Lessons from Taiwan

During the recent outbreak of severe acute respiratory syndrome (SARS), several medical institutes in Taiwan were closed temporarily because medical staff developed SARS-like symptoms. To help prevent this situation from occurring at our hospital, we built an outdoor emergency department (Fig. 1). In designing the facility, we incorporated some principles from the management of other types of disasters, including "hot" and "cool" areas and a decontamination zone. In addition, the infection control team and emergency physicians implemented a new triage algorithm. No one with a fever, cough, relevant contact or travel history, or any other suspicious symptoms was allowed indoors. Several procedures were performed outdoors, such as chest radiography and the drawing of blood specimens. After complete evaluation and observation, patients who did not have SARS could be discharged directly or moved into the hospital after undergoing a decontamination procedure. Patients with suspected or probable SARS were admitted into an isolation ward via a specific route outside the emergency department. Infectious disease physicians were required to be on call 24 hours a day and were consulted in ambiguous cases. By virtue of the triage algorithm and the outdoor location, our emergency department continued to operate efficiently throughout the critical period, even when the other 2 emergency departments in this city of 3 million people were shut down.

Protecting health care workers has been our first priority in the fight against SARS. All staff at our hospital have been under a strict follow-up protocol, and no secondary or tertiary transmission has been discovered. Even though SARS spreads rapidly, has high infectivity and is associated with significant morbidity and mortality,[1] we believe that our outdoor emergency department was a factor in protecting the health care workers and in allowing the entire hospital to maintain its normal functions.

Fig. 1: Outdoor emergency department at Veterans General Hospital, Taiwan. T = triage area and body temperature screening station, C = cardiopulmonary resuscitation area, L = low-risk area for patients with fever or cough, H = high-risk area for suspect and probable cases of SARS.

Although this novel approach to emergency care has been effective and efficient, it has consumed considerable resources with a low yield of cases (less than 1% of patients seen actually had SARS). However, voluntary quarantine within a hospital also carries significant costs.[2]

Li-chien Chien
Wen-Bin Yeh
Hong-Tai Chang
Department of Emergency Medicine
Veterans General Hospital
Kaohsiung City, Taiwan

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SARS case-fatality rates

A *CMAJ* editorial[1] published in May 2003 cites the case-fatality rate for severe acute respiratory syndrome (SARS) in Canada as 12.4%. We have been examining death rates in Taiwan, Canada and Hong Kong[2-6] and have found significant variation over time (Fig. 1, next page).

In Taiwan the first case of SARS was reported on Mar. 14, 2003;[7] there were 78 cases by the end of April and 676 cases by the end of May.[6] The first death was reported on Apr. 27, at which time the case-fatality rate (the number of deaths divided by the sum of deaths and recoveries) was only 3.8%. However, in mid-May the case-fatality rate rose

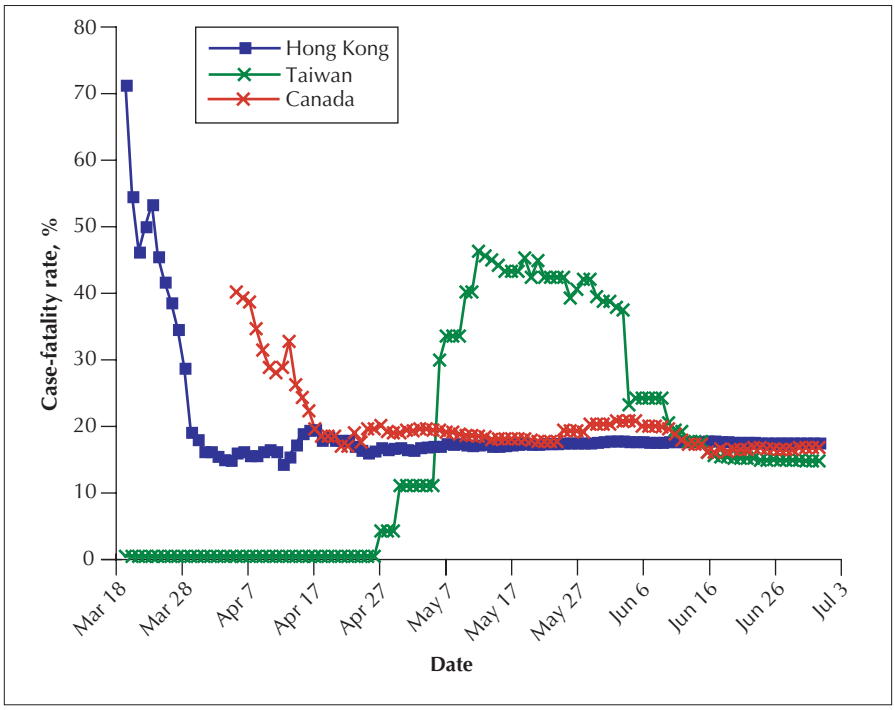

Fig. 1: Case-fatality rate for SARS in Taiwan, Canada and Hong Kong.[2-6]

markedly to a peak of about 45% and then stabilized at about 15% in June.

In Canada the first case of SARS was reported on Feb. 23 and the first 10 deaths were reported on Apr. 7. The case-fatality rate on that date was 38.5%; it fell to about 20% by the end of April and stabilized at about 17% in late June.

Finally, in Hong Kong case-fatality rates were very high (about 71%) early in the outbreak but later fell to about 17%.

According to recent data from Hong Kong,[8] hospital death rates among patients at least 60 years of age peaked sharply at about 12 days after admission, whereas younger patients showed maximal (and lower) peak death rates at about 18 days. Thus, to understand the differences in case-fatality rates over time and between countries it is important to take age into account. Another factor that might explain initially high rates is physicians' lack of clinical experience with the disease early in the outbreak. Published reports[9,10] suggest wide variation in therapy, which might also affect outcome. In addition, there has been considerable speculation that there may be different genetic forms of the virus (i.e., different degrees of virulence for the same virus) or that the virus has become less virulent over time.

Wing K. Fung
Philip L.H. Yu
Department of Statistics and Actuarial Science
The University of Hong Kong
Hong Kong, China

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Pediatric diabetic ketoacidosis

We read with interest the recent review of diagnosis and treatment of diabetic ketoacidosis by Jean-Louis Chiasson and associates.[1] However, it was unclear whether the therapeutic approach was being recommended for all patients, regardless of age.

We feel that diabetic ketoacidosis should be approached and treated differently in children. In particular, the risk of cerebral edema is significantly higher in children and adolescents: the reported incidence ranges from 0.7% to 3%, and this complication is associated with substantial morbidity (21% to 35%) and mortality (20% to 25%). Although the mechanism and risk factors remain controversial, it appears that the risk is higher among those presenting with new-onset diabetes,[2,3] with lower initial partial pressure of carbon dioxide and higher initial blood urea suggesting more severe acidosis and dehydration.[3] Possible aspects of treatment include rapid administration of hypotonic fluids[4,5] and use of bicarbonate.[2]

As a result of these factors, pediatric treatment protocols recommend more conservative fluid replacement.[6,7] Whereas Chiasson and associates[1] recommend starting with 15 to 20 mL/kg of isotonic saline, for children the recommendation is 5 to 10 mL/kg in the first hour, with higher rates used only in patients with significant hemodynamic compromise. Fluid replacement should be calculated over a 48-hour period. In addition, the use of bicarbonate is not routine for all pediatric patients with pH less than 7.0, and bicarbonate may in fact increase the risk of cerebral edema in children.[2] We agree with the recommendation not to give an intra-