Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

CORRESPONDENCE

RETROSPECTIVE ANALYSIS OF LIVER FUNCTION DERANGEMENT IN SEVERE ACUTE RESPIRATORY SYNDROME

To the Editor:

In March 2003, a hospital outbreak of severe acute respiratory syndrome (SARS) caused by a novel coronavirus (SARS-coronavirus, SARS-CoV) was reported in Hong Kong (1–4) and cases were subsequently reported globally (5). In this study, we investigated the pattern of hepatic damage as well as the effect of chronic hepatitis B on the clinical outcome of SARS.

This was a retrospective analysis of the patient cohort as reported previously (1). Lamivudine 100 mg daily was prescribed to all chronic hepatitis B patients. Oxygen therapy was given when patients' oxygen saturation fell below 90%. Mechanical ventilation was offered when patients could not achieve 90% oxygen saturation while on 5 L/min oxygen supplement.

Anticoronavirus immunoglobulin (Ig) G antibody was measured by immunoflourescence assay in paired sera samples. Histologic and electron-microscopic examination was performed on the autopsy liver specimens. Liver tissue samples were inoculated onto African green monkey kidney (Vero) monolayers for viral isolation. Reverse transcriptase polymerase chain reaction (RT-PCR) was performed to confirm the presence of SARS-CoV in positive cell cultures (6).

Categorical variables were compared using the chi-squared test and continuous variables using the Mann-Whitney U test. All tests were 2-tailed. Statistical significance was taken as $P < 0.05$.

One hundred and eighteen patients who had hepatitis B surface antigen (HBsAg) results available were studied. Paired serum samples for SARS-CoV serology were available in 113 (96%) patients and all were positive. The baseline characteristics and clinical outcomes of patients are shown in the Table.

Two of the 12 chronic hepatitis B patients had liver cirrhosis. Among the patients without cirrhosis, 3 were admitted for chronic hepatitis B flare-up and contracted SARS nosocomially; on admission, 5 patients had elevated alanine aminotransferase levels and 2 patients had normal levels. One patient died of respiratory failure related to SARS and another died of multiorgan failure. Among the survivors, the alanine aminotransferase levels in 6 patients had returned to normal and in 3 other patients had decreased on follow-up.

Table. Baseline characteristics and clinical outcomes of HBsAg-positive versus HBsAg-negative patients

Baseline Characteristics	HBsAg positive (n = 12)	HBsAg negative (n = 106)	Overall (n = 118)	*P* Value
	Number (%) or (Range)			
Age (years)	38 (21–64)	33 (20–83)	33 (20–83)	0.62
Male sex	9 (75)	46 (43)	55 (47)	0.080
Comorbid illness	2 (16.7)	14 (13.2)	16 (13.6)	1.00
Fever before admission (days)	2.5 (0–4)	3 (0–11)	3 (0–11)	0.099
Hemoglobin level (g/dL)	13.9 (10.3–15.4)	13.5 (6.3–16.6)	13.5 (6.3–16.6)	0.45
Platelet count ($\times 10^3/\mu L$)	140 (31–207)	151.5 (14–359)	150 (14–359)	0.54
White blood cell count ($\times 10^3/\mu L$)	5.4 (2.8–7.3)	6.7 (1.0–10.9)	4.7 (1.0–10.9)	0.37
Lymphocyte ($\times 10^3/\mu L$)	0.8 (0.3–1.3)	0.8 (0.2–6.5)	0.8 (0.2–6.5)	0.72
PT (sec)	11.21 (9.8–17.1)	10.4 (9.0–60.1)	10.5 (9.0–60.1)	0.03
Creatinine (μmol/L)	91 (67–117)	84 (56–1318)	85 (56–1318)	0.26
TB (μmol/L)	8.5 (3–163)	6 (2–266)	6 (2–266)	0.14
TB >50 (μmol/L)	2 (17)	1 (1)	3 (3)	0.021
ALT (IU/L)	106.5 (23–1513)	24.5 (9–199)	25.5 (9–1513)	<0.001
↑ ALT	9 (75)	16 (15)	25 (21)	<0.001
Outcomes				
Peak PT (sec)	11.5 (10.5–22.8)	11.1 (9.6–60.1)	11.2 (9.6–60.1)	0.19
Peak TB (mg/dL)	2.0 (1.1–13.5)	1.2 (0.4–22.6)	1.2 (0.4–22.6)	0.008
Peak TB >3.0 (mg/dL)	3 (25)	10 (9)	13 (11)	0.26
Peak ALT (IU/L)	152.5 (35–1513)	107 (19–2940)	116 (19–2940)	0.40
↑ Peak ALT (IU/L)	9 (75)	80 (76)	89 (75)	1.00
Oxygen supplement	8 (67)	59 (56)	55 (47)	0.67
Mechanical ventilation	3 (25)	13 (22)	16 (14)	0.44
Liver failure	1 (8)	0 (0)	1 (0.8)	0.19
Death	2 (17)	7 (7)	5 (8)	0.50

ALT = alanine aminotransferase, HBsAg = hepatitis B surface antigen, PT = prothrombin time, TB = total bilirubin

0002-9343/04/$–see front matter

Among the 106 HBsAg-negative patients, 16 (15%) patients had elevated alanine aminotransferase levels on admission. None of these patients had comorbid illnesses or had taken any medications. Seventy-one percent of patients who had normal baseline levels experienced elevation of alanine aminotransferase during the course of their illness. Clinical outcomes including oxygen requirement, need for intubation, and case fatality rate were not different in patients with normal and elevated baseline alanine aminotransferase levels. No patient developed liver failure. Overall, 66 of 80 (83%) patients who experienced alanine aminotransferase elevation had normalization of alanine aminotransferase levels on discharge and another 10 (13%) patients had decreasing levels on follow-up.

Autopsy was performed in 4 patients, including the 2 chronic hepatitis B patients who died. Specific hepatic lesion, acute hepatitic changes, or hepatic necrosis were not observed. Coronavirus particles were not detected by electromicroscopy. Positive cell culture for SARS-CoV confirmed by RT-PCR was found in 1 patient who had normal alanine aminotransferase levels throughout the admission.

In this study, three-quarters of patients experienced alanine aminotransferase elevation while the levels of the majority had returned normal after recovery. The liver derangement in SARS was likely a nonspecific transient reaction caused by the infection rather than by a direct cytopathic effect of SARS-CoV. The clinical outcomes of chronic hepatitis B patients were not different from those of HBsAg-negative patients, in contrast to the outcomes in a previous report (7). The absence of specific hepatic lesions in hepatitis B virus and SARS-CoV coinfected patients on autopsy supports the lack of influence that SARS has on chronic hepatitis B infection.

In conclusion, we found that chronic hepatitis B virus infection did not influence the clinical outcomes of SARS. Most SARS patients had transient elevation of liver enzyme levels that did not have serious clinical consequences.

Henry L. Y. Chan, MD
Wai-Keung Leung, MD
Ka-Fai To, MBChB
Paul K. S. Chan, MD
Nelson Lee, MBBS
Alan Wu, MBChB
John S. L. Tam, PhD
Joseph J. Y. Sung, MD, PhD
The Chinese University of Hong Kong
Hong Kong, SAR China

1. Lee N, Hui D, Wu A, et al. A major outbreak of severe acute respiratory syndrome in Hong Kong. *N Engl J Med.* 2003;348:1986–1994.
2. Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome. *N Engl J Med.* 2003;348:1953–1966.
3. Drosten C, Gunther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. *N Engl J Med.* 2003;348:1967–1976.
4. Peiris JSM, Lai ST, Poon LLM, et al. Cornoavirus as a possible cause of severe acute respiratory syndrome. *Lancet.* 2003;361:1319–1325.
5. Booth CM, Matukas LM, Tonlinson GA, et al. Clinical features and short-term outcomes of 144 patients with SARS in the greater Toronto area. *JAMA.* 2003;289:2801–2809.
6. World Health Organization. PCR primers for SARS developed by WHO Network Laboratories. Available at: http://www.who.int/csr/sars/primers/en/. Accessed May 5, 2003.
7. Peiris JSM, Chu CM, Cheng VCC, et al. Clinical progression and viral load in a community outbreak of coronavirus-associated SARS pneumonia: a prospective study. *Lancet.* 2003;361:1767–1772.

DESCENDING AORTIC THROMBOSIS AND CEREBRAL INFARCTION AFTER MASSIVE WASP STINGS

To the Editor:

The wasp is the third most common venomous animal responsible for human fatalities in the United States (1). The clinical manifestations of wasp sting can be divided into three groups: local reactions, such as pain, wheal, edema, and swelling, that are generally self-limiting; immunological reactions usually leading to anaphylaxis with subsequent anaphylactic shock; and systemic toxic reactions caused by large doses of venom, resulting in hemolysis, coagulopathy, rhabdomyolysis, acute renal failure, and hepatotoxicity (2–6). We describe a case of massive *Vespa affinis* stings associated with acute descending aortic thrombosis and cerebral infarction, which are uncommon following wasp stings and therefore extend the clinical spectrum of wasp envenomation.

A 71-year-old woman without remarkable medical history was stung by a swarm of wasps. Initially, only itchy and painful sensations were noted on the venom-exposed sites. Twenty-four hours after envenomation, she complained of sudden onset, right-sided mouth angle deviation, and numbness and weakness of the left limbs followed by paraplegia. Computed tomographic (CT) scan of the brain showed normal results. Her blood pressure was 176/63 mm Hg. Physical examination revealed cold lower limbs, weak pulsation over bilateral dorsalis pedis arteries, generalized muscle tenderness, shallow left nasolabial fold, left hemiplegia, paraplegia, absent bilateral extensor plantar reflexes, and hyporeflexia over the lower limbs. There were an estimated 150 stings over her head, face, and limbs. Platelet count was $104 \times 10^3/\mu L$, prothrombin time was 0.89 international normalized ratio, and activated partial thromboplastin time was 24 seconds (control, 31 seconds). She was noted to have coagulopathy with an elevated D-dimer level of 1151 ng/dL (reference, $<$500 ng/dL), an elevated fibrinogen degradation product of 4 μg/mL (reference, $<$1 μg/mL), and a positive protamine sulfate time. Biochemical tests revealed the following values: blood