-Pugh score	8(6–9)	11(10–13)
	MELD ≤16	MELD >17
No.	21	19
MELD score	13(7–16)	20(17–27)

[Number of patients (No). Scores as median (range).]

**RESULTS.** Spearman's correlation coefficients indicated that the thromboelastometric parameters did not correlate with Child-Pugh or MELD scores. The ROC curves of the thromboelastometric parameters could not differentiate advanced stages from early stages of liver cirrhosis.

**CONCLUSIONS.** standard thromboelastometry cannot discriminate the stage of chronic liver disease in patients with severe chronic liver disease.

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