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# SARS – Lessons from Two Singapore General Hospital Cases

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## ABSTRACT

**Two cases of Severe Acute Respiratory Syndrome (SARS) that occurred in Singapore General Hospital are described. The lessons learnt are outlined.**



**Keywords: Coronavirus, arteriovenous graft, leukopenia, thrombocytopenia, thrombolysis**

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## INTRODUCTION

In November 2002 a businessman from the city of Foshan in Guangdong might have been the first victim of a mysterious illness called Severe Acute Respiratory Syndrome (SARS).

The causative agent identified as a new coronavirus is transmitted by droplets and direct contact[1].

Healthcare workers are at high risk, accounting for about one quarter of all cases of SARS in Hong Kong including the Chief Executive of the hospital authority[2].

SARS has become a global health hazard and its high infectivity is alarming. More needs to be known about its transmission and studies of its behaviour are crucial to an understanding of this new disease. A rapid reliable diagnostic test will be of great importance in the future management of this disease especially in patients with co-morbidities with an atypical presentation.

## CASE REPORT

### Case 1

Mr T.K.C. was a 60-year old Chinese male patient. He was admitted to the Singapore General Hospital from the Accident & Emergency Dept on a trolley on 24 March 2003. He gave a history of a recent discharge from the Tan Tock Seng Hospital on 20 March 2003 for melaena and still had melaena for a few days. He however did not have haematemesis. He had felt giddy and fell backwards on the day of admission. There was a past history of Type II diabetes complicated by nephropathy and renal failure and also peripheral vascular disease. He had a left middle cerebral infarct and epilepsy previously.

In addition he had a non healing right heel ulcer for three months.

When first seen he had pallor, a slurred speech, atrial fibrillation and was mentally slow. Four days after admission he developed a spiking temperature reaching 40ºC. In addition he had a slight cough and decreased breath sounds in his chest. However the chest X-ray was clear.

In view of his gastrointestinal bleeding he had a number of investigations to elicit the source of bleeding. A gastroscopy showed antral gastritis. He was scheduled for a colonoscopy but this failed as the sigmoid was loaded with faeces. He was again prepared with PEG and laxatives, fleet enemas for a repeat scope, a barium enema and also was scheduled for a small bowel series and finally an abdominal CT scan which was ultimately cancelled. While in hospital he was visited by his wife.

Investigations revealed a Hb of 7.5 g/dl, WBC 8.99 x $10^9$/L, Lymphocytes 8.1%/L. The urea and creatinine were both elevated and he had hyponatraemia. The blood cultures grew a gram-ve bacilli, Escherichia coli. He also had pus cells in the urine.

The cause of the fever was attributed to the heel ulcer with a differential diagnosis of a urinary tract infection. He was a poor historian and was not able to give more information. He was transfused with two units of blood during this admission for anaemia.

He was given IV Augmention 1.2 g tds after admission and this was changed to IV Imipenem 250 g six hourly for his bacteraemia.

He was seen on the 28 March 2003 by the I.D. physician as he was febrile. However the CXR was clear and an initial decision to isolate him was deferred as the impression was unlikely to be SARS.

On 29 March 2003 he demonstrated shortness of breath and mild cough. He had debridment of the right heel in the emergency theatre under local anaesthesia. The following day he had debridment of the right heel again as the heel ulcer was sloughing and his temperature was 40ºC.

On 1 April 2003 because of his E coli and persistent fever he had an ultrasound of the kidneys and this


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showed a mass in the right kidney. He was sent for a CT abdomen but this was cancelled and he was returned to the ward.

He was febrile and clingling on to the cot sides. On 2 April 2003 he became abusive and started regulating his own intravenous drip.

On 3 April 2003 some shadowing was noted on the CXR. Blood at this point was sent for coronavirus by the I.D. physician. The patient's ulcer was still sloughing. By this time his urine culture was growing klebsiella and the right heel ulcer pseudomonas aeruginosa. The temperature was coming down, however he had right loin tenderness and was transferred to the isolation ward. The temperature settled on the 5 April 2003. He developed an episode of hypoglycaemia. He was finally transferred to Tan Tock Seng Hospital on 5 April 2003 where the initial diagnosis made was still non-SARS by the I.D. team. His diagnosis was finally confirmed. When a throat swab and blood for corona total antibody became positive.

**Case 2**

Mr KR a 63-year-old male.

The abovenamed first presented at SGH with vomiting on 27 July 1998 as he was found by his general practitioner to have renal impairment with hypertension. He had hypertension for eight years and had renal impairment since 1991 with anaemia but defaulted follow up. An ultrasound during this admission showed chronic renal parenchymal disease.

In December 1999 he had sepsis from a right leg cellulitis. He had depression previously. He had a left arteriovenous graft created earlier for renal failure and he underwent a revision of his left upper arm graft on 17 January 2003. On 28 January 2003 he had a left AVG thrombosis requiring thrombolysis. He was also investigated for ischaemic heart disease and had a non ST elevation MI with fluid overload.

In early March 2003 he had inflammation over the AV graft site and proteus mirabilis was grown. He was admitted on 16 March 2003 with a history of fever since the morning associated with chills and rigors. There were no other symptoms. He was on haemodialysis via a right internal jugular vein and was noted on examination to be shivering and the temperature was 38.6ºC. Some yellow pus was noted over the entry site of the right internal jugular line. The CXR was unremarkable. The cause of the fever was attributed to sepsis at the right internal jugular or left AV graft site. The patient was started on IV gentamicin and vancomycin and the right internal jugular line was removed. The lungs were clear clinically. Blood culture grew MRSA. On 16 March 2003 the CXR suggested pulmonary congestion. On 25 March 2003 a left internal jugular vein catheter was inserted (non tunneled) for dialysis.

On 29 March 2003 he was prepared for right AV graft but became breathless with wheezing. He was nebulised with Ventolin after which he became more comfortable. On 31 March 2003 he developed pleuritic pain and became acutely breathless the following day. Also the jugular site was infected and the fever was attributed to this.

On 5 April 2003, he had intermittent fever and by now had a productive cough for 11 days. He was febrile but otherwise well.

On 6 April 2003 he was to be transferred to Tan Tock Seng Hospital for possible SARS. However the patient was very breathless and this was the 3rd episode in the week that he was in pulmonary oedema. He was nebulised and given IV diuretics.

On 7 April 2003 he was noted to be leukopenic and thrombocytopenic. Prior to transfer to Tan Tock Seng Hospital he developed acute onset of breathlessness and was intubated in the Neurointensive Care Unit.

The post intubation chest X-ray showed bilateral infiltrates more infective than cardiogenic in nature.

He was isolated and identified as a possible SARS contact based on the low grade fever and being with a cohort of patients who had been exposed to an earlier SARS patient.

There was marked chest X-ray deterioration. He needed to be transferred to Tan Tock Seng Hospital. However Tan Tock Seng Hospital was tight on ICU beds and the plan was to extubate and transfer to Tan Tock Seng Hospital when he was stable. His line tip grew Acinetobacter and Klebsiella. He was then transferred to isolation. His Hb was 7.9 gm/dl and platelets 94,000 and Lymphocytes 10.6% L. On 12 April 2003 the patient was breathless, sweaty and restless. Blood culture grew a gram positive cocci stomatococus mucilaginosus and coagulate negative staphylococus. He had pulmonary oedema and the case was prepared for transfer to Tan Tock Seng Hospital.

On 13 April 2003 a decision was made to cancel transfer because the patient became unstable. He was started on IV meropenem and was on assisted ventilation.

He was finally transferred on 14 April 2003. Virus isolation for SARS cononavirus and blood for corona total antibody became positive.

## DISCUSSION

The two cases illustrate interesting points. They typify the type of patients we see often in the wards. The diagnosis of SARS is difficult if there are co-morbidities and the manifestations protean. However, on close scrutiny of their histories the important symptoms are

present e.g. fever etc. What is most important is a contact history and sufficient weightage for this must be given and SARS must enter the principle diagnosis until proven otherwise by relevant investigations. If these patients are not transferred to the appropriate hospital for investigations and management then they must be isolated until investigations are complete to prove or disapprove the diagnosis[3]. It must be realised that haematological and biochemical changes and X-ray changes may be late in the course of the disease and waiting for all manifestations to develop before making the diagnosis could be dangerous. Fever was present in both patients inspite of their co-morbidities. There were signs of sepsis and both patients had positive blood culture confirming the presence of bacteraemia. What needs to be appreciated is that two infective conditions could co-exist in the same patients as in the Hong Kong experience[4].

The fairly large number of HCWs who were exposed in the case of these two patients could be explained in a number of ways. Firstly they were ill patients and were sent on a number of occasions for specialised investigations and in some instances these had to be repeated because the patient were not initially properly prepared necessitating repeat visits to the departments concerned. There were also several visits to the radiology department.

Which brings me to the next point that these ill patients should have their investigations planned in a systematic manner and completed quickly. This is to minimise exposure of HCWs. We see patient Case I manipulating his own drip and pulling on the cot sides. It is not surprising that contact could be an important mode of spread to HCWs. His frequent bowel preparations must be another area of concern for possible exposure to HCWs[5]. In Case 2 his breathlessness was treated by nebulisation. There are reports that this may aggravate the spread of the disease by droplet infections[6]. Admission of the patients into the general ward together with administration of bronchodilator using a jet nebuliser was associated with infection of a large number of staff, patients and visitors. Early identification of infected cases, proper isolation and meticulous infection control measures are all very important steps in controlling the spread of this infection. A sensitive and specific rapid diagnostic test is very much needed for the early confirmation of the disease[7].

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