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Incidence of early burn-induced effects on liver function as reflected by the plasma disappearance rate of indocyanine green: A prospective descriptive cohort study

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ABSTRACT

Organ dysfunction and failure are important for burned patients as they increase morbidity and mortality. Recent evidence has suggested that organ injuries are occurring earlier after burns, and are more common than previously thought. In this study we have assessed the extent to which liver function, assessed by the plasma disappearance rate of indocyanine green (PDR_{ICG}), is affected in patients with severe burns. This is a prospective, descriptive exploratory study at a national burn centre. Consecutive adult patients with a percent total body surface area burned (TBSA%) of 20% or more, were examined prospectively by dynamic (PDR_{ICG}) and static liver function tests (plasma: bilirubin concentration, prothrombin complex, and alanine aminotransferase and alkaline phosphatase activities). Early liver dysfunction was common, as it is assessed by both dynamic (7 of 17) and static liver function tests (6–17 of 17). A regression model showed that changes in PDR_{ICG} were associated with age, TBSA%, plasma bilirubin concentration, plasma C-reactive protein concentration, and cardiac index. Persistent and advanced hepatic dysfunction was associated with mortality. The PDR_{ICG} seems to give a comprehensive assessment of liver function after major burns. Hepatic dysfunction seems to be as common as dysfunction in other organs. We interpret the recorded effects on liver function as part of a multiple organ dysfunction syndrome, primarily induced by the burn itself. However, this needs to be further investigated.

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1. Introduction

The liver plays a central role after burns, both in the host's defence response and as a target for remote organ dysfunction in systemic inflammation. It is not surprising, therefore, that patients with pre-existing liver disease have an almost doubled risk of mortality compared with burned patients with similar histories, injuries, and medical profiles [1].

Thermal injury can cause liver damage by several mechanisms (hypoperfusion, proinflammatory cytokines, or other signals of cell death, formation of oedema, and fatty changes). Yet, the incidence of liver dysfunction after burns seems to be low, and it does not qualify on the list of top 10 clinically most relevant complications from the American Burn Association database [2]. A few studies have reported the results of serum analyses that have assessed static liver dysfunction among adult burned patients. In a group with 30% TBSA and 15% mortality, hepatic dysfunction was detected in 3.5% [3].

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Another study showed a 36% incidence of liver failure, the reported onset of which was in the second week (median day 9) after a lethal burn injury [4]. In a recent paper, enlargement of the liver and impaired protein synthesis have been shown to develop early among children with major burns, and the extent of that dysfunction correlated with the size of the burn. The activities of hepatic enzymes (serum aspartate aminotransferase (AST), and alanine aminotransferase (ALT)) were increased two-fourfold immediately after the burn, and took four to six weeks to return to the reference range, which suggested burn-induced liver damage [5].

1.1. *Assessment of liver function*

Traditionally assessment of liver function is based on static laboratory tests that provide indirect measures of hepatic function, while dynamic tests such as the plasma disappearance rate of indocyanine green (PDR_{ICG}), provide measures of the actual functional state of the liver at the time of the measurement [6]. PDR_{ICG} has been shown to be a good predictor of survival in critically ill patients [7–10], and the method has been validated in several studies. Invasive PDR_{ICG} reflects clearance of ICG from the blood of critically ill patients [11]. Non-invasive PDR_{ICG} has been compared with invasive PDR_{ICG} in critically ill patients during fluid loading and infusion of dobutamine [12], and in recipients of liver transplants [13], but has not previously been used in studies on patients with burns.

The aim of the present study was to assess the early (<two weeks after injury) burn-induced effects on dynamic (PDR_{ICG}) and static (plasma bilirubin concentration, activities of AST, ALT, and alkaline phosphatase (AP), and prothrombin complex) markers of liver function, and relate these changes to important burn indices and dysfunction in other organs. We set up the following hypotheses: first, liver dysfunction develops soon after a severe burn. Secondly, the non-invasive PDR_{ICG} method can capture changes in liver function that may be caused by the specific aspects of a burn injury, such as TBSA% and perhaps inhalation injury. It also adds new information as compared to conventional liver function tests regarding burn induced liver effects.

2. Patients and methods

2.1. *Design, and selection of patients*

During a four-year period (2006–2009) consecutive adult patients (18 years and over) admitted to a national burn centre (the Linköping University Hospital Burn Unit) with thermal burns involving 20% or more of the total body surface area (TBSA%) were included in this prospective explorative cohort study.

Criteria for exclusion were: allergy to ICG or iodine; pregnancy; superficial burns that did not require arterial and central intravenous lines or central haemodynamic monitoring; and patients who died within the first 2 days.

2.2. *Measurements*

PDR_{ICG} as an indicator of liver function was measured using the non-invasive liver function monitoring system (LiMON, Pulsion Medical Systems, Munich, Germany), on days 1, 2, 3, 5, 7, 9, and 14 after injury. A dose of ICG 0.5 mg/kg body weight was given through a central venous line, and measured by a transcutaneous sensor (Sensor PV50100) on a finger or toe. Reference values used for PDR_{ICG} were 18–25%/min [13]. We chose to group the patients according to how many of their measurements of PDR_{ICG} were below the lower reference value (18%/min). ''No liver dysfunction'' indicated patients with none or only one PDR_{ICG} value below the reference, whereas ''liver dysfunction'' indicated patients with two or more PDR_{ICG} values below the reference range.

Measurements of central haemodynamics and extravascular lung water by transpulmonary thermodilution (PiCCO, Pulsion Medical Systems, Munich) were made at the same time as liver function was measured. Standard laboratory tests were drawn from an arterial line on the same days, and analysed in the University Hospital laboratory. The lowest PaO_2 indicated which PaO_2/FiO_2 value to list as the worst daily value, and the lowest arterial pH value indicated which base excess value to use. The highest value each day was chosen for expiratory minute volume, and it is presented as ml/kg predicted body weight [14].

Details of vital signs, nutrition, and organ dysfunction, including sepsis, were recorded separately. Information about previous medical conditions was extracted from medical charts, and clinical data such as TBSA% and age were recorded in the prospectively maintained local burn registry [15]. The diagnosis of inhalation injury was verified by bronchoscopy. Sepsis was classified according to the international intensive care definition [16]. The Sequential Organ Failure Assessment (SOFA) score was used to assess organ dysfunction [17]. The neurological part of SOFA was left out because of the difficulties in assessing the Glasgow coma scale in sedated patients. For this study multiple organ failure was defined as 3–4 score points in 2 or more organ dimensions of the SOFA score [18]. Organ dysfunction was defined as a SOFA dimension score point of over zero. The definition used for enteral dysfunction was ''inability to continue enteral feeding for more than 24 h because of enteral feeding intolerance (persistent residual)''. It was derived from one of the criteria for sepsis from the American Burn Association consensus conference to define sepsis and infection [19]. Acute respiratory distress syndrome (ARDS) was graded according to the lung injury score by which scores that exceed 2.5 indicate a severe lung injury [20].

The regional ethics committee (the Regional Ethical Review Board in Linköping) waived the need for their approval for descriptive and explorative studies that not include procedures that not are considered as ordinary burn care.

2.3. *Burn care*

Patients were treated in a protocol based way according to our usual regimen [15], including early excision and grafting, standard ventilation, fluid management, early enteral nutrition, and laboratory assessment according to a preset protocol. Different aspects of this have previously been described [21–24]. Ringer's acetate was used for fluid resuscitation in volumes according to the Parkland formula (4 ml/kg body weight (BW) × TBSA%), with adjustments for individual

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