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International Journal of Cardiology 100 (2005) 167–169

International Journal of Cardiology

www.elsevier.com/locate/ijcard

Letter to the Editor

Tachycardia amongst subjects recovering from severe acute respiratory syndrome (SARS)

Suet-Ting Lau[a,*], Wai-Cho Yu[a], Ngai-Shing Mok[a], Ping-Tim Tsui[a], Wing-Lok Tong[a], Stella,W.C. Cheng[b]

[a]*Department of Medicine and Geriatrics, Princess Margaret Hospital, Lai Chi Kok, Kowloon, Hong Kong Special Administrative Region, China*
[b]*Occupational Therapy Department, Princess Margaret Hospital, Hong Kong Special Administrative Region, China*

Received 12 April 2004; accepted 19 June 2004

Abstract

SARS is a new infection in human. Patients recovering from SARS had palpitation in the form of sinus tachycardia. This study to identify the possible causes for the tachycardia excluded active disease, thyroid dysfunction, haematological, cardiac, autonomic and significant pulmonary defect at 2 months from onset of disease. The symptomatology was attributed to physical deconditioning and anxiety state. Physical and psychological fitness should be restored with rehabilitation.

Keywords: Severe acute respiratory syndrome; Tachycardia

SARS is a new infection in humans caused by a novel coronavirus [1] with unknown medium- or long-term complications. Hong Kong reported a total of 1755 cases and 299 deaths. Palpitation in the form of tachycardia at rest and becoming more marked during mild exertion was noted amongst patients recovering from SARS. Possible causes are deconditioning [2], impaired pulmonary function, impaired cardiac function, cardiac arrhythmia, thyroid dysfunction, anaemia, autonomic dysfunction [3] and anxiety state. This prospective cross-sectional cohort study was conducted to assess the extent of tachycardia and identify possible cause for it.

Fifteen consecutive patients with resting heart rate of more than 90 beats per min (BPM) were recruited from the initial 100 patients who underwent lung function testing at about 2 months from onset of illness. All patients had at least 4-fold rise in SARS-coronavirus antibody titre. The resting 12-lead ECGs showed a sinus heart rate ranging from 90 to 109 BPM. Holter monitoring results are listed in Table 1. Overall heart rate faster than 100 BPM was observed during the daytime (9am–9pm) but not at night. No other arrhythmia was detected. Heart rate variability using the time domain analysis showed that the standard deviation of all normal RR interval (SDNN) was normal (>100 ms in patients <60 years of age and 97 ms in the 62-year-old patient) (Table 1). Signal-average ECG was within normal in all subjects. Echocardiography revealed no abnormality apart from the 62-year-old patient who had mild diastolic dysfunction.

Only one patient had a moderate restrictive pulmonary function defect (Table 1). Chest radiography findings, haemoglobin level, length of hospital stay, time elapsed after discharge, presence of complications, WHO Quality of Life (QOL) score and Monitored Functional Task Evaluation (MFTE) score [4] are shown in Table 2.

The normal CBP, ESR, LFT, LDH, CK, CRP, as well as results of clinical assessment, suggested that ongoing active disease is unlikely. Normal thyroid function tests excluded thyrotoxicosis. Although coronavirus infection had been demonstrated to cause an autoimmune myocar-

* Corresponding author. Tel.: +86 852 29903581; fax: +86 852 29903148.
E-mail address: laust1@ha.org.hk (S.-T. Lau).

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doi:10.1016/j.ijcard.2004.06.022

Table 1
Holter monitoring and pulmonary function test

	Sex	Age	Min HR	Max HR	Mean HR	HR variability SDNN (ms, *N*>100ms)	FEV1 % predicted	TLC % predicted	DLCO/VA % predicted	PFT interpretation
1	M	25	50	141	87	133	97	97	103	Normal
2	F	30	46	154	77	181	107	102	127	Normal
3	F	25	47	153	84	167	71	78	120	Restrictive mild
4	M	40	52	136	82	131	86	101	92	Normal
5	F	34	61	152	85	104	78	90	128	Restrictive mild
6	F	28	53	145	91	123	52	78	95	Restrictive moderate
7	M	33	50	137	87	118	79	85	79	Restrictive mild
8	M	49	51	142	83	137	120	121	128	Normal
9	F	26	49	129	77	135	84	114	123	Normal
10	F	39	51	116	79	103	109	121	97	Normal
11	M	62	53	114	70	97	98	123	88	Normal
12	M	36	57	123	83	111	112	91	110	Normal
13	F	25	55	163	85	147	107	102	NA	Normal
14	M	26	54	147	83	122	79	82	97	Restrictive very mild
15	M	47	50	135	82	140	91	103	103	Normal

Age=Age in years; Min HR=Minimum heart rate beats per minute; Max HR=Maximum heart rate beats per minute; SDNN=Standard deviation mean RR interval; FEV1%=Force expiratory volume in the first second; TLC%=Total lung capacity; DLCO/VA%=Diffusing capacity for carbon monoxide corrected for alveolar volume; PFT=Pulmonary function test.

ditis in rabbits that may progress to dilated cardiomyopathy [5], normal troponin I, echocardiography and other negative cardiac investigations in our cohort excluded myocarditis and cardiomyopathy. Mild residual CXR changes, minor lung function impairment and normal blood gas makes pulmonary defect unlikely to be a significant cause of sinus tachycardia during normal activity.

This cohort of patients had more severe disease with a high proportion having various complications. The prolonged hospitalization of 18–54 days together with confinement at convalescence could lead to physical deconditioning. The QOL domain score was impaired (score<75) in 11/15 in physical health, 13/15 in psychological well-being, with a very low score of ≤50 in six patients. MFTE score was less than 20 (range 17.6–19.7) in 10 patients indicating the presence of mild functional difficulties.

Deconditioning and anxiety state causes tachycardia in the daytime but not at night, and is compatible with the

Table 2
Other factors

	CXR finding	Hb (g/dl)	LOS (days)	Discharge to PFT (days)	Complication	QOL			MFTE
						Physical health domain	Psychological health domain		
							I	II	
1	Mottling RLZ	13	35	31	AD	75	63	69	20
2	N	12.8	25	36	AD	*44	*50	*50	20
3	Bilat shadowing	13.3	54	21	SP, CI	69	69	75	19.4
4	N	14.5	37	14	Pne	56	*44	*50	19.7
5	Bilat lower zone hazziness	12.8	30	18	ICU	75	75	75	18.8
6	Bilat lower zone hazziness	12.9	39	23	ICU	88	81	81	19.5
7	Bilat middle zone hazziness	14.1	35	21	ICU	63	75	75	18.7
8	Mild hazziness RLZ, LMZ	13.1	21	51	AD	69	63	63	20
9	N	13.9	24	33	AD	63	56	56	19.6
10	Bilat lower zone hazziness	10.6	31	18	UTI	63	56	63	20
11	N	12.6	18	34		63	*50	*50	18.8
12	Bilat diffuse hazziness	14.6	21	45	CI	75	63	63	19.8
13	N	13.6	21	21	CI	55	*50	56	17.6
14	Bilat M and LZ hazziness	14.9	25	23	ITP	69	*50	56	20
15	Bilat lower zone hazziness	13.5	29	29	ICU	63	*25	*38	19.2

Hb=Haemoglobin level; LOS=Length of hospital stay; Discharge to PFT=Discharge to day of pulmonary function test in days; ICU=ICU Care; ITP=Idiopathic thrombocytopenic purpura; UTI=Urinary tract infection; CI=Chest infection; CD=Anxiety depression; SP=Steroid psychosis; Pne=Pneumomediastinum and subcutaneous emphysema; QOL=Quality of life score; MFTE=Monitoring functional task evaluation; *=Score<50; QOL score—Normal>75; MFTE score—Normal>20.

pattern observed in this cohort. In the absence of significant cardiac, pulmonary, thyroid and haematological dysfunction, we believe that sinus tachycardia is attributable to physical deconditioning and contributed by impaired psychological well-being. Appropriate rehabilitation programs should be instituted to enhance recovery of physical and psychological fitness.

Acknowledgements

We wish to thank the staff of the Medical and Geriatrics Department, the Electrodiagnostic Unit and the Occupational Therapy Department of Princess Margaret Hospital for their assistance with this project.

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