

Message from the Chief Hospital Manager

Dear Colleagues,

I am afraid that we are not out of the woods yet with the Covid-19 pandemic. Cases are rising to more than six thousand a day with the public hospitals under pressure again for isolation beds with negative pressure facility. Private hospitals will be or are doing all we can to take up convalescent patients from the public sector or other services in order to free up beds and/ or human resources for the Hospital Authority to concentrate in dealing with the ever increasing work-load. While on the subject I would like to point out a fairly common misconception in the community. People are getting a false reassurance with a negative RAT (rapid antigen testing), thinking that it will be safe to attend a gathering with such findings. One needs to know that the RAT has high specificity but relatively low sensitivity. In other words, there will be a certain number of 'false negatives'. People who have been infected are contagious during the two to three days of incubation when the viral load in the body is building up. Only when it is up to the level detected by RT-PCR testing with CT value of 20 or less will the RAT become positive. Thus when the Centre of Health Protection (CHP) reports 3000 or 4000 positive cases diagnosed by RAT testing one can assume that there would be at least an equal number or even 1.5 times more infectious individuals looming around! Thus one can only feel 'coming home safe' after attending a gathering where everyone has a negative RT-PCR test!

Looking out from my office window I can see the construction work of our three-storeyed extension building on the site of the previous podium garden. My impression is that it is reaching the final stage of construction with cement having been laid on the top of the structure. Workers were preparing the ground work of a landscaped garden on the roof. It is scheduled to apply for occupation permit from the Building Department towards the end of the year. The upper two floors of the completed building will give us forty six en-suite rooms for single occupancy. The remaining floor on the lowest level will house our new clinical laboratory which will be equipped with state-of-the-art automated instruments for various modalities in pathology examinations. This set-up will be able to cater for the demands of our eventual expansion to become a 600-bedded healthcare facility to serve the community.

With the increase number of beds, the corresponding human traffic for access will be beyond the handling capacity for the three original elevators serving the old hospital block. They are being replaced one by one and the process had begun three months ago. Actually the first newly installed lift was commissioned in early August (this month). Although the speed of this new elevator moving up and down remains the same as the old ones, we are able to record an improvement in efficiency of up to 30% because of new designs in the circuit board on the operative functions. It is expected to take another six months for the other two elevators to be replaced. By then there should be more improvement in the running efficiency and moving capacity because of better co-ordination of the three elevators which are now controlled by Al-incorporated circuiting. I would like to thank our patients, staff and visiting doctors for their patience and understanding during these difficult times. Your forbearing effort is being much appreciated. I would like to end this communication here with my sincere wishes that we shall see some light at the end of the tunnel very soon.

Have a Happy Mid-autumn Festival with your family and loved ones.

Yours most sincerely,

Dr Anthony K Y Lee Chief Hospital Manager & Medical Director

Sharing Corner

Beriberi in an Adolescent with Malignancy



Background

Thiamine, a water-soluble vitamin, is a necessary cofactor in many biochemical pathways, such as the metabolism of glucose and the synthesis of amino acids and neurotransmitters, its deficiency can cause beriberi, a disease characterised by inflammatory or degenerative changes of the nerves, the gastrointestinal system and the heart. Beriberi is very rare nowadays, especially in developed countries, * However, such diagnosis requires high level of suspicion and so it may be easily missed in this era. We herein report a case of an adolescent girl with beriberi who had malignancy undergoing chemotherapy. Her cardiac function was remarkably normalised by thismine administration.

Union Hospital

Case presentation

A 17-year-old girl who had high grade osteosarcoma at right knee, previously treated with turnour excision, right total knee replacement, and completed 28-week chemotherapy including Doxorubicin, Cisplatin, Methotrexate, Ifosfamide, Cyclophosphamide and Etoposide. She developed neutropenic fever, tachycardia with heart rate 140 per minute, and hypotension with blood pressure 70/55 mmHg. She received volume resuscitation and antibiotic treatment with Vancomycin and Meropenum. After two days, blood pressure was normalised, but tachycardia was similar and fever was still persistent at 38.5°C. Lactate was slightly improved from 3.6 to 2.5 mmoVL. However, her conscious state was reduced as she suddenly became mute. She had tachypnoea with respiratory rate around 24 per minute. Venous blood gas showed pH 7.53, pCO2 2.3 kPa, HCO3 14.3 mmol/L and BE -5.2 mmol/L, a mixed picture of respiratory alkalosis and metabolic acidosis. In view of the altered consciousness and tachypnoea, with the recent onset of septic shock, she was transferred to PICU for further care.

More thorough examination in PICU revealed gallop rhythm with warm peripheries. There was no heart murmur, Electrocardiography showed sinus tachycardia only. Troponin-T was elevated to 78 ng/L. N-terminal pro b-type natriuretic peptide was >35000 pg/ml. Echocardiography showed globally impaired contractility and dilated left ventricle. Fractional shortening was only 15%. Thus, she had both cardiogenic and septic shock. Her previous chemotherapy was reviewed, showing cumulative Doxorubicin dose 428 mg/m2 and latest Doxorubicin infusion was more than two months ago. Regarding her neurological aspect, she had no meaningful response to her name. Neurological examination showed decorticate posture, generalised flaccidity and areflexia. There was absence of spontaneous movement over upper and lower limbs. Magnetic resonance imaging (MRI) of her brain showed microhaemorrhage and restricted diffusion, which were confined to bilateral frontal cortex, mammary bodies, medial thalami, periaqueductal grey matter and tectal plates. The spatial distribution of the abnormalities was suggestive of Wernicke's encephalopathy. Red cell transketolase level was 68 mU/L/min and thiamine pyrophosphate (TPP) effect was 5%, which were normal. The review of her nutritional history disclosed poor intake for a few months during chemotherapy. After considering the whole clinical picture, the differential diagnoses of her acute heart failure included wet beriberi secondary to thiamine deficiency, and Doxorubicin-induced cardiotoxicity with acute decompensation due to sepsis.

She received infusions of multiple inotropes including Adrenaline, Milrinone and Dopamine. High dose thiamine was given intravenously. Lactate was improved to 2.22 mmol/L. Arterial blood gas was normalised. All inotropic infusions were titrated off gradually after two weeks. Enalapril was commenced. Repeated echocardiography at the third week after PICU admission showed only mildly impaired basal septal motion. Fractional shortening and left ventricle size were normalised. Neurologically, there was gradual improvement in neurological and mental status. She had more voluntary control of her eyes and head after a month. With intense neurorehabilitation, she could raise her upper and lower limbs after 5 months.

Discussion

The recovery of the heart function was fast in this case after the treatment with high dose thiamine and inotropic support. Taking this into account, the most likely diagnosis was wet and dry beriberi secondary to thiamine deficiency, despite its rarity nowadays. The time course of Doxorubicin-induced cardiotoxicity is usually chronic and progressive, and the cardiac function cannot be fully recovered as the damage is usually irreversible, which render such diagnosis unlikely in this case.^[2]

Patients receiving chemotherapy are at risk for nutritional deficiencies including thiamine. And One of the possible mechanisms is chemotherapy-induced anorexia, stomatitis, and gastrointestinal disturbances. (4) Other patient groups who are susceptible to thiamine deficiency or beriberi include those with chronic alcoholism, eating disorders, malabsorption or prolonged parenteral nutrition. And patient's presentation was compatible with the features of both dry and wet beriberi, namely neurological deficits including altered mental status and impaired motor function, and decreased cardiac function from tachycardia with increased lactate to the extent of acute heart failure, respectively. Her MRI brain showing features of Wernicke's encephalopathy was a turning point in diagnosing wet beriberi, because such rare diagnosis is usually difficult to make. Diagnosing wet beriberi requires high index of suspicion and the observation of significant cardiac function improvement after thiamine administration. Laboratory tests like red cell transketolase level and TPP effect may help support the diagnosis, but the sensitivity and specificity of these are doubtful, like the normal results in our case.

Learning points

We have reported a case with mixed picture of wet and dry beriberi probably due to long term poor feeding associated with chemotherapy for high grade osteosarcoma. Wet beriberi should always be considered in patients who are at risk of thiamine deficiency, present with acute heart failure and elevated lactate not responsive to standard interventions. Timely replacement of thiamine can strikingly improve the cardiac function.

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Sharing Corner

The Role of Cardiac MRI in Chest Pain

Dr Chan Siang Hua, Victor Specialist in Radiology Union Hospital

Introduction

Cardiac MRI (CMR) is widely accepted as the non-invasive gold standard for quantifying biventricular volumes, myocardial mass, as well as regional / global systolic function. CMR is also considered to be the most appropriate non-invasive method to assess tissue characteristics in vivo. The fundamental principle is based on the close relationship between the magnetic properties of the tissue and its molecular composition. Standard CMR techniques include contrast-enhanced T1 weighted CMR after administration of gadolinium based contrast agents. These have been successfully applied to evaluate for necrosis, scar, infiltration, inflammation, and intraventricular thrombi. Oedema-sensitive T2 weighted imaging can help to identify acute injury. T2* imaging have also been useful in the detection of myocardial haemorrhage and thrombi. Myocardial mapping allows for the direct measurement of the change of magnetic properties as expressed by native T1, T2, T2* and extra-cellular volume derived from post contrast T1.

Chest pain is an established frequent cause for emergency department visits. This short editorial serves to provide an introduction of the role of CMR in the evaluation and workup of chest pain.

Discussion

Acute Coronary Syndrome (ACS)

Patients with ACS have myocardial ischemia/injury resulting from disruption to coronary blood flow. In STEMI, after invasive coronary angiography, CMR with its imaging technique of late gadolinium enhancement (LGE) allows one to evaluate in vivo, the extent of myocardial injury and hypointense regions within the infarct, which may indicate microvascular obstruction (MO). CMR may help to guide post STEMI care when complicated by heart failure, arrhythmias, or left ventricular (LV) dysfunction. The transmural extent of myocardial damage by LGE is inversely related to the likelihood of functional recovery, with even worse prognosis in the presence of MO. CMR can also help in characterising the affected region in STEMI, as well as to assess complications such as right ventricular (RV) infarct, post MI pericarditis, and thrombus formation. Novel mapping techniques using native T1, T2 and T2* values correlating with LGE affords detailed tissue characterisation. Myocardial oedema is an early manifestation of myocardial damage that can be evaluated with oedema sensitive CMR methods.

MINOCA

A small subset of patients with suspected ACS may have angiographically non obstructive coronary arteries termed myocardial infarction with non-obstructive coronary arteries (MINOCA). Amiling at a diagnosis is hence challenging and may pose a dilemma as the underlying aetiology may not be always immediately apparent. Despite having non obstructive coronary arteries, patients with MINOCA have an increased risk of experiencing major cardiovascular events, including death. Pasupathy et all reported 4.7% annual mortality, which is lower than for MI with CAD (8.7%), but higher than in patients with stable chest pain (0.2% mortality). Causes of MINOCA include AMI with spontaneous recanalization, acute myocarditis, Takotsubo cardiomyopathy and other cardiomyopathies. CMR is increasingly being recognised as a first-line imaging modality in the diagnostic workup of MINOCA to detect the relevant aetiology in a timely manner.

Acute Myocarditis

Multiple causes of acute myocarditis are present, including viral infections (such as COVID), autoimmune disease and toxins. Although endomyocardial biopsy remains the gold standard in diagnosis, its routine use is declining and non-invasive imaging modalities such as CMR is now becoming first-line in the workup for acute myocarditis. CMR with LGE can show characteristic changes especially if performed early, within 2 weeks of the index presentation. It can also frequently distinguish between acute myocarditis, other cardiomyopathies occult MI and other causes of MINOCA CMR adds diagnostic value to a standard clinical followup. The diagnostic targets of CMR include oedema, and an increased extracellular space caused by necrosis or scar. The recently updated Lake Louise Criteria for CMR in Non-ischemic Myocardial inflammation recommend assessment using T1 based and T2 based markers that detect myocardial edema and injury. Myocarditis is typically benign and myocardial oedema disappears within weeks, whereas ineversible injury results in scars with persisting LGE.

Takutsubo Cardiomyopathy

Stress-induced cardiomyopathy, also known as Takutsubo cardiomyopathy, is characterised by a reversible, extensive systolic wall motion abnormality, typically primarily involving the mid and apical LV. CMR can visualise systolic "ballooning" of the LV. On the tissue level, the hallmark of stress induced cardiomyopathy is transmural extensive oedema. LGE is rarely observed.

Non Ischaemic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) and dilated cardiomyopathy (DCM) are the 2 most common forms of nonischemic cardiomyopathy presenting as MINOCA. The hallmark of HCM include an inadequate, mostly asymmetric increase of wall thickness and typically increased LV mass, associated with structural abnormalities, regional fibrosis and LV outflow tract obstruction. These can be quantitatively assessed by CMR. In DCM, features are characterised by ventricular dilatation, global systolic dysfunction, and of accompanied by global myocardial fibrosis. LGE imaging can visualise regional fibrosis, which allows for discriminating nonischemic DCM from ischaemic cardiomyopathy by the predominant subendocardial involvement in ischaemia. An intramural layer of hyperintensity, typically involving the basal anteroseptal segment, seen in one quarter of patients, may be a predictor of sudden death and ventricular arrhythmia.

Chronic Coronary Artery Disease

Delineation of ischaemic myocardium with stress myocardial perfusion imaging can guide coronary artery revascularisation, targeting the ischaemic segments towards relief of symptoms. Several head-to-head trials now endorse stress CMR with vasodilator perfusion is more accurate modality in evaluating the symptomatic CAD patient, with better utilisation of costly resources like ICA. The MR-INFORM trial places stress CMR on equal footing as assessment of what many deem the gold standard for CAD severity – invasive fractional flow reserve. CMR has distinct advantages over other modalities. Having both LGE and perfusion imaging techniques, CMR allows one to better distinguish between scar and hibernating myocardium. Given its high spatial resolution, this size, location and transmural extent of myocardial infarction by LGE CMR closely matches histopathology. Myocardial segments with more than 50% transmural infarction have a low likelihood of functional recovery, while segments exhibiting less than 50% transmural enhancement are more likely to have contractile function. The presence and extent of scar detected by LGE is considered to be a powerful predictor of prognosis, independent of LVEF.

Conclusion

With the emergence and maturation of novel techniques in CMR such as multi-parametric mapping for tissue characterisation, as well as the "workhorse" sequences of LGE and cine imaging for the evaluation of scar/viability and cardiac quantification, CMR holds great potential to be clinically useful in the evaluation and workup of the patient presenting with chest pain. Dedicated rapid CMR imaging protocols or compressed sensing tine may be able to shorten scanning times and permit diagnostic acquisition even in patients who are deemed critically II. CMR would be able to aid in clinical diagnosis, assist in risk stratification and guide further therapy where appropriate.

Acknowledgements

Dr. Chan would like to acknowledge Dr. John Hul and the staff at MEI department for their support. He would also like to acknowledge the effort and dedication of the radiographers and allied staff who contribute to petient care, image acquisition and post processing.

Profile

Dr. Victor Chan is a Specialist in Radiology and serves as a full-time staff radiologist in the Medical imaging Department at Union Hospital. He received his medical degree from the National University of Singapore and completed his post-graduate specialist training in Clinical Radiology at Queen Mary Hospital, Hong Kong. Dr. Chan subsequently went on to pursue a one-year clinical fellowship training in Cardiothoracic imaging at Toronto General Hospital, a quaternary referral centre fully affiliated with the University of Toronto. His research focused on ischaemic and non-ischemic cardiomyopathy, COVID myocarditis and genetic aortopathies. Dr. Chan was awarded the American Heart Association Young investigator Award (2018) and Asian Society of Cardiovascular imaging Best Scientific Presentation Award (2018) for his work on Cardiac MRI. He is dual accredited in SCMR and SCCT level III, and is passionate about the development of CMR service in the local community. Dr. Chan can be contacted via the Medical imaging Department in Union Hospital.

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News & Events

Awards

Union Hospital Garnered CAPTIAL Service & Innovative Product Awards 2022

Union Hospital has garnered the CAPITAL Service & Innovative Product Awards 2022 in the Healthcare Category. This accolade recognizes the hospital's continued commitment for providing high quality and innovative healthcare service for the patients. The recognition was received by Professor Henry Chan, Deputy Chief Hospital Manager of Union Hospital during the award ceremony held on 12 August 2022 at Island Shangri-la Hotel.





The award was received by Prof Henry Chan



CME Post-Event Hightlights:

"The Medical Council of Hong Kong" focusing on how it deals with complaints against a Medical Doctor

Union Hospital hosted a CME programme on 29 July 2022. Professor Lau Wan Yee, Chairman, Medical Council of Hong Kong was invited to give a lecture by sharing knowledge and views on the topic of "The Medical Council of Hong Kong" focusing on how it deals with complaints against a Medical Doctor. A discussion was chaired by Dr Anthony Lee, Union Hospital Chief Hospital Manager and the lecture was well received with great response from the doctors.





From left: Dr Wu Wing Yee, Clara, Prof Lau Wan Yee, Dr Anthony Lee, Dr Cheung Chin Pang

New Clinical Sessions

Specialty Clinic - Paediatrics		
Booking & Enquiry: 2608 3366	Time Schedule	
Dr Tsang Wing Yan, Jennifer	Mon 15:00 - 18:00 Tue 09:30 - 13:00 Wed 09:30 - 13:00 Fri 15:00 - 18:00 Sat 09:30 - 13:00	
Specialty Clinic - Obstetrics & Gynaecology		
Booking & Enquiry: 2608 3222	Time Schedule	
Dr Chow Kei Man	Mon 10:00 - 13:00 15:00 - 18:00 Tue 10:00 - 13:00 Thu 15:00 - 18:00 Sat 10:00 - 13:00	
Specialty Clinic - Ophthalmology		
Booking & Enquiry: 2608 3368	Time Schedule	
Dr Lau Sing Lok, Charles	Mon 11:00 - 13:00 (By Appointment)	
Minimally Invasive Centre		
Booking & Enquiry: 2608 3383	Time Schedule	
Neurology Dr Soo Oi Yan, Yannie	Fri 14:00 - 17:30	

Union Hospital Polyclinic (Tsim Sha Tsui)		
Booking & Enquiry: 2375 3323	Time Schedule	
Neurology Dr Soo Oi Yan, Yannie	Tue 15:00 - 17:30	
Union Hospital Polyclinic (Tseung Kwan O)		
Booking & Enquiry: 2721 0100	Time Schedule	
Paediatrics Dr Fung Cheuk Man, Ronald	Tue 10:00 - 13:00 Wed 15:00 - 18:00 Fri 15:00 - 18:00 Sat 09:30 - 12:30	
Paediatrics Dr Tsang Wing Yan, Jennifer	Tue 15:00 - 18:00 Thu 10:00 - 13:00	
Union Hospital Polyclinic (Tsuen Wan)		
Booking & Enquiry: 2608 3377	Time Schedule	
Otorhinolaryngology Dr Lau Tak Yin, Felix	Mon 14:00 - 17:00 Wed 10:00 - 12:00 Fri 15:00 - 18:00	

New Doctors

Please extend a warm welcome to the following doctors for joining our clinical team!



Dr Tse Cheuk Kwun Specialist in Anaesthesiology



Dr Lau Tak Yin Consultant in Otorhinolaryngology



Dr Chang Wei Yin Consultant in Emergency Medicine



Dr Lau Sing Lok Specialist in Ophthalmology

Regular Meeting

Meeting : Clinical Pathologic Conference	
Date : Time :	14 September 2022 (Wednesday) 8:30 a.m 9:30 a.m
Co-ordinator:	Dr Fung Ming Kit, Terence Deputy Head, Department of Surgery Union Hospital
	Dr. Lui Chi Wai, Philip Consultant Pathologist Union Hospital
Venue	Training Room, 8/F MIC, Hospital Building, Union Hospital
Booking & Enquiry:	2608 3151 (Quality Assurance and Training Dept.)

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