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香 港 醫 訊

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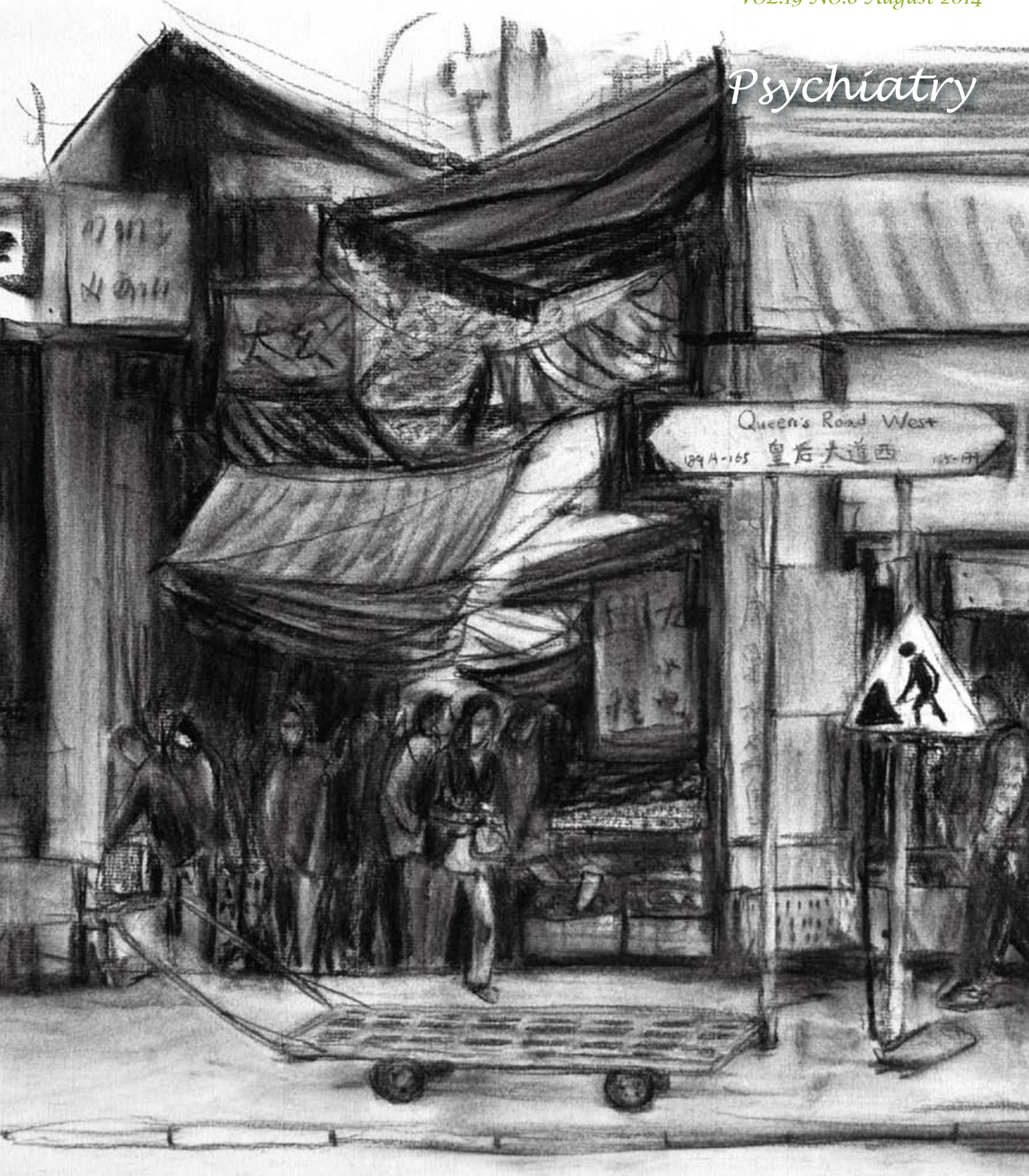
Psychiatry



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The Cover Shot

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Life

This is a picture featuring the old district of Sheung Wan. People are busy with their routines at the roadside. I used charcoal to portray this picture in order to express my feelings of the contrast between the old and the modern Sheung Wan. The picture also illustrates the beauty of the uneven thick and thin lines, rough but refined.

The pace of life in Hong Kong is so fast and stressful. I hope that people in Hong Kong can take a moment's pause and appreciate the scenes of everyday life.

Jianzhong WU
Painter,
Graphic Designer

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No Health without Mental Health

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Editor

Prof Yun-kwok WING

This issue of the Medical Diary is focusing on various facets of mental health. Mental health is not an arena owned by psychiatrists, but a cross-specialties territory. It is an integrated part of our daily clinical practice. From the public health perspectives, a substantial portion of the global burden of diseases is contributed by the mental disorders because of their chronic, relapsing course and handicapping nature.[1] Apart from the high prevalence, they often have an adverse and bidirectional relationship with physical disorders. A notable example is the double burden of diabetes and depression with reciprocal adverse influences on each other.[2]

Dr SP Lam has an overview of recent advances and the paradigm shift of the nosology and management of one of the commonest symptoms and disorders in mental health – insomnia. Being a highly prevalent and co-morbid condition, we encounter many patients with insomnia complaints in our daily practice. Instead of being a benign self-limiting condition, insomnia often runs a chronic course with intriguing and bidirectional relationship with both physical and mental health. The persistent nature of insomnia disorders argues for an integrated approach with both pharmacotherapy and psychotherapy.

Dr Marco Lam writes on the recent advances in the management of resistant depression. Although antidepressants are the mainstay for the treatment of depression, especially those with moderate severity or above, only 50-60% of subjects would respond to the initial course of antidepressants. The subsequent intervention includes a variety of treatment strategies from switching, combination and augmentation of antidepressants to psychotherapy and electroconvulsive therapy. The latest evolvement of minimally invasive surgical techniques and better understanding of the neuro-circuitry of depression has prompted the development of newer and safer neuro-surgical treatments such as deep brain stimulation in the management of seriously handicapped treatment-resistant depression.

Dr Arthur Mak writes on the interface of the irritable bowel syndrome (IBS) and mental disorders. Both disorders share a lot of common aetiologies including genetic predisposition, HPA axis, fear conditioning and neuroimaging findings. The recognition of the close comorbidities between IBS and mental disorders (especially anxiety and depressive disorders) is important as it will guide the proper management of these disorders. The use of antidepressant drugs and psychological intervention will help the individuals with IBS, especially with comorbid mental disorders.

The ageing population of Hong Kong calls on a better understanding of neurodegenerative disorders among our practitioners. Drs Yan and Tsoh write on a much under-recognised type of neurodegenerative disorder – frontotemporal lobe dementia (FTD). The case vignettes of these FTD cases illustrated their common presentations with behavioural, emotional, memory and language disturbances. The

delayed diagnosis of FTD suggests the need for a comprehensive assessment of a patient who presents with psychiatric features, especially in his late 50-60s.

The close relationship of lifestyle, stress and mental disorders is aptly depicted by the cover picture on the buzzing nature of an old district of Hong Kong - Sheung Wan– "where tradition meets modernity and East meets West". Dr TS Lee, a regular columnist, a gastronomist and a psychiatrist in private practice writes on the importance of balanced lifestyle and mental health, not only for our patients but also for ourselves. As Dr Lee pleaded, could we stop a moment to appreciate the tranquility and scenery of our surrounding atmosphere, shops, history and people?

What would be the future of mental health? From the public health and societal perspectives to individual care, mental health is an integral part of holistic management of our patients. Proper understanding and mastery of mental health knowledge is important for every doctor. Hopefully, the emerging neuroscience would further deepen our understanding in brain neurocircuitry, genetics and psychopharmacology, and thus facilitating the translational integration of bio-psycho-social management into clinical excellence.

No health without mental health. This is time to appreciate and integrate the importance of mental health management in our daily clinical practice.

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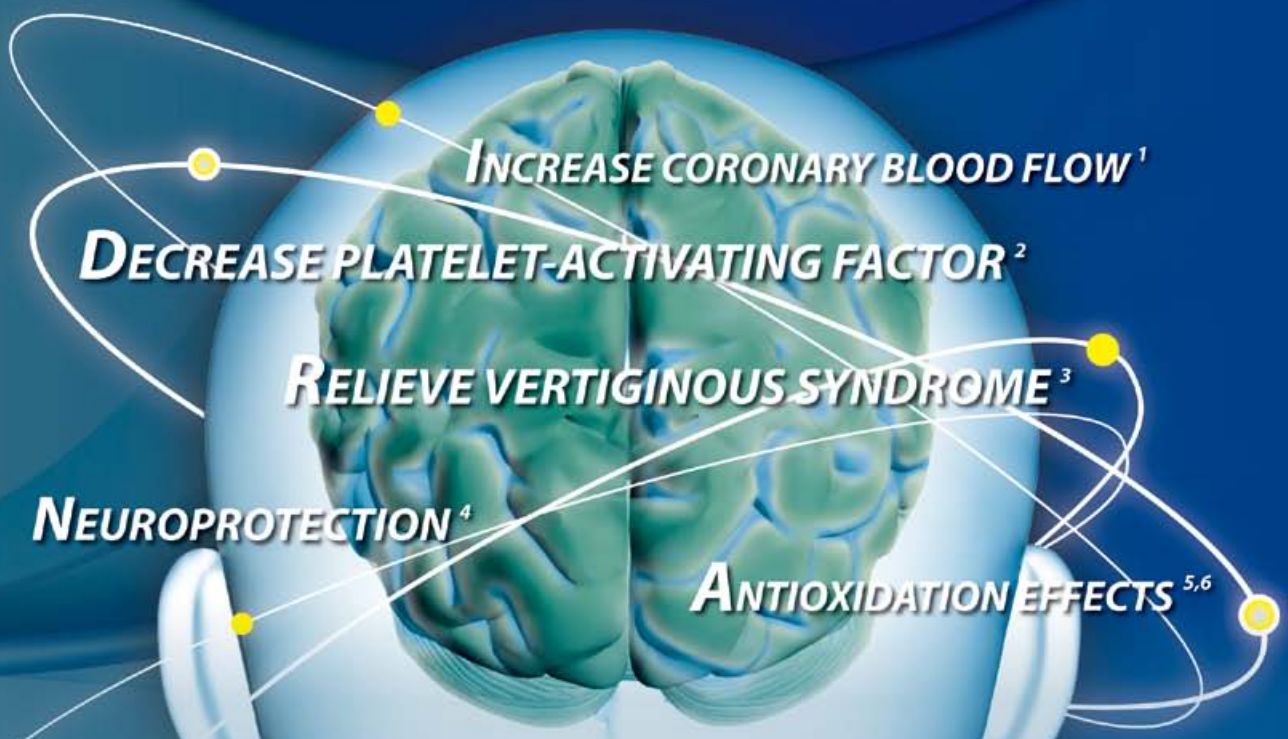

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Examining the Link between Irritable bowel syndrome and Common Mental Disorders – from Aetiology to Treatment

Dr Arthur DP MAK

M.B.,Ch.B., MRCPsych, FHKCPsych, FHKAM(Psychiatry)

Assistant Professor, Department of Psychiatry, Faculty of Medicine, The Chinese University of Hong Kong

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 August 2014.

Irritable bowel syndrome (IBS), affecting 5.4% of the Hong Kong population[1], is a common gastrointestinal disorder characterised by chronically recurring abdominal pain and discomfort, altered bowel habits, and increased anxiety and hypervigilance to symptom-related stimuli[2].

Common mental disorders, such as generalised anxiety disorder (GAD) and depression, frequently occur in IBS patients. Up to 94% of clinic IBS subjects have been reported to have depressive, anxiety and somatoform disorders, a finding leading to doubts about the link between IBS and mental disorders being a function of referral bias driven by abnormal illness behaviour in the mentally ill[3]. Recent community-based findings refuted this assertion. In a representative community sample in Hong Kong, IBS was found to predict a 5-fold increased risk of comorbid GAD, with depressive symptoms as a risk correlate[1].

This is significant because patients with GAD incur the highest health care costs from primary care and specialist physicians among all mental disorders[4]. Comorbid IBS and GAD in particular predicted increased functional impairment[1], and GAD may increase health care costs in FGID patients, via unnecessary repeated medical consultations and investigations driven by health anxiety, as well as prolonged medical care required due to unsatisfactory treatment response from common management paradigms which lacked integrative consideration of these commonly comorbid conditions[5].

Aetiological link

The multifactorial biopsychosocial aetiologies of IBS shows substantial overlap with those of anxiety and depressive disorders.

Serotonin

Serotonin (5-HT) is a ubiquitous signalling molecule in the gut involved in constitutive gut transit, mucosal maintenance, neurogenesis, as well as an essential component of gastrointestinal inflammatory response[6]. It is also a major brain neurotransmitter implicated in depressive and anxiety disorders. 5-HT transporter-linked promoter region (5-HTLLPR) polymorphism, implicated in a range of depressive and anxiety disorders, has been associated with altered serotonin metabolism in IBS. An 'SS' genotype, associated with reduced SERT expression, has been linked to enhanced bowel activity and sensitivity, as well as higher risk of lifetime history of depression[7], while LS and SS genotypes were significantly associated with high somatic symptom scores in IBS patients[8].

G x E - Genes, Early Experience, Neuroticism

Relatives of individuals with IBS are 2-3 times as likely as others to have IBS. The 5 extant twins studies estimated genetic liability for IBS at a range of 1–20%, with heritability estimates ranging between 0–57%. In fact, all[9-12]but 1[13]of the twins studies which used Rome II criteria, yielded higher concordance rates in monozygotic than dizygotic twins (heritability estimates 22%-57%). There have so far been no twin studies with twins reared apart to discern the influence of shared environmental effects on the hereditary estimates. However, when the Rome IBS criteria were converted into a quantitative trait based on the number and severity of IBS symptoms, using data from families but not twin pairs, heritability has been estimated at a range of 0.19-0.35, broadly similar to the range quoted for other anxiety and depressive disorders[14].

IBS also shares with anxiety disorders a host of common genetic associations. Apart from the 5-HTTLPR findings stated above, a number of genes encoding serotonin receptors including HTR2A, HTR7, HTR4 and HTR1E have been found to be associated with IBS. On the other hand, the COMT (catechol-O-methyltransferase) Val158Met variant, associated with many psychiatric disorders, was found to be significantly more common in those with constipation subtype of IBS[15].

As to environmental aetiologies, up to 50% of IBS patients reported lifetime victimisation, a commonly cited risk factor for development of PTSD and other anxiety and depressive disorders. While there is no evidence to show that abuse history moderated gastrointestinal motor and sensory functions in IBS, somatisation and trait neuroticism was found to attenuate the association between abuse history and IBS in population and clinical studies[14].

HPA, pain and fear conditioning

Bilateral brain-gut interactions, involving the HPA axis, autonomic nervous system, and central pain modulation systems, are crucial to normal digestive processes, modulation of gut-associated immune system and coordinating overall physical and emotional state with gastrointestinal activity[16]. There is substantial evidence

to show that IBS, GAD and other anxiety disorders show grossly overlapping neurobiological perturbations in these systems.

Central pain amplification, involving hypervigilance and selective attention to visceral and somatic stimuli, is a core feature of IBS[17] and common to anxiety disorders[18,19]. In anxiety disorders such as PTSD, sensitisation of the hypothalamic-pituitary-adrenal axis to repeated traumatic stress mediates symptoms such as hypervigilance, increased startle response, insomnia and memory processing problems[20].

Impairment of fear conditioning and extinction learning, implicated in perpetuating these anxiety symptoms, has been found to underlie visceral hypersensitivity in IBS and other functional gastrointestinal disorders. A recent fMRI study found upregulated corticotrophin-releasing factor (CRF) signalling in the CRF/CRF-R1 signalling pathway in IBS patients, accounting for impaired extinction learning observed in IBS patients[21]. Altered activation of the thalamus, prefrontal, and somatosensory cortices was found to underlie aversive visceral learning, where expectation of visceral pain relief was shown to substantially change perceived painfulness of visceral stimuli[22]. In IBS subjects, anxious expectation of aversive rectal sensation was found to account for abnormal brain activation to aversive rectal stimuli[23], suggesting that the inability to extinguish conditioned fear responses, similar to patients with anxiety disorders, may account for symptom persistence[24].

Treatment

Management of depressive and anxiety disorders typically involves a holistic biopsychosocial approach including a combination of medications, behavioural and psychosocial interventions. A biopsychosocial approach is also valid for IBS treatment. According to the 2008 NICE guidelines[25], IBS treatment usually starts with anti-spasmodics and other motility agents, taken as-needed according to clinical response, alongside dietary and lifestyle advice, while antidepressant drugs are considered 2nd-line when these measures are ineffective. In practice, comorbidity with depressive and anxiety disorders would commonly necessitate treatment with antidepressants and psychological treatments, many of which have been shown to be effective for IBS (See Figure 1 for a summary of a suggested approach).

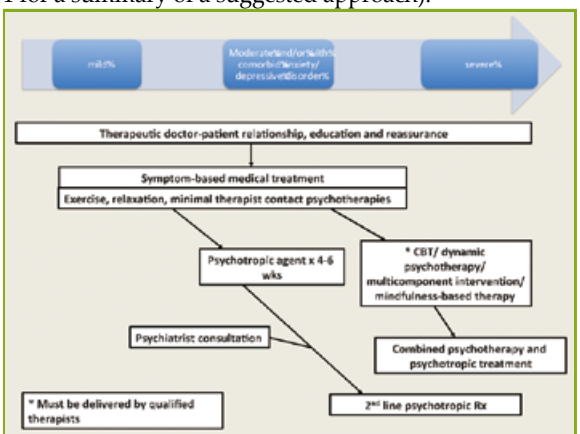

Figure 1. Integrated bio-psychosocial approach to management of Irritable Bowel Syndrome with or without psychiatric comorbidities.

Medications

IBS patients are commonly treated with bulking agents and antispasmodics. In the 2011 Cochrane review on bulking agents, antispasmodics and antidepressants for IBS[26], no significant benefit was found for bulking agents over placebo on abdominal pain, global assessment and symptom scores. Antispasmodics significantly benefited IBS patients in terms of abdominal pain (NNT=7), symptom score (NNT=5) and global assessment (NNT=3). It should be noted that long-term data on effectiveness are lacking in antispasmodic agents.

Increasingly commonly, IBS patients are treated with antidepressants, such as tricyclic antidepressants (TCA) and selective-serotonin reuptake inhibitors (SSRI), putatively on the premises of central nociceptive properties and also its effect on comorbid anxiety and depressive disorders, but some evidence did show that its effectiveness was independent of psychiatric comorbidities. In the 2011 Cochrane review, TCAs and SSRIs were shown to be superior to placebo in improvement of abdominal pain (NNT = 5), global assessment (NNT = 4) and symptom score (NNT = 4). Subgroup analyses showed a statistically significant benefit of SSRIs on improvement of global assessment and for TCAs on improvement of abdominal pain and symptom score. Practically, SSRIs are usually better tolerated than TCAs, especially if initiated at low doses with slow titration to minimise the early and transient side-effects of gastrointestinal upset[27].

In treating anxiety and depressive disorders, benzodiazepines are frequently prescribed for their effects on sleep, anxiety and somatic symptoms, which may make them relevant to patients with comorbid IBS. Commonly prescribed benzodiazepine drugs have not been studied systematically for treatment of IBS, while their use should also be limited due to the propensity for dependence[28].

Psychological and other Behavioural Treatment

Psychological treatment, including cognitive behavioural therapy, interpersonal therapy, relaxation and stress management, has been commonly recommended for IBS given its common psychiatric comorbidity. Meta-analytic evidence is in support of the effectiveness of psychological treatment on IBS.

In an American College of Gastroenterologist's systematic review of 20 RCTs, benefit of psychological therapy over usual care was shown (RR of IBS not improving = 0.67, 95% CI = 0.57–0.79; NNT = 4; 95% CI = 3–5). No benefit was found from relaxation therapy alone, while cognitive therapy, dynamic therapy, and multicomponent psychological therapy were found to be similarly effective for IBS[29].

A recent meta-analysis found consistent effects of mindfulness-based therapy, a newer form of psychological treatment, on pain, symptom severity and quality of life in IBS[30], as well as small to moderate effect on somatisation, anxiety and depressive disorders in general, which are all frequently comorbid with IBS.

Notably, despite the proven effectiveness of psychological treatment, availability has been limited in many low-resource settings, such as the Hong Kong public hospitals system. Self-administered or minimal therapist contact

psychotherapies have been shown to be effective for Depression and Generalised Anxiety Disorder, while for IBS, the relatively sparse data showed that self-administered therapies were ineffective while minimal-contact therapies produced positive results for physical symptom relief[31].

Exercise interventions, such as regular moderate-intensity aerobic exercise, has been shown to be effective for mild depression and Generalised Anxiety Disorders[28]. The evidence for exercise interventions on IBS has been relatively sparse. An RCT of 12-week person centred exercise consultation intervention found daily moderate intensity aerobic exercise to be effective for reducing constipation in IBS[32]. Another large RCT involving 102 IBS subjects found physical activity to significantly improve GI symptoms in IBS[33].

Conclusion

In summary, IBS and common mental disorders such as GAD are highly comorbid with similarities in aetiological factors. Especially when comorbid with psychiatric disorders, ample evidence supports the use of antidepressant drugs and psychological treatment such as CBT, as well as exercise intervention, for individuals with IBS. An integrative approach to management embodying agents modifying gut transit, dietary and lifestyle advice, antidepressant drugs, psychological and exercise intervention should be core to successful management of patients with IBS, with or without psychiatric comorbidities.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Examining the Link between Irritable bowel syndrome and Common Mental Disorders – from Aetiology to Treatment" by Dr Arthur DP MAK and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 August 2014. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. The high prevalence of Irritable Bowel Syndrome in patients with anxiety disorders is solely explained by referral bias.
2. Despite normal colonoscopy findings, repeated colonoscopies should be performed at the request of patients with IBS in order to relieve their anxiety.
3. Benzodiazepines should be routinely prescribed to patients with comorbid IBS and GAD.
4. Treatment-emergent gastrointestinal upset upon commencement of selective serotonin reuptake inhibitors (SSRI) in patients with comorbid GAD and IBS contraindicates further antidepressant use.
5. Being a functional gastrointestinal disorder, IBS does not have any hereditary basis and is completely environmentally mediated.
6. Sensitisation of the hypothalamic-pituitary-adrenal axis to repeated traumatic stress may mediate the pathogenesis of both IBS and GAD.
7. The lack of a history of abuse victimisation rules out the diagnosis of IBS.
8. Evidence supports the effect of exercise intervention and low-intensity psychological interventions on IBS.
9. Relaxation therapy alone may not be sufficient for treatment of IBS.
10. Psychological treatment such as cognitive behavioural therapy should only be delivered to patients by qualified professionals, such as clinical psychologists.

ANSWER SHEET FOR AUGUST 2014

Please return the completed answer sheet to the Federation Secretariat on or before 31 August 2014 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Examining the Link between Irritable bowel syndrome and Common Mental Disorders – from Aetiology to Treatment

Dr Arthur DP MAK

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Answers to July 2014 Issue

Current Status of Laser Use in Oculoplastic Surgery

1. T **2.** T **3.** T **4.** F **5.** T **6.** T **7.** T **8.** F **9.** F **10.** T

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Ref: 1. Dworkin RH et al. Pregabalin for the treatment of postherpetic neuralgia: A randomized, placebo-controlled trial. NEUROLOGY 2003;60:1274-1283. 2. Freynhagen R, et al. PAIN 2005;115:254-263 3. Lyrica (Pregabalin) Prescribing Information. Pfizer Corporation Hong Kong Limited. version May 2013. 4. Bril V et al. Evidence-based guideline: treatment of painful diabetic neuropathy. Report of the American Academy of Neurology, the American Association of Neuromuscular and Electrodiagnostic Medicine, and the American Academy of Physical Medicine and Rehabilitation. Neurology 2011; 76: 1758-65. 5. International Association for Study of Pain. Pharmacological management of neuropathic pain. Pain Clinical Updates 2010; 18: 5. 6. O'Connor A, Dworkin R. Treatment of neuropathic pain: an overview of recent guideline. Am J Med 2009; 122:S22-S32. 7. National Institute for Health and Clinical Excellence. Neuropathic pain: the pharmacological management of neuropathic pain in adults in non-specialist settings. March 2010. 8. Dubinsky RM et al. Practice parameter: treatment of postherpetic neuralgia: an evidence-based report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2004; 63: 959-65. 9. Institute for Clinical Systems Improvement (ICSI). Assessment and management of chronic pain. Bloomington (MN): Institute for Clinical Systems Improvement (ICSI); 2008. 84. 10. Moulin DE et al. Pharmacological management of chronic neuropathic pain – consensus statement and guidelines from the Canadian Pain Society. Pain Res Manag 2007; 12: 13-21. 11. Bandelow B et al. Guidelines for the pharmacological treatment of anxiety disorders, obsessive – compulsive disorder and posttraumatic stress disorder in primary care. International Journal of Psychiatry in Clinical Practice. 2012; 16: 77–84. 12. Attal N et al. EFNS guidelines on pharmacological treatment of neuropathic pain. Eur J Neurol 2006; 13: 1153-69. 13. DORLEGA, S. et al. (2010) Guidelines for the Pharmacological Treatment of Peripheral Neuropathic Pain: Expert Panel Recommendations for the Middle East Region. The Journal of International Medical Research. 38 (2) 10 p.1-29. 14. Lyrica worldwide registration status. Pfizer Corporation. Accessed on 16 Jun 2014. 15. Pregabalin completed clinical trials. https://clinicaltrials.gov/. Accessed on 18 Jun 2014. 16. 2011 Facility of the Year Award Winner for Sustainability: Pfizer Manufacturing Deutschland GmbH, ISPE. 17. HK IMS Sales Data 1st Quarter MAT 2014

LYRICA ABBREVIATED PACKAGE INSERT 1. TRADE NAME: LYRICA 2. PRESENTATION: Each Lyrica hard capsule contains 25mg, 50 mg, 75 mg, 100mg, 150 mg, 200mg or 300 mg of pregabalin. (not all strengths may be marketed). 3. INDICATIONS: Treatment of peripheral and central neuropathic pain in adults; Adjunctive therapy in adults with partial seizures (epilepsy with or without secondary generalisation); Treatment of Generalised Anxiety Disorder (GAD) in adults; Management of fibromyalgia. 4. DOSAGE: 150 to 600 mg/day to be taken in two or three divided doses with or without food. For neuropathic pain: start at 150 mg/day, increase to 300 mg/day after 3 to 7 days, if needed, then to a maximum of 600 mg/day after an additional 7-day interval. For epilepsy: start with 150 mg/day, increase to 300 mg/day after 1 week if needed, then to a maximum of 600 mg/day after an additional week. For GAD: start with 150 mg/day, increase to 300 mg/day after 1 week if needed, then increase to 450mg/day following an additional week if needed, then to a maximum of 600 mg/day after an additional week. For fibromyalgia, recommended dose is 300 to 450 mg/day, dosing should begin at 75 mg BID (150mg/day) and may be increased to 150mg BID (300 mg/day) within one week based on efficacy and tolerability. Patients who do not experience sufficient benefit with 300 mg/day may be further increased to 225 mg BID (450 mg/day). Renal impairment: daily dose should be adjusted based on renal function. Elderly may require a dose reduction. Discontinuation of pregabalin should be done gradually over a minimum of 1 week independent of indication. 5. CONTRAINDICATIONS: Hypersensitivity to the pregabalin or to any of the excipients. 6. WARNINGS & PRECAUTIONS: Avoid in patients with galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption. Adjust hypoglycaemic medications if weight gain occurs in diabetic patients. Use with caution in patients with severe congestive heart failure. Withdrawal symptoms may occur after discontinuation of short term and long term treatment. May cause dizziness and somnolence, which could increase the occurrence of accidental injury (fall) in the elderly population and influence the ability to drive or use machinery. The incidence of adverse events especially somnolence may be increased in the treatment of central neuropathic pain due to spinal cord injury which may be attributed to the additive effect from concomitant medication for the condition. 7. INTERACTIONS: Oxycodone, ethanol, lorazepam, other CNS depressant medications and medications that have the potential to produce constipation such as opioid analgesics 8. PREGNANCY AND LACTATION: Should not be used during pregnancy unless in the opinion of the physician, the potential benefit outweighs the potential risk. Effective contraception must be used in women of child bearing potential. Breast feeding is not recommended. 9. SIDE EFFECTS: Dizziness, somnolence, appetite increased, euphoric mood, confusion, libido decreased, irritability, ataxia, disturbance in attention, coordination abnormal, memory impairment, tremor, dysarthria, paraesthesia, vision blurred, diplopia, vertigo, dry mouth, constipation, vomiting, flatulence, erectile dysfunction, fatigue, oedema peripheral, feeling drunk, oedema, gait abnormal, weight increased, disorientation, insomnia, balance disorder, amnesia, sedation, lethargy, abdominal distension, feeling abnormal.

Reference: HK PI (May 2013). Date of preparation: May 2014. Identifier number: LYR1405

FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.

*Search Drug Data Base, HKSAR. (2014). [Online]. Available: https://www.drugoffice.gov.hk/eps/productSearchOneFieldAction.do [Accessed on public information up to 1 June 2014]

*Pregabalin clinical trials which completed in clinicaltrial.gov as of 2014.

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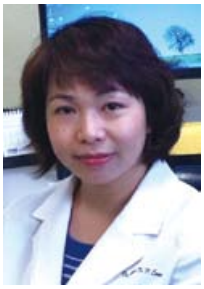
LYR1405(PL)JUN

Insomnia Update – Implication for the Management of Insomnia in Hong Kong



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Insomnia is the commonest sleep complaint across the primary to tertiary level of care. Insomnia disorder (Insomnia symptoms ≥ 3 times/ week for at least 1 month, associated with daytime consequences) affects 10-12% of the general population and is much more common in clinical populations.[1-4] In contrary to previous belief that insomnia tends to be self-limiting without significant repercussion, recent data suggested that insomnia will run a chronic and persistent course[5-7] and is associated with a constellation of physical, pain and mental consequences.[7-10] Insomnia has significant impact on health care cost of the society, including a high rate of absenteeism and a lower productivity at work.[11,12 13] There is an increasing recognition of the impact of insomnia in mental and physical health. In line with the recent release of the fifth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-5) by the American Psychiatric Association in May 2013,[14] the nosology of insomnia, its clinical significance, aetiology and treatment options are revisited in this article.

Major changes about insomnia in DSM-5

Insomnia was previously classified into primary and secondary insomnia, with an implicit assumption to denote the causality of the sleep problem. Primary insomnia referred to insomnia with no identifiable cause while the secondary type referred to insomnia that occurred during the course of other physical or mental disorders. However, as stated in the 2005 National Institutes of Health (NIH) State of the Science Conference statement on the Manifestations and Management of Chronic Insomnia in Adults,[15] the limited understanding about the pathophysiological pathways of insomnia precludes the conclusions about the causality between insomniac symptoms and the medical or mental illnesses. Furthermore, there was concern that the term secondary insomnia might result in under-recognition and under-treatment of insomnia. Hence, the NIH conference recommended the term "comorbid insomnia" as a preferable alternative to the term "secondary insomnia." The evolvement of the terminology into "insomnia disorder" in DSM-5 further underscores the need for independent clinical attention of insomnia regardless of other concurrent mental or medical problems.

Other major changes of insomnia disorder in DSM-5 are summarised in table 1. They included the change of disease duration from 1 month to 3 months to denote the chronic course of insomnia disorder; specification of a chief complaint of sleep dissatisfaction, the inclusion of early morning awakening as a subtype of insomnia and removal of non-restorative sleep as a subtype of insomnia.

Table 1: Major changes of Insomnia diagnostic criteria in DSM-5

	DSM-IV	DSM-5
Terminology	Primary Insomnia	Insomnia Disorder
Types of Insomnia	DIS, DMS or NRS	Complaint of sleep dissatisfaction: DIS, DMS or EMA (Children: bedtime resistance)
Duration & Frequency	1 month ≥ 3 times/ week	3 month ≥ 3 times/ week
Specification	--	Sleep difficulty occurs despite adequate opportunity for sleep

Abbreviations:
DIS- Difficulty in initiating sleep; DMS- Difficulty in maintaining sleep; EMA- Early morning awakening; NRS- Non restoring sleep

Course of insomnia

Insomnia could be a transient condition in reaction to acute stress, life events and changes in sleep-wake schedules (such as jet lag). The transient sleep disturbance usually resolves when the precipitating event is over. However, for some individuals, the acute and transient insomnia will continue to evolve into a chronic sleep disturbance – insomnia disorder. Albeit there is variability of sleep symptoms, insomnia tends to run a persistent course for years. Longitudinal studies reported that the persistence rate of insomnia varied from 20% to 70% in adult populations.[9,16,17] A longitudinal study of over 380 subjects reported that 46% still had insomnia symptoms up to 3 years later. Among those remitted subjects, about a quarter of them had relapse of the insomnia during the study period.[18] In other words, insomnia disorder should be regarded as a chronic problem rather than a self-limiting one.

Insomnia as a co-morbidity and risk factor of physical and mental disorders

Insomnia is a prevailing complaint among various medical conditions. The prevalence ranged from 30% to 70% in clinical populations with chronic heart failure, malignancy and chronic pain conditions.[8, 9, 19-21] Apart from being a common co-morbid condition, increasing evidence from longitudinal studies reported that insomnia heightens the future risk of hypertension, chronic pain and diabetes mellitus (DM).[22-24] A meta-analysis reported that the relative risk of developing DM in chronic insomnia was 1.57- 1.84.[25] The possible mechanisms linking up insomnia and metabolic consequences included hyperarousal-related activation

of the hypothalamic-pituitary-adrenal (HPA) axis (hypersecretion of ACTH and cortisol) and increased sympathetic drive with consequent alteration of the glucose metabolism.[26,27] Chronic sleep disturbance was also found to have altered secretion of inflammatory markers, such as IL6 and the tumour necrosis factor, which could result in weight gain and glucose intolerance.[28]

It has long been recognised that insomnia is one of the commonest presenting symptoms of mental illnesses such as depression and anxiety. The co-occurrence of insomnia and mental disorders were found to be as high as 50% in the general population[29,30] and nearly 80% of sufferers of active depression complained of insomnia.[31] Apart from being a concurrent symptom, longitudinal follow-up studies suggested that insomnia is a predictor of future development of mental illnesses.[29,32,33] A follow-up study of 3.5 years reported that the risk of developing anxiety disorders, depression and alcohol dependence ranged from twice to seven times higher in subjects with insomnia.[32] Similar findings have been reported in adolescents that insomnia is a predictor of behavioural problems and substance misuse.[6,34,35] While insomnia increases the risk of future development of mental illnesses, it is also found to be a common residual complaint in depression.[36] The persistence of residual insomnia in patients with depression predicted future recurrence and non-remission of depression.[37-39] In other words, there is a bidirectional relationship between insomnia and mental disorders, especially depression.

Aetiology of insomnia

The development of insomnia could be conceptualised through a 3'P"model, with predisposing factors, precipitating events, and perpetuating issues cumulating the course. While neurotic and perfectionistic personality may predispose insomnia, there are increasing findings of genetic predisposition of insomnia as suggested by the familial aggregation[40- 42] and twin studies.[43] Acute insomnia (symptoms lasting < 3 month) may run a transient or recurrent course. However, it may become a persistent problem when other factors, such as persistent stressors, cognitive and behavioural hyperarousal responses set in.[44] There are evidences of somatic and autonomic hyperarousal responses among patients with insomnia, such as increased cortical and norepinephrine activities, increased heart rate variability and neuro-hormonal disturbances. Cognitively, insomniac subjects tend to ruminate, become more attentive to the sleep complaints and daytime consequences of insomnia. Together with the maladaptive behaviours and poor sleep hygiene (like spending too much time lying awake at bed), these may contribute to the learned conditioning associations between the bed and wakefulness, which further contribute to persistent insomniac symptoms.

Cognitive behavioural therapy for insomnia

Given the chronicity, its high prevalence, the long-term effects on physical and mental aspects, early recognition and intervention are imperative. Pharmacological treatment with benzodiazepine and preferably, non-benzodiazepine hypnotics is a mainstay of symptomatic treatment. However, there are some setbacks that limit its long term use, such as side effects of hangover, reports on hypnotics related complex sleep behaviours[45, 46], and risk of tolerance and dependence. There is accumulating evidence on the effectiveness of cognitive behavioural therapy for insomnia (CBT-I).

CBT-I is a brief psychological intervention focusing on changing the psychological and behavioural perpetuating factors of insomnia. Common behavioural interventions include relaxation, education of sleep hygiene, paradoxical intention, sleep restriction and stimuli control. The psychological component targets on the common cognitive distortion among insomnia patients, such as exaggerated worries, dysfunctional beliefs and unrealistic expectations. Evidence from meta-analysis and systematic review suggested that CBT-I has moderate to large effect size for various sleep parameters, such as sleep onset latency, sleep quality, total sleep time, wake after sleep onset and number of awakenings.[47-49] A randomised control trial reported over 70% of study subjects had significant therapeutic response while 40% achieved clinical remission.[50] CBT-I has also been shown to have comparable effect with pharmacological treatment[47] and has a more superior effect than drug treatment at long-term (6-month).[50] Its efficacy has been replicated in elderly and clinical populations.

In brief, CBT-I is usually conducted in 4-6 structured sessions with combination of various behavioural and psychological components. Except for sleep hygiene education, most behavioural interventions have been shown to be effective and could be used singly or in combination.[49] A recently published randomised controlled trial compared the efficacy CBT-I components (behaviour therapy (BT), vs cognitive therapy (CT)). The result found that the improvement in clinical symptoms of BT was faster but less sustainable when compared to CT.[51] This findings suggested that both components of CBT-I are unique and could have a complementary treatment effect. While it is commonly conducted by sleep specialists/ psychologists in individual or group basis, a variety of formats have been under investigation, e.g. web-based, group therapy by trained professionals. Given CBT-I is an effective intervention and insomnia is a highly prevalent condition, a wide dissemination of CBT-I with training and supervision is highly encouraged.[52]

Conclusion

Insomnia is a highly prevalent condition across all specialties and levels of care. Over the past 2 decades, there are significant advances in the understanding and conceptualisation of insomnia. Insomnia should be regarded as an independent disorder that warrants separate clinical attention and treatment. It is also recognised that insomnia often runs a chronic and persistent course with significant physical, mental and economic repercussions. Early recognition and intervention is indeed necessary. While pharmacological intervention is a mainstay of short term treatment, increasing evidence suggested psychological intervention is as good as, if not superior to, drug intervention. Training and wide dissemination of the cognitive behavioural therapy for insomnia would be an important strategy for future management of insomnia.

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TRADE NAME: PRISTIQ® **PRESENTATION:** 50 mg, light pink, square (pyramid-one sided) tablet debossed with "W" (over) "50" on the flat side **INDICATIONS:** Treatment of major depressive disorder (MDD) **DOSAGE & ADMINISTRATION:** 50 mg once daily at approximately the same time, with or without food. The recommended dose in patients with severe renal impairment or end-stage renal disease (ESRD) is 50 mg every other day. Supplemental doses should not be given to patients after dialysis. **CONTRAINDICATIONS:** Hypersensitivity to desvenlafaxine succinate, venlafaxine hydrochloride or to any excipients in the Pristiq formulation. Must not be used concomitantly in patients taking monoamine oxidase inhibitor (MAOI) or in patients who have taken MAOIs within the preceding 14 days. **WARNINGS & PRECAUTIONS:** All patients being treated with antidepressants for any indication should be monitored appropriately and observed closely for clinical worsening, suicidality, and unusual changes in behaviour, especially during the initial few months of a course of drug therapy, or at times of dose changes, either increases or decreases. Not approved for use in treating bipolar depression; Serotonin syndrome or Neuroleptic Malignant Syndrome-like reactions; Activation of mania/hypomania; Elevated blood pressure & abnormal bleeding; Serum cholesterol & triglyceride elevation; Narrow-angle glaucoma; Renal impairment; Seizure; Hyponatremia; Interstitial lung disease & Eosinophilic pneumonia; New symptoms and serious discontinuation symptoms were reported in discontinuation of treatment; Caution is advised to patients with cardiovascular, cerebrovascular, or lipid metabolism disorders; Co-administration of drugs containing desvenlafaxine & venafaxine is not recommended. **INTERACTIONS:** Risk in combination with other CNS-active drugs; MAOI; Serotonergic drugs; Drugs that interfere with hemostasis; Alcohol; Concomitant use with potent inhibitors of CYP3A4 may result in higher concentrations of Pristiq; Minimal inhibitory effect of desvenlafaxine on CYP2D6. **PREGNANCY AND LACTATION:** Pregnancy Category C. Carefully consider the potential risks and benefits of treatment when treating a pregnant woman during pregnancy especially in the third trimester, labor and delivery. Only breastfeed if the expected benefits outweigh any possible risk as desvenlafaxine is excreted in human milk. **SIDE EFFECTS:** Most commonly observed adverse reactions in short-term fixed-dose studies were: nausea, dizziness, insomnia, hyperhidrosis, constipation, somnolence, decreased appetite, anxiety, and specific male sexual function disorders. Hypersensitivity, effects on blood pressure, abnormal bleeding, mydriasis, hypomania & mania, serum cholesterol & triglyceride elevation, and seizure were also reported. **DRUG ABUSE AND DEPENDENCE:** Not systematically studied in preclinical/clinical studies for its potential for abuse. Physicians should carefully evaluate patients for a history of drug abuse and follow such patients closely, observing them for signs of misuse or abuse of Pristiq. Limited clinical experience with desvenlafaxine succinate overdose in humans. Reference: HK PI (Version Date JAN2011) Date of preparation: JUL2012 Identifier number: PRIS0712

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What Should We Do When a Depressed Patient Does Not Respond to An Antidepressant?

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Introduction

Major depressive disorder (MDD) affects over 8% of the adult general population in Hong Kong[1]. It was projected that by 2020, depression will be the second leading cause of disability by adjusted life years worldwide[2]. Days lost from work due to depression and the economic burden on individuals, families and society is substantial[3,4]. Depression would also enhance the risk of alcohol and substance misuse[5,6]. Presence of MDD enhances risks and poorer management of comorbid medical problems including cardiovascular disease[7,8], diabetes mellitus[9], and cancer[10,11]. Depression is also the most important predicting factor for suicide attempt and eventually about ten percent of depressed patients may end up committing suicide[12,13].

For mild and moderate MDD, the NICE guideline suggested that either pharmacotherapy or psychotherapy can be considered as monotherapy[14]. Selection of an initial treatment modality is influenced by several factors, including symptom profiles, the presence of comorbid disorders or psychosocial stressors, prior treatment, and the patient's preference. On the contrary, antidepressant treatment is needed for severe MDD as psychotherapy alone is considered inadequate in severe MDD[15].

It usually takes two to three weeks before antidepressant treatment effects will be seen. Eventually about 50-60% of patients will respond to antidepressant treatment provided that the dosage and duration are adequate[16]. The remaining patients fail to return to their premorbid functional levels or their lives are persistently debilitated by the depressive symptoms. Clinically they are regarded as non-responders.

Risk factors of treatment non-response

Multiple factors may contribute to the aetiology of depression. They include genetic predisposition, developmental history, personality profile, precipitating factors and on-going stressors. Marked disturbances in one or more of these factors may be related to non-response to treatment. For instance, treatment non-response is related to low social support, negative social interaction and personality disorder[17]. Comorbid anxiety disorders, chronic pain and substance abuse are also related to treatment non-response[17,18]. It can also be contributed by sleep and circadian disturbances[19,20]. The characteristics of depression itself can also predict the prognosis. For instance, severe depression, the presence of suicidal history, melancholia, multiple hospitalisations, early age at onset, and non-response to the first antidepressant implicate treatment resistance[17].

In managing treatment non-response, we should review the treatment compliance, revise the diagnosis, look for associated personality disorder and comorbid medical and psychiatric conditions. Optimisation of co-morbid medical and psychiatric conditions and removal of on-going psychosocial stressors help to relieve the severity of depression. Since residual depressive symptoms are one of the strongest indicators of subsequent relapse[21], the goal of management is to achieve complete remission as incomplete remissions significantly increase the recurrence rate[22]. For those who do not respond to the above recommendations and have already received an adequate duration and dosage of an antidepressant, the following alternative treatment strategies can be considered.

Switching to intra/interclass antidepressants

There are different classes of antidepressants, including selective serotonin re-uptake inhibitor (SSRI), serotonin–norepinephrine reuptake inhibitor (SNRI), noradrenergic and specific serotonergic antidepressants (NaSSA), norepinephrine and dopamine disinhibitor (NDDI), tricyclic antidepressant (TCA) and monoamine oxidase inhibitor (MAOI). Switching from one antidepressant to another is an option when the side effects of one antidepressant are not well tolerated. Nevertheless, most studies suggested limited additional efficacy no matter whether it was switching to an intraclass or interclass antidepressant[23], albeit SNRI may be slightly superior to SSRI[24]. A washout period is required for certain antidepressants. For instance, a few weeks is needed when switching from fluoxetine to other serotoninergic antidepressants as fluoxetine has an active metabolite, norfluoxetine, which has a long half-life of about five weeks. A two weeks' interval is needed when switching antidepressants to MAOI in order to avoid neurotoxicity complications such as the serotonin syndrome. Serotonin syndrome is a potentially life threatening condition that is caused by excess serotonin in central nervous system. Dependent on the severity, it is characterised by shivering, sweating, mental confusion, myoclonus and seizure[25].

Combination of antidepressants

Combination is the prescription of two antidepressants at the same time. Usually an interclass combination is preferred to an intraclass combination as they can offer different mechanisms of action. Among all antidepressants, the add-on therapy with mirtazapine[26] and bupropion[27] have been supported by some data. Sleep disturbances including insomnia and nightmare are common in depressed patients, which also predict future suicidal risks[20,28]. Medications that promote sleep

may add additional benefits in depressive patients with sleep problems[29]. The disadvantage of combination strategy is the significant increase in adverse effects and drug toxicity[30].

Augmentation

In contrary to combination, augmentation is the addition of a non-antidepressant medication. Atypical antipsychotics[31], lithium and triiodothyronine are the commonest augmentation agents. Among all atypical antipsychotics, quetiapine[32] and aripirazole[33] were found to provide some additional efficacy to antidepressants. Atypical antipsychotics play an important role in treating psychotic depression. However, the addition of atypical antipsychotics may affect the metabolic profile and increase the risk of obesity, diabetes and dyslipidaemia. Lithium[34] and triiodothyronine[35] were found to be effective as an augmentation agent to tricyclic antidepressants but their effect on newer antidepressants has not been systemically studied. Thyroid function monitoring is needed for both lithium and triiodothyronine augmentation and additional surveillance on lithium level, renal function and electrocardiogram is needed for lithium use. Type II bipolar affective disorder, characterised by hypomania and depression, is sometimes misdiagnosed as unipolar depression as hypomanic episodes are difficult to be captured[36]. Bipolar depression is relatively resistant to antidepressant treatment and may require the addition of mood stabilisers[37,38].

Psychotherapy

Different psychotherapies have been developed to treat mild to moderate depression. Meta-analysis showed that the effectiveness of different kinds of psychotherapy is comparable. Among all psychotherapies, cognitive behavioural therapy, interpersonal therapy and problem solving therapy have robust data of efficacy[39]. Not only drug-psychotherapy combination improves treatment outcome[40], depressive patients may also find the combination more acceptable and their adherence can be promoted[40].

Nevertheless, in a recent large scale naturalistic treatment study called Sequenced Treatment Alternatives to Relieve Depression (STAR*D), there were four levels of treatment. The first level was the use of a single SSRI. The second level included seven different treatment options including switching, combination of antidepressants and addition of psychotherapy. The third level was the addition of lithium or triiodothyronine or switching to dual action antidepressants. The final level was the use of monoamine oxidase inhibitor or a combination of two dual action antidepressants. Remission rates for treatment levels 1 to 4 ranged from 7% to 30%. There was no difference in effectiveness between any treatment at any treatment level[41].

Electroconvulsive therapy (ECT)

Up till now, ECT remains one of the most effective treatments of depression with an efficacy superior to antidepressants[42]. Some patients may respond to maintenance ECT[43]. Common adverse effects included muscle soreness, confusion and memory loss. Although studies found that maintenance ECT was not associated with a significant increase in mortality[43,44], compliance issue and long-term cognitive decline[45] limited its use in many TRD patients.

Repetitive transcranial magnetic stimulation (TMS)

TMS is a non-invasive technique to stimulate the cerebral cortex. A coil of wire, encased in plastic, is held to the head. When the coil is energised with rapidly changing current flows in its windings, a magnetic field is produced and it passes through the skin and skull, inducing an oppositely directed current in the brain. Although it was proven useful for the treatment of mild to moderate depression[46-48], its efficacy in TRD has not been well established[49].

Others: diet, yoga and exercise

There are reports stating that depression can be partially relieved by omega-3, vitamin B12 and a folate rich diet[50]. Yoga and exercise may also improve the mental well-being through enhancing the endorphin and brain derived neurotrophic factor (BDNF) system. Nevertheless, these adjunctive therapies have not been extensively studied.

Psychiatric neurosurgery for TRD

Even with the above treatment strategies, about 10% of patients could hardly respond to any intervention and their lives are conspicuously debilitated by the disease[51,52]. Clinically they are regarded as suffering from TRD. One of the commonly adopted criteria for TRD is the failure to respond to a minimum of four treatment modalities, including different classes of antidepressant, evidence-based psychotherapy, and electroconvulsive therapy, administered at adequate doses and duration[16,53,54]. Upon surgical advancement with safer, more precise and less invasive techniques in the last two decades, vagus nerve stimulation (VNS) and deep brain stimulation (DBS) have been developed to combat against TRD.

Vagal Nerve Stimulation

VNS was indicated in the treatment of refractory epilepsy[55]. The procedure was later adopted in the treatment of TRD for several reasons. First, as anticonvulsants could be utilised as antidepressants in mood disorders, the anticonvulsant effect of VNS might alter mood state[56]. Second, epileptic patients receiving VNS reported improvement in depressive symptoms[57,58]. Third, VNS was found to alter concentrations of neurotransmitters implicated in mood disorders, including serotonin, norepinephrine, gamma aminobutyric acid, and glutamate[59-61]. The efficacy of VNS in TRD was preliminarily supported by a small scale open-label study[53], a randomised sham-controlled short term trial[62,] and long term follow-ups of the two cohorts[62]. Unfortunately, only 30% patients responded to VNS and the remission rate was about 15% [53].

Adverse events of VNS fell into two categories: vagus nerve related and surgery related[53]. Since the vagus nerve highly innervates both the respiratory and digestive systems, complaints over respiratory (e.g. voice alternation, dyspnoea and cough) and digestive (e.g. dysphagia and dyspepsia) symptoms were reported. Surgical related adverse events included incision site pain and headache. Most of the adverse events were self-limiting or could be well tolerated by

the majority of patients. About 3% of patients receiving VNS developed hypomanic features, which were responsive to titration of either concomitant psychiatric medications or VNS output current. Serious adverse events of VNS included prominent pain and infection over the implantation site, myocardial infarction and worsening of mood resulting to suicidal intents. In 2005, VNS has been approved by the U.S. Food and Drug Administration for the adjunctive long-term treatment of chronic or recurrent depression[63]. To our knowledge, the procedure has been adopted locally in the treatment of resistant epilepsy[64] but not in resistant depression.

Deep Brain Stimulation

Compared with VNS, DBS was more extensively studied in the treatment of TRD because of its prior excellent efficacy in the treatment of movement disorders[65,66]. It has been adopted in the treatment of TRD with preliminary success in different sites: inferior thalamic peduncle[67], lateral habenula[68], ventral capsule/ventral striatum(VC/VS)[69], subcallosal cingulate gyrus[70], nucleus accumbens[71] and medial forebrain bundle (MFB)[72]. Depending on the site, the effect can be immediate[72] and long-lasting[69,73]. One study showed that over half of patients could achieve remission[72]. The major limitation of the DBS study is that all were open trials but its efficacy was partially supported by the associated differences in clinical ratings when the stimulator was turned on and off. Common side effects of DBS included anxiety, perioperative headache, pain at generator site, blurred vision, strabismus, dizziness and increased sweating. It also poses the risks of seizure, infection and intracranial haemorrhage but most are mild and self-limiting. Irreversibility and cost are the main concerns of the surgery. Ostensibly, it is considered as the last resort in the management of TRD. To our knowledge, it has been adopted locally to treat TRD.

Conclusion

By adopting different treatment strategies, most depressive patients can be benefited from a comprehensive bio-psycho-social approach. Augmentation and combination strategies can relieve depression in most non-responders. With the advances of minimally invasive surgical techniques and newer brain imaging techniques that improve our understanding in the neuro-circuitry of depression, neuro-surgical treatment will offer a new chapter and hope for the management of seriously handicapped TRD patients.

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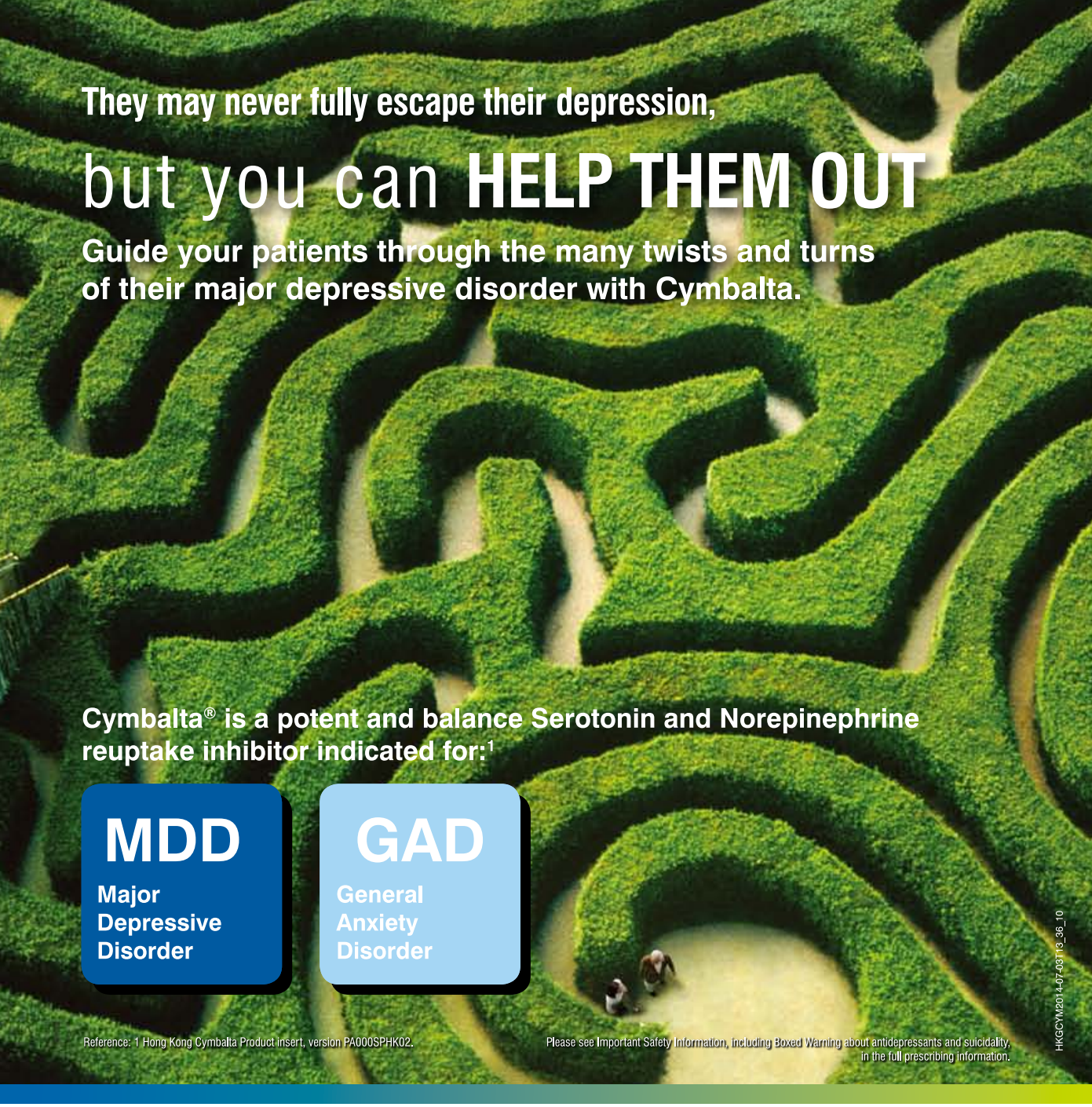

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Frontotemporal Dementia (FTD): A Common Dementia Commonly Misdiagnosed

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Introduction

Frontotemporal dementia (FTD) is a clinically heterogeneous syndrome of progressive behaviour or language decline associated with frontal and anterior temporal lobe degeneration (Rabinovici & Miller, 2010). Being relatively common and a leading cause of presenile dementias second to Alzheimer's Disease (AD)(Hokoishi et al., 2001; Panegyres, Davies, & Connor, 2000; Panegyres & Frencham, 2007), FTD is frequently misdiagnosed and is an underappreciated cause of dementia in older individuals (Barker et al., 2002; Brunnstrom, Gustafson, Passant, & Englund, 2009; Lebert, Pasquier, Souliez, & Petit, 1998). The onset is typically presenile and in the sixth decade though some may present later (Hodges, Davies, Xuereb, Kril, & Halliday, 2003; Johnson et al., 2005; Ratnavalli, Brayne, Dawson, & Hodges, 2002).

The prevalence of FTD in Hong Kong has not been formally reported, but a study profiling attendees of a local memory clinic diagnosed 1.8% of their 385 demented subjects with FTD (Sheng, Law, & Yeung, 2009), while 10.9% of them had either "other irreversible dementias" or "undetermined dementia". A Chinese study (Ren et al., 2012) reported no difference in age of onset and sex distribution between FTD diagnostic subtypes. Interestingly, FTD has been reported to be as common in Asians as Caucasians (Hou, Yaffe, Perez-Stable, & Miller, 2006). This apparent discrepancy in prevalence may be due to the late presentation of FTD patients in China (Chao et al., 2013) and other Asian regions (Ghosh, Dutt, Ghosh, Bhargava, & Rao, 2013), and also due to the lack of criteria in identifying early FTD in Asians (Ghosh et al., 2013; Sheng et al., 2009).

The three clinical syndromes of FTD – behavioural variant frontotemporal dementia (bvFTD), semantic dementia (SD) and progressive non-fluent aphasia (PNFA) – can overlap in presentation, especially later in the course when degeneration spreads (Neary et al., 1998). Clients with bvFTD show marked personality changes, behavioural disturbances and impaired insight. Their cognitive decline is less striking and deficits in frontal and executive tasks are demonstrated. Memory complaints are not uncommonly encountered, but, unlike AD, studies show these are usually related to inattention and not disruption in memory faculties (Wittenberg et al., 2008). The principle in FTD diagnosis is to exclude treatable conditions (Rabinovici & Miller, 2010). It is common for FTD to be misdiagnosed as a psychiatric disorder, though a small subset of bvFTD patients do have a comorbid psychiatric, usually affective, disorder (Davies et al., 2006; Kipps, Nestor, Dawson, Mitchell, & Hodges, 2008).

The three case vignettes below are exemplary of the FTD spectra, illustrating how initial presentations can masquerade as psychiatric symptoms, leading to a lapse before establishing the diagnosis of FTD. The implications of delayed diagnosis and missed opportunities for treatment are self-evident.

Case 1

Ms A was a 76-year-old widow living with her daughter and functioned independently. She had a history of diabetes, hypertension, cataract and bilateral ptosis. There was no family history of mental illnesses or neurodegenerative diseases.

She was first known to the mental health service at 59, presenting with distress from chronic discomforts – described as "a clumsy, entangled mind" (「腦筋黐住」), low mood, anhedonia and a suicide attempt. Her diagnosis was "depressive illness". In the ensuing years, Ms A continued to suffer from similar symptoms despite medication titration, electroconvulsive therapy (ECT) and repeated hospitalisations. Computed tomography (CT) scan of her brain was unremarkable. From 75 onwards, she began seeing non-existent images that were associated with REM sleep disturbances. She also suffered from concurrent memory decline with confusion, short-term memory loss and apraxia with preservation of basic functioning. Her family noticed that she grew slower and clumsier. At this point, possible differential diagnoses including Parkinson's Disease and Parkinson-plus Syndromes were considered.

Neurocognitive assessment revealed impairment in delayed recall, working memory, short-term memory (STM), cognitive flexibility, language fluency and constructional ability. Her Mini-Mental State Examination (MMSE) score was 28/30 (within normal range) and the Montreal Cognitive Assessment (MoCA) score was abnormally low at 19/30. Magnetic Resonance Imaging (MRI) of the brain showed atrophy in the frontal, superior temporal, parietal and pontine regions. Magnetic Resonance Spectroscopy (MRS) of the brain showed features suggestive of frontotemporal dementia (FTD). Single-Photon Emission Computed Tomography (SPECT) perfusion scan of the brain showed mild to moderate and symmetrical frontal hypoperfusion

suggestive of frontal lobar degeneration (Figures 1 and 2). She was referred to Neurology for assessment of possible Parkinson symptoms.

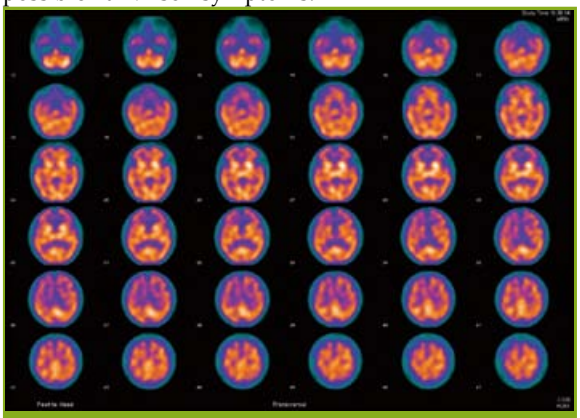

Figure 1: Single-Photon Emission Computed Tomography (SPECT) brain perfusion scan of Ms A showing typical frontal-temporal perfusion deficits of FTD (1: Transverse view)

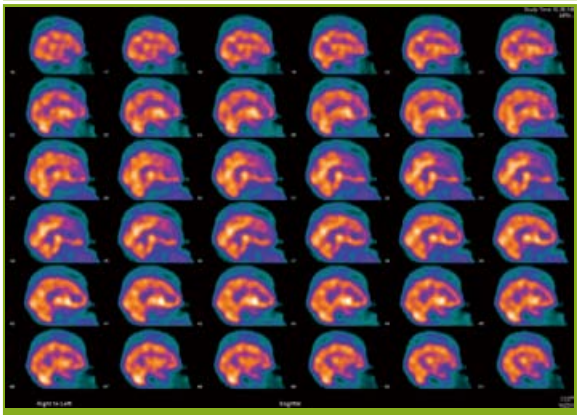

Figure 2: Single-Photon Emission Computed Tomography (SPECT) brain perfusion scan of Ms. A showing typical frontal-temporal perfusion deficits of FTD (2: Sagittal view)

Therefore Ms A's diagnosis was revised to frontotemporal dementia (FTD). Her medications were adjusted and antipsychotics were stopped. The patient and her family were duly advised on cognitive compensation strategies.

Comments: This case illustrates the difficulty in distinguishing whether patients suffer from FTD or psychiatric disorders. With longstanding psychiatric symptoms and prolonged psychotropic use, in addition to a history of hospitalisation and ECT, the significance of the neuroimaging findings may appear equivocal. However, with the neurocognitive assessment showing poor STM and impairment in executive function and language fluency – this would suggest FTD as a more likely diagnosis.

Neuroimaging occasionally detects frontal and temporal lobe atrophy in patients with psychiatric illness, this poses diagnostic difficulty between the two entities. This may be explained by multiple factors, including comorbidity, the utility of consensus criteria and neuroimaging results (Panegyres & Frencham, 2007). It is suggested in recent studies that progressive brain structural changes may be found after prolonged treatment with antipsychotic medications or ECT (Madsen, Keidling, Karle, Esbjerg, & Hemmingsen, 1998). This further illustrates the need to elucidate the relationship between psychiatric illness, neuroactive medications and structural brain changes and the importance of interpreting radiological results with the clinical and neuropsychometric findings in mind.

Unfortunately there is no biological marker to assist in diagnosis. The presence of a family history might be helpful for patients presenting with presenile dementia, but FTD is more likely to be sporadic. It is important to remember that FTD is more common than most realise. It might be beneficial to develop a clinical profile for at-risk cases to facilitate the diagnostic process. Panegyres et al. (2007) suggested this to include relevant clinical information, frequency of neurological signs and symptoms, neuropsychometric assessments – especially comprehensive speech and language tests to detect communicative dysfunction, neuroimaging results, etc. This profile should be further refined to better represent the patient population, in the hope that this would be augmented by biological markers and genomic analysis in future. Although tools such as functional brain imaging (in the form of SPECT, PET scans) aid in differentiating the two groups, a practical limitation remains as not all clinicians have access to these tools.

Case 2

Ms B was a 64-year-old retired labourer living with her family. She was referred by her general practitioner for psychiatric assessment for "3 years of frequent forgetfulness and dizziness". The patient reported anxious feelings associated with "forgetfulness" – she could recognise but not name her acquaintances and had similar problems in her daily chores. Outwardly her memory and functioning appeared preserved. Except for her frustrations in naming difficulty and her family's negative comments on her being "lazy", Ms B did not experience any disturbance in sleep, appetite or mood. There was no history of psychotic features, self-harm or violence. Ms B's physical health was good and her family history was unremarkable. The patient appeared anxious during the initial assessment and failed in several cognitive tests with impaired short term memory and clock-drawing. Her diagnosis was Generalised Anxiety Disorder with suspected cognitive impairment. She was started first on an antidepressant and later on cognitive enhancers with partial improvement.

Ms B was subsequently admitted to a psychogeriatric day unit. By then her mood was less anxious, though there was further memory decline. The patient's performance in neurocognitive assessments was all along affected by her difficulty in word-finding and her impaired comprehension –for example, her latest MMSE was 13/30 – but moderate to severe deficits with impaired recall were consistently found. Routine dementia screening blood tests were unremarkable. CT brain showed mild generalised volume loss of the left cerebral hemisphere. MRI confirmed mild generalised decrease in the left cerebral hemisphere. The SPECT brain perfusion scan showed moderate hypoperfusion of the left cerebral hemisphere – involving regions like the Wernicke's area, Broca's area, the speech fasciculus in the occipital and frontal lobes – suggesting a typical primary progressive aphasia pattern (Figure 3).

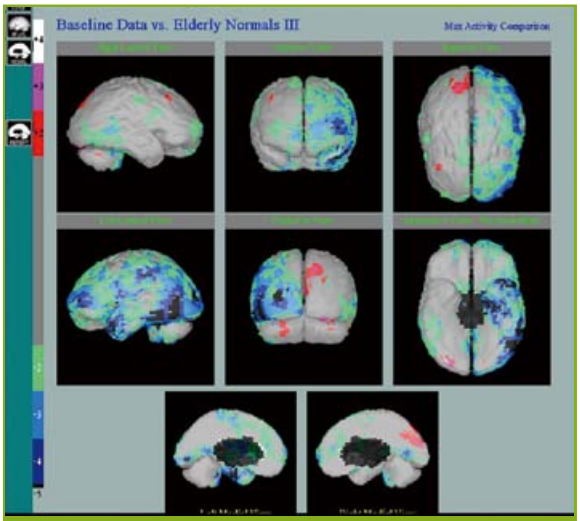

Figure 3: SPECT brain perfusion scan of Ms B (Using the Talairach analysis to compare with normal elderly, which showed unilateral left cerebral moderate hypoperfusion involving Wernicke and Broca areas, occipital and frontal lobes inside which are the speech fasciculi. The right cerebral hemisphere was essentially normal.)

Ms B's diagnosis was revised to FTD and of the Primary Progressive Aphasia (PPA) variant. In addition, her family was educated on the patient's condition and caregiver skills (e.g. to avoid negative comments). The patient was referred for day care service on discharge with out-patient cognitive training.

Comments: The diagnosis of Ms B was the semantic variant of PPA. The client exhibited progressive deterioration in the comprehension of words (especially nouns) and object recognition, with sparing of verbs and abstract words. These patients are able to produce fluent speech but cannot generate key words, adding to the difficulty in understanding and the distress. The performance of Ms B in verbally mediated memory tests was affected by her word-finding difficulty and problems in comprehension – again a distinctive feature among PPA patients.

PPA, as defined by Mesulam (2007) and his associates, is the diagnosis made in a patient in whom a language impairment (*aphasia*), caused by a neurodegenerative disease (*progressive*), constitutes the most salient aspect of the clinical picture (*primary*). The language impairment can be fluent or nonfluent and may or may not interfere with word comprehension. This neurodegeneration goes on to involve most of the cerebral cortex and many additional deficits are ultimately displayed.

There are many terms employed to denote PPA variants, this might cause confusion in terminology but the term "PPA" should still be used as a root diagnosis as not all progressive aphasias fulfil the diagnostic criteria of PPA, and not all PPA cases are caused by frontotemporal lobar degeneration. In 60-70% of PPA cases, the neuropathology shows FTD subtypes, but shows the plaques and tangles characteristic of AD in the others. There are 3 variants of PPA: (a) nonfluent variant PPA (NFPA) characterised by the breakdown in speech production with variable speech apraxia and agrammatism; (b) semantic variant PPA (SD) characterised by early loss of vocabulary with impaired word comprehension and associated deficits in non-verbal semantic memory; and (c) logopenic variant PPA describing an intermediate state between NFPA and SD, with prolonged word-finding pauses (anomia) and impaired auditory verbal short-term memory.

Due to the phenotypic diversity and the overlapping clinical, neuroanatomical, molecular and pathological profiles of PPA, clinical diagnosis remains the most important criterion. The deficits must not be better accounted for by other neurodegenerative disorders or medical conditions (e.g. stroke). It is common to exclude major cerebrovascular accident (CVA) or small vessel disease by CT or MRI. Brain perfusion scans (SPECT or PET) can aid in diagnostic confirmation and subtyping (Boxer et al., 2003; Nestor & Hodges, 2000; Silverman et al., 2001). MR tractography can sometimes delineate the speech conduction fibre atrophy. Typical neuroimaging investigations and findings are asymmetry in cerebral atrophy (left more than right) and left peri-Sylvian fissure atrophy – the area responsible for speech production in the dominant hemisphere – on CT and MRI. Brain perfusion scans such as SPECT and PET scans show the Broca and Wernicke areas being affected with left arcuate fasciculus atrophy in the classical type, other brain parts for language coordination (such as longitudinal and uncinated fasciculus) may also be involved, manifesting different patterns for different subtypes. PET scan is the most sensitive tool to detect PPA, followed by SPECT, MRI and CT in decreasing order of sensitivity.

Case 3:

Mr C was a 66-year-old married retired teacher who lived with his family and first presented with one year of low mood, emotional dulling, social withdrawal and preoccupations with somatic discomforts. He was premorbidly gentle and introverted and was new to the mental health service. No psychosocial stressors were identified previously. He was diagnosed with Depression but only showed limited response despite two adequate antidepressant trials. Mr C's symptoms worsened with increased irritability and more ruminations about physical discomforts; he also quarrelled with his wife more often and resulted in severe carer stress. A computed tomography (CT) brain scan did not show any abnormalities (Figure 4). Before attending the psychiatrist specialist out-patient clinic for planned medication changes, Mr C made an impulsive attempt on his life by trying to jump from height at home after arguing with his wife – this was stopped and he was hospitalised for two weeks.

Although the patient improved on Venlafaxine, he was again admitted to the in-patient unit several months later for hypomanic features. His diagnosis was revised to Bipolar Disorder and he was switched to a mood stabiliser. Upon discharge, Mr C's care was continued at a psychogeriatric day hospital for maintenance and monitoring. The patient's family reported that he appeared aloof and apathetic despite his apparent improvement. Interestingly, the patient also exhibited frontal lobe impairment signs– such as problems in

abstract thinking and verbal fluency – on neurocognitive assessment. In view of the disease course and cognitive findings, a SPECT brain perfusion scan was arranged and showed bilateral moderate frontal hypoperfusion characteristic of the frontal variant of frontotemporal dementia (Figure 5). An additional note was that Mr C's wife, on learning about the patient's "organic brain condition", fared better with less guilt and stress as his spouse and main caregiver.

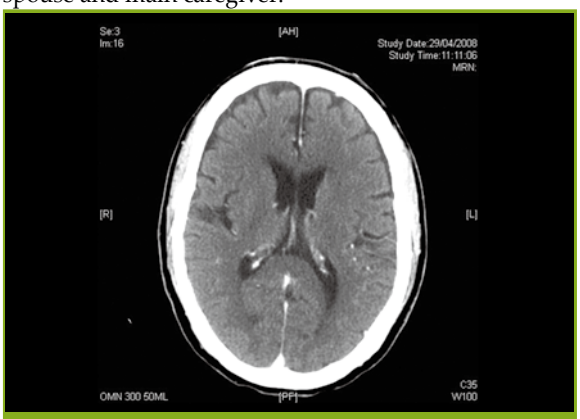

Figure 4: Normal Computerized Tomography (CT) brain scan (with contrast) of Mr C despite the severity of the clinical symptoms

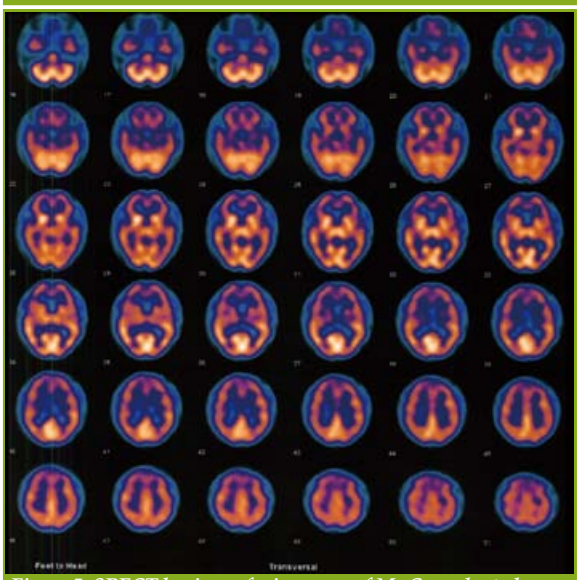

Figure 5: SPECT brain perfusion scan of Mr C conducted shortly after the negative CT brain scan, which again showed typical frontal-temporal perfusion deficits of FTD

Comments: As aforementioned, the behavioural variant frontotemporal dementia (bvFTD) describes a clinical syndrome with personality change and behavioural disturbances, such as repetitive motor behaviour, idiosyncratic collecting and hoarding and changed eating habits. Neurocognitive assessments tend to show impaired planning and judgement, inattention and easy distractibility. It is common for FTD to be misdiagnosed as a psychiatric disorder due to the mimicry of its early symptoms to those of affective or other mental disorders (Woolley et al., 2007). The fact that CT brain scans remain unremarkable until an advanced disease stage does not help in early diagnosis.

It is most imperative to discover and institute effective treatments in these patients. Currently pharmacotherapy focuses on symptomatic treatment (Rabinovici & Miller, 2010); with serotonin-modifying medications having the strongest biological rationale (Huey, Putnam, & Grafman, 2006) while some advocate the use of Trazodone (Lebert, Stekke, Hasenbroekx, & Pasquier, 2004). Atypical antipsychotics should be used with caution as FTD patients are sensitive to extrapyramidal side effects and increased mortality has been reported in the elderly (Curtis & Resch, 2000; Fellgiebel, Muller, Hiemke, Bartenstein, & Schreckenberger, 2007; Pijnenburg, Sampson, Harvey, Fox, & Rossor, 2003). Reed et al. (2004) reported negative results in treatment with bromocriptine in a small controlled trial. With no cholinergic deficits in FTD, the role and benefits of acetylcholinesterase inhibitors become debatable (Hansen, Deteresa, Tobias, Alford, & Terry, 1988; Meier-Ruge, Iwangoff, & Reichlmeier, 1984; Yates, Simpson, Maloney, & Gordon, 1980). Memantine is found to improve the neuropsychiatric inventory (NPI) scores with good tolerability in FTD clients and larger randomised controlled studies are underway (Boxer et al., 2009; Diehl-Schmid, Forstl, Perneczky, Pohl, & Kurz, 2008; Reisberg et al., 2003; Swanberg, 2007).

Notwithstanding the discussion on medications, the first-line therapy for behavioural disturbances in FTD should be non-pharmacological as current drug therapies are associated with serious adverse effects but are only modestly effective at best (Merrilees, 2007; Perry & Miller, 2001; Talerico & Evans, 2001). This comprises carer education and support, interventions to minimise undesired behaviour and a multidisciplinary care plan (Talerico & Evans, 2001). Modalities such as speech therapy, communication enhancement devices and sign language are helpful, and structured physiotherapy and exercise programmes are recommended. Carers should be targeted in treatment plans too; with FTD being a less well-known diagnosis, carers have difficulty enlisting support and experience high levels of caregiver burden (Riedijk et al., 2006). With a comprehensive approach including psychosocial interventions, support groups and targeted education programmes (Weintraub & Morhardt, 2005), this buoys caregivers who will adapt better over time (Riedijk et al., 2006).

Conclusion

A useful learning point is for the practitioner to be vigilant of the possibility of FTD in clients first presenting in their fifties or sixties with psychiatric symptoms or behavioural disturbances. Since the condition is fairly common and yet underestimated, delay in diagnosis implies that timely treatment cannot be instituted, this both affects patient care directly and poses considerable distress to carers. It is recommended that baseline cognitive assessments should be performed at the initial interview or during early consultations as these often prove to be invaluable in the evaluation of the patient's neurocognitive profile. Neuroimaging studies such as functional brain imaging (SPECT, PET scans) can aid in establishing the diagnosis of FTD, however not all clinicians have access to these tools. There is a dire need to develop or modify existing diagnostic criteria to help identify FTD patients at an early stage; and possible measures would include

devising less stringent clinical diagnostic criteria and increasing the role of neurocognitive assessments and neuroimaging in the diagnosis of FTD (Ghosh et al., 2013).

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A Day Out in Sheung Wan

Dr Dominic TS LEE

MRCPsych, FHKAM (Psych)

Psychiatrist in Private Practice

Dr Dominic TS LEE

Located in the intricate weavings of endless streets of Hong Kong lies the neighbourhood of Sheung Wan, a small yet lively neighbourhood, set away from the bustling of the busy streets of Hong Kong. The cover of this issue depicts one of the finest areas in Hong Kong where tradition meets modernity and East meets West. Naturally relaxing and punctuated with shops of fun, this is also a rapidly developing area which could be modernised in a few years' time. Whether you are a food junkie or a shopping enthusiast, it is now high time to visit and enjoy a hidden gem that may be gone before long.

One of the best spots to start a tour of Sheung Wan is to enjoy a tranquil and refreshing breakfast at the Cafe Deadend. A glass of freshly squeezed orange juice and a basket of artisan bakery from the Po's Atelier is a sure way to quiet down the spirit. If you want a full breakfast, try the poached eggs with prosciutto & rocket on AOP toast. Don't forget to post a photo on your Facebook or Instagram and earn a few likes.

After the breakfast, a stroll along the Po Hing Fong and Tai Ping Shan Street area (or PoHo area as it is commonly called) will open windows of many possibilities. There are lifestyle shops, boutiques, galleries, florists (try visiting the Tallensia Floral Art store), old-style barber shops, and second hand shops (much like flea markets), allowing one to slowly explore these fascinating shops, moving from one to the other. Whether you are befriending the cats in the antique shops or sitting on the stairs to breathe in the relaxing atmosphere, it will leave you with memories to keep forever.

When I was there, I always liked to visit the pottery shop on Po Hing Fong. The shop-cum-workshop has an amazing collection of on-site made cups, bowls, vases and even lamps. Many of my female doctor friends have already enrolled at the florist class in the Tallensia, and I always envy their works posted on social media. For those who enjoy cooking, I would suggest you to visit Yuan Heng spices shop on 19 Tung Street, whom sells fresher and higher quality spices than your usual supermarket spices.

If you find yourself guilty for not connecting with medicine for a whole morning, you may redeem yourself by visiting the Museum of Medical History, which is only a flight of steps away from the Cafe Deadend. Along the same vein, it is hard to believe that it was only a century before that a plague broke out at Tai Ping Shan Street, for the same reason that the street was named.

Next door to Yuan Heng is a silversmith who proudly places his finished products behind the windowsill for all to see. There are quite a few artisan shops in the area where you may find treasures of your liking. A walk further west will lead you to the Tung Wah Hospital, around which you should see a few more galleries and boutiques. I find it fun to pop in and explore whatever art exhibits are there, and I particularly enjoy the Para Site Gallery which provides a venue for local artists to display their works. Next door is the Lomography store, a mecca for those who prefer photography off the beaten track.

Around the corner, on New Street, lies the Shelter Lounge which is one of the most relaxing areas to enjoy a cup of tea after window shopping. For a Hong Kong style lunch, there is no better way but to head to For Kee Restaurant to try their Curry beef sandwich or the Pork Chop rice. Do avoid the lunch hours as there is always a queue there.

After lunch, I like to cross the Queen's Road and explore another patch of Sheung Wan that is ever so mesmerising. My first stop is the Barista Jam, located on Jervois Street, where I enjoy my post-lunch expresso. Barista Jam is one of the first and surely the most professional barista and artisan cafe in town.

On Jervois Street, Wing Lok Street and Bonham Strand lies a few dozen shops that one can leisurely explore: traditional Chinese medicine shops, French grocery, Chinese tea shops, noodle shop, bird nest shops, just to name a few. One of my favourite shops is the old-style rice grocery, Sun Hing. There they still use the old-style barrels and bags to sell rice and cooking oil. Ask them for a catty or two of the best quality rice and you will never want to shop rice from supermarket again.

Feel tired from the walking again? On Hillier Street, you can find Seng Kee, which is infamous for their fish congee and beef balls. Close to Seng Kee is La Rotisserie where you can buy a French roast chicken home for dinner. However, if you are in a mood to cook a meal in the evening, you may like to visit Fusion Gourmet where you can find Australian waygu beef steak, American Iberico pork chops, New Zealand lamb rack and mussels. Fusion Gourmet also stocks chilled fish, like sea bream, kept fresh with acupuncture.

The possibilities are simply endless in Sheung Wan. I have only just highlighted the shops that I am familiar with, but it would be equally fascinating to explore and identify other shops, diners or sites that you and your family enjoy. Hong Kong is a fast-paced and stressful city, and as medical doctors we live a life that is even more stressful than average Hongkongers. Finding a day to get out of your routine life and submerging yourself into a new neighbourhood is a good way to unwind. A small neighbourhood like Sheung Wan requires little research, and because of its richness in history and diversity, you are very likely to discover something that is much to your liking.

Hong Kong Parkinson's Disease Foundation
香港柏金遜症基金

Annual Scientific Meeting 2014

Update in Management of Parkinson's Disease

Date: 6th September 2014 (Saturday)

Time: 2:00pm – 5:00pm

Venue: Function Room, 3/F, South Tower, YMCA of HK
41 Salisbury Road, Tsim Sha Tsui, Kowloon

Chairpersons: Dr. Mandy Au-Yeung & Ms. Sally Liu

Opening Address	
Diagnosis of Parkinson's Disease	Dr. Helen Yip
MIGB Scintigraphy	Dr. TK Au Yong
Tea Break	
Cognitive Assessment and Treatment for Parkinson's Disease: OT Perspective	Mr. Marko Chan
Occupational Lifestyle Redesign for Patients with Parkinson's Disease	Ms. Abby Chau
Closing Remarks	

Registration Fee: Free Admission

150 seats are available on a first come first served basis. CME & CPD points will be accredited and certificate will be provided. Please register online www.hkpdf.org.hk/activity_tc.php.

For Enquiry

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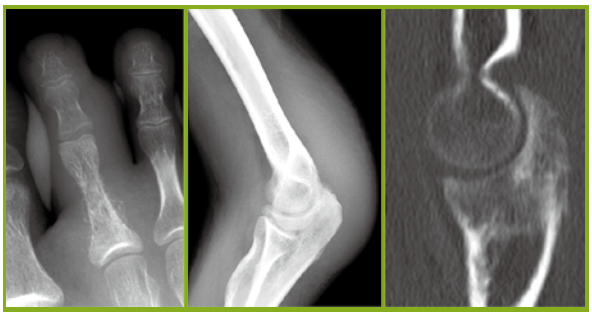
A Young Lady with Insidious Onset of Multiple Bone Pain

Dr KC LAM

MBBS, FRCR

Department of Radiology, Queen Mary Hospital, Hong Kong



A 20-years-old lady complained of insidious onset of right second toe pain for 5 months. Subsequently she also complained of left elbow pain. There was no history of injury. X-rays of right foot and left elbow were performed.

Questions:

1. What were the imaging findings?
2. What was the most likely diagnosis?
3. How would you further investigate and manage the patient?

(See P.36 for answers)

5th 5-Continent-Congress

11th – 13th December, 2014 • Hong Kong
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LASERS AND AESTHETIC MEDICINE

FACULTY

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Robert Weiss (USA)
Flora Xiang (China)
Chi-keung Yeung (Hong Kong)

WORKSHOP HIGHLIGHTS

- Laser
- Botulinum Toxin
- Fillers
- Body Contouring

IMPORTANT TIMELINES

Deadline for Abstract Submission: 31 July 2014
Deadline for Early Bird Registration: 31 August 2014

VENUE

Hong Kong Convention and Exhibition Centre
1 Expo Drive, Wanchai, Hong Kong

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Public Talk for Bowel Diseases

The Public Talk for Bowel Diseases was held at the Federation Lecture Hall on 14 June 2014. It was our pleasure and privilege to invite Dr Benjamin CY WONG, Specialist in Gastroenterology and Hepatology, who delivered a talk on "Irritable bowel syndrome" and Dr Ting-kin CHEUNG, Specialist in Gastroenterology and Hepatology, who delivered a talk on "Chronic constipation". The participants were interested to learn about the eating habit to stay a healthy digestive system. Their active questions and engagement in the talk helped mark a fruitful and interactive event. The event concluded with Dr Mario CHAK, Hon. Secretary of the Federation, thanking the two speakers with presentation of souvenirs.

Society News

Hong Kong Clinical Psychologists Association (HKCPA)

Clinical Psychologists have been serving the Hong Kong community since the early 1970s. Today, slightly over 400 clinical psychologists are practising in Hong Kong, mostly in government departments, public hospitals, NGOs, and universities. Most clinical psychologists work with people with health, emotional and behavioural issues, while others do research and training. Referrals often come from medical and health professionals, social workers, courts and other authorities. More clinical psychologists are getting into private practice and accept self-referrals.

HKCPA, established in 1980, is a trade union of qualified clinical psychologists. Today, we have 200 active members. Nearly all are also members of the much larger Hong Kong Psychological Society's Division of Clinical Psychology (DCP), which is a professional and learned society. Together, HKCPA and DCP represent about 90 percent of all qualified clinical psychologists in Hong Kong. We work closely for the integrity and development of the profession while serving the community through public education.

We are most delighted to have become Associate Member of FMSHK. We are deeply thankful for those who have supported our application. We are looking forward to contribute to FMSHK as well as to share our knowledge and mission with you.

Ephraem TSUI, BA, MSc, PhD, RCP (HKPS)
Chairman, HKCPA (2014-2015)
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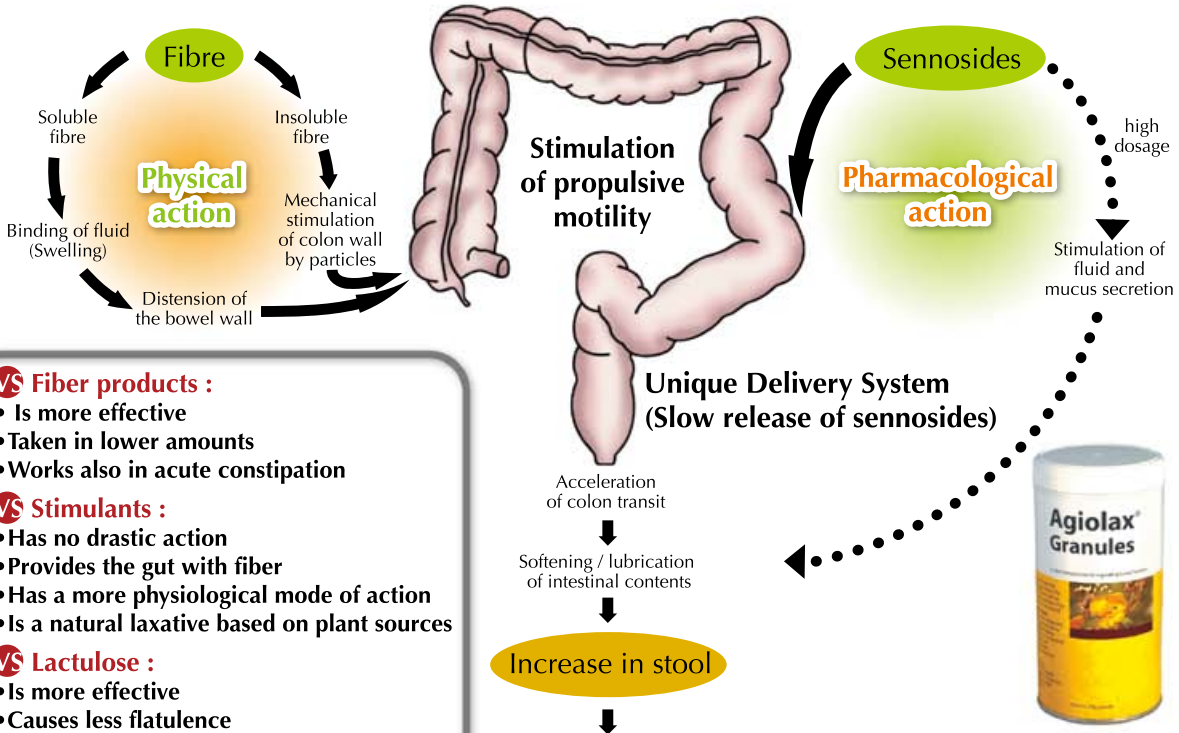

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
					★ HKMA Shatin Doctors Network – Male LUTS: Beyond BPH Management 1	2
★ HKMA Dragon Boat Fun Day 2014 3	4	★ HKMA Council Meeting ★ FMSHK Officers' Meeting 5	6	7	8	9
10	11	12	★ Hong Kong Neurosurgical Society Monthly Academic Meeting – CSF hydrodynamics and primary syringomyelia ★ HKMA Kowloon City Community Network - Importance of Muscle Training for Adults ★ HKMA Central, Western & Southern Community Network - A Review on Onychomycosis 13	★ HKMA Kowloon East Community Network - Sarcopenia in Elderly ★ HKMA Hong Kong East Community Network - The Latest Scientific Evidence of Proteins to Infant and Childhood Development ★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Management Update on Colorectal Cancer 14	★ HKMA Yau Tsim Mong Community Network - Diabetic Nephropathy – A Call for Early Effective Intervention 15	16
17	18	19	20	★ HKMA New Territories West Community Network - New Once-Daily ICS/LABA Regimen on Asthma Management ★ FMSHK Executive Committee Meeting ★ FMSHK Council Meeting 21	22	★ KECN/HKCFP/UCH – CME Course for Health Personnel 2014 - Update on Gynaecological Oncology 23
24	25	★ HKMA Kowloon West Community Network - The Role of the Incretin Axis in Type 2 Diabetes Management 26	27	★ HKMA Hong Kong East Community Network - Principles of Exercise Prescription ★ HKMA Kowloon East Community Network - Effective Use of Antidepressants in the Management of Chronic Pain ★ HKMA New Territories West Community Network - Hypertension Guidelines Update 2014 28	★ HKMA Yau Tsim Mong Community Network - Common Cause of Lower Abdominal Pain in Women 29	★ HKMA Yau Tsim Mong Community Network - Certificate Course on Bringing Better Health to Our Community (Session 4) 30
31						

Calendar of Events

Date / Time	Function	Enquiry / Remarks
1 FRI 1:00 pm	**HKMA Shatin Doctors Network – Male LUTS: Beyond BPH Management** Organiser: HKMA Shatin Doctors Network, Chairman:Dr. MAK Wing Kin, Speaker: Dr. HOU See Ming, Venue:Jasmine Room, Level 2, Royal Park Hotel, Shatin, Hong Kong	Mr. Wilson HON Tel: 2965 1311 Fax: 2976 0778 1 CME Point
3 SUN 3:00 pm	**HKMA Dragon Boat Fun Day 2014** Organiser: The Hong Kong Medical Association, Chairman:Dr. YAM Chun Yin, Abraham , Venue:Sai Sha Wan, Sai Kung	Miss Nadia HO Tel: 2527 8285
5 TUE 8:00 pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman:Dr. TSE Hung Hing , Venue:HKMA Head Office, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
13 WED 7:30 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting – CSF hydrodynamics and primary syringomyelia** Organiser: Hong Kong Neurosurgical Society, Chairman: Dr Larry WONG, Speaker: Dr CHOW Shuk Wan, Joyce, Venue: Seminar Room, Ground Floor, Block A, Queen Elizabeth Hospital	Dr. LEE Wing Yan, Michael Tel: 2595 6456 Fax: 2965 4061 1.5 CME Points
1:00 pm	**HKMA Kowloon City Community Network - Importance of Muscle Training for Adults** Organiser: HKMA Kowloon City Community Network, Chairman:Dr. CHIN Chu Wah, Speaker: Dr. CHAN Hoi Chung, Samuel, Venue:Sportful Garden Restaurant [陶源酒家] 2/F, Site 6, Whampoa Garden, Wonderful Worlds of Whampoa, 8 Shung King Street, Hung Hom	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
1:00 pm	**HKMA Central, Western & Southern Community Network - A Review on Onychomycosis** Organiser: HKMA Central, Western & Southern Community Network, Chairman:Dr. LAM Ming Yuen, Speaker: Dr. CHUNG Chun Kin, Alex, Venue:HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
14 THU 1:00 pm	**HKMA Kowloon East Community Network - Sarcopenia in Elderly** Organiser: HKMA Kowloon Eat Community Network, Chairman:Dr. AU Ka Kui, Gary, Speaker: Dr. YIP Wai Man, Venue:Lei Garden Restaurant [利苑酒家] Shop No. L5-8, apm, Kwun Tong, No. 418 Kwun Tong Road	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
1:00 pm	**HKMA Hong Kong East Community Network - The Latest Scientific Evidence of Proteins to Infant and Childhood Development** Organiser: HKMA Hong Kong East Community Network, Chairman:Dr. YOUNG Ying Nam, Dominic, Speaker: Dr. NG Yin Ming, Venue:The HKMA Wanchai Premises,5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Management Update on Colorectal Cancer** Organiser: Hong Kong Medical Association,Hong Kong Sanatorium & Hospital, Speaker: Dr. Liu King Yin, Rico, Venue:Function Room A, HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 Fax: 2865 0943 1 CME Point
15 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network - Diabetic Nephropathy – A Call for Early Effective Intervention** Organiser: HKMA Yau Tsim Mong Community Network, Chairman:Dr. LEE Wai Lun, Speaker: Dr. HO Chung Ping, MH, JP, Venue:Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
21 THU 1:00 pm	**HKMA New Territories West Community Network - New Once-Daily ICS/LABA Regimen on Asthma Management** Organiser: HKMA New Territories West Community Network, Chairman:Dr. CHUNG Siu Kwan, Ivan, Speaker: Dr. TAI Kian Bun, Venue:Plentiful Delight Banquet [元朗喜尚嘉喜酒家] 1/F., Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
7:00 pm	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Veune: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 pm	**FMSHK Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Veune: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
23 SAT 1:30 pm	**KECN/HKCFP/UCH – CME Course for Health Personnel 2014 - Update on Gynaecological Oncology** Organiser: HKMA Kowloon East Community Network & Hong Kong College of Family Physicians & United Christian Hospital, Chairman:Dr. Danny MA, Speaker: Dr. CHENG Oi Ling, Edith, Venue:Lecture Theatre, G/F, Block P, United Christian Hospital, 130 Hip Wo Street, Kwun Tong, Kowloon	Ms. Polly TAI Tel: 3513 3430 Ms. Cordy WONG Tel: 3513 3087 Fax: 3513 5505 1.5 CME Points
26 TUE 1:00 pm	**HKMA Kowloon West Community Network - The Role of the Incretin Axis in Type 2 Diabetes Management** Organiser: HKMA Kowloon West Community Network, Chairman:Dr. LEUNG Kin Nin, Kenneth, Speaker: Dr. TSANG Man Wo, Venue:Crystal Room I - III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
28 THU 1:00 pm	**HKMA Hong Kong East Community Network - Principles of Exercise Prescription** Organiser: HKMA Hong Kong East Community Network, Chairman:Dr. YIP Yuk Pang, Kenneth, Speaker: Dr. CHAN Hoi Chung, Samuel, Venue:The HKMA Wanchai Premises,5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
1:00 pm	**HKMA Kowloon East Community Network - Effective Use of Antidepressants in the Management of Chronic Pain** Organiser: HKMA Kowloon East Community Network, Chairman:Dr. MA Ping Kwan, Speaker: Dr. CHAU Shuk Yi, Lucia, Venue:East Ocean Seafood Restaurant [東海海鮮酒家] Shop 137, 1/F, Metro City Plaza 3, 8 Mau Yip Road, Tseung Kwan O, Kowloon	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
1:00 pm	**HKMA New Territories West Community Network - Hypertension Guidelines Update 2014** Organiser: HKMA New Territories West Community Network, Chairman:Dr. LEE Huen, Speaker: Dr. TONG Chun Yip, Peter, Venue:Plentiful Delight Banquest [元朗喜尚嘉喜酒家] 1/F, Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point
29 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network - Common Cause of Lower Abdominal Pain in Women** Organiser: HKMA Yau Tsim Mong Community Network, Chairman:Dr. LEUNG Chi Shan, Fiona, Speaker: Dr. LAM Siu Keung, Venue:Jade Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Miss Hana YEUNG Tel: 2527 8285 Fax: 2865 0943 1 CME Point

Date / Time	Function	Enquiry / Remarks
30 *SAT* 1:00 pm	**HKMA Yau Tsim Mong Community Network - Certificate Course on Bringing Better Health to Our Community (Session 4)** Organiser: HKMA Yau Tsim Mong Community Network, Speakers: Dr. YEUNG Wai Hong & Dr. HO Chin Hung, Venue:Block M, Lecture Theatre, Queen Elizabeth Hospital, 30 Gascoigne Road, Kowloon, Hong Kong	Ms. Noel AU YEUNG Tel: 2958 8608 Ms. Mandy LEUNG Tel:2958 8613 Miss Hana YEUNG Tel:2527 8285 Fax: 2958 8614

Upcoming Meeting

24-28/9/2014	**CUHK Sleep 2014 Comprehensive Polysomnography Workshop, Psychotherapy for Insomnia Workshop and Conference** Organisers: Department of Psychiatry and Paediatrics, The Chinese University of Hong Kong, Chairmen: Prof. Wing Yun Kwok and Prof. Albert Martin Li, Speakers: Prof. Phyllis C Zee and Prof. Mary A. Carskadon, Venue: Postgraduate Education Centre, Prince of Wales Hospital, Shatin, Website: http://www.pae.cuhk.edu.hk/SLEEP2014/, Enquiry: Ms. Mandy Yu Tel: 2636 7593 Fax:2647 5321
4-5/10/2014	**The 8th Hong Kong Allergy Convention (HKAC 2014) – Novel Revelations in Allergies** Organiser: Hong Kong Institute of Allergy, Chairman: Dr Robert Tseng, Venue: Hong Kong Convention and Exhibition Centre Online Registration: www.allergy.org.hk, Enquiry: HKAC 2014 Secretariat Tel: 2559 9973 Fax: 2547 9528, CME/CPD points application in progress

Certificate Course on
Clinical Ophthalmology 2014

Date : 6 October 2014 – 10 November 2014 (Every Monday)
Time : 7pm – 8:30pm
Venue : Lecture Hall, The Federation of Medical Societies of Hong Kong,
4/F Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Enquiry: The Secretariat of The Federation of Medical Societies of Hong Kong
Tel.: 2527 8898 Fax: 2865 0345 Email: info@fmshk.org

The course details will be announced very soon

Rental Fees of Meeting Room and Facilities at The Federation of Medical Societies of Hong Kong

(Effective from October 2009)

Venue or Meeting Facilities	Member Society (Hourly Rate HK$)			Non-Member Society (Hourly Rate HK$)		
	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays
Multifunction Room I *(Max 15 persons)*	150.00	105.00	225.00	250.00	175.00	375.00
Council Chamber *(Max 20 persons)*	240.00	168.00	360.00	400.00	280.00	600.00
Lecture Hall *(Max 100 persons)*	300.00	210.00	450.00	500.00	350.00	750.00
Non-Peak Hour: 9:30am - 5:30pm Peak Hour: 5:30pm - 10:30pm						
LCD Projector	500.00 per session					
Microphone System	50.00 per hour, minimum 2 hours					

HONG KONG SANATORIUM & HOSPITAL

LI SHU PUI SYMPOSIUM 2014

CURRENT DEVELOPMENTS IN MEDICAL PRACTICE

Date : Sunday, 14 September 2014
Venue : Ballroom, JW Marriott Hotel Hong Kong

08:50 – 09:00	Welcome		Dr. Walton LI
09:00 – 09:30	**Keynote Lecture 1:** **A Decade of Advances in Percutaneous Coronary Intervention**		Dr. Vincent KWOK
	Symposium 1 Orthopaedic / Traumatology	Chairperson	Dr. Stephen WU | Dr. Michael LI
09:30 – 09:45	Cervical Radiculopathy and Myelopathy – Diagnosis and Treatment		Prof. Keith LUK (HKU)
09:45 – 10:00	Managing Osteoporotic Fractures: Typical and Atypical		Dr. MAK Kan Hing
10:00 – 10:15	Recent Updates on Arthroscopic Surgery		Dr. Jimmy WONG
10:15 – 10:30	The Recent Advances in Hip Reconstruction		Dr. TANG Wai Man
10:30 – 10:40	Q & A		
10:40 – 11:00	Coffee Break		
	Symposium 2 New Developments	Chairperson	Dr. Raymond LIANG | Dr. WONG Wai Sang
11:00 – 11:15	The Armamentarium of Urology		Dr. Steve CHAN
11:15 – 11:30	Neurology for the Non-Neurologist		Dr. TSOI Tak Hong
11:30 – 11:45	Diet for the Irritable Bowel		Ms. June CHAN
11:45 – 12:00	Why should I see an Oncologist?		Dr. Rico LIU
12:00 – 12:10	Q & A		
12:10 – 13:00	**Li Shu Pui Lecture** **Update on Infectious Diseases and Implications for Clinical Practice**	Chairperson	Dr. Raymond YUNG **Prof. Peter V CHIN-HONG (UCSF)**
13:00 – 14:00	Lunch		
	Symposium 3 Cardiology	Chairperson	Dr. Elaine CHAU | Dr. AU-YEUNG Kai Ming
14:00 – 14:15	Update on Interventional Cardiology 2014		Dr. Duncan HO
14:15 – 14:30	How to Manage Atrial Fibrillation in 2014?		Dr. Kathy LEE
14:30 – 14:45	Device Therapy in Cardiology		Dr. Elaine CHAU
14:45 – 15:00	Minimally Invasive Heart Valve Surgery		Dr. Timmy AU (QMH)
15:00 – 15:10	Q & A		
15:10 – 15:40	**Keynote Lecture 2 : Precision Knee Arthroplasty Surgery**		Dr. Stephen WU
15:40 – 16:00	Coffee Break		
	Symposium 4 GP Forum	Chairperson	Dr. LAI Kar Neng | Dr. PANG Siu Leung
16:00 – 16:15	Oncology Made Simple		Dr. Stephen CHAN (CUHK)
16:15 – 16:30	Essential Neurology		Dr. Patrick LI
16:30 – 16:45	Recent Developments in "Imaging"		Dr. Gladys LO
16:45 – 17:00	Assisted Reproduction – Current Status		Prof. HO Pak Chung (HKU)

**Content is subject to change without prior notice*

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Answers to Radiology Quiz

1. Fusiform soft tissue swellings were seen at the right second toe and left elbow. There were fluffy spiculated periosteal reaction and permeative changes at the diaphysis of the proximal phalange of the right second toe. The anterior fat pad of the left elbow was elevated. The cortex of the left olecranon was ill-defined, which was proved in the subsequent CT scan. A rim-enhancing collection was seen at the extensor compartment of the left arm (not shown).

2. Imaging features favoured an aggressive process affecting multiple bones. Osteomyelitis (pyogenic or tuberculosis) is a readily treatable disease that had to be considered. A neoplastic process would be less likely since there was polyostotic involvement.

3. Incision and drainage of the left elbow collection and debridement were performed. Microbiological culture and PCR of the surgical specimens confirmed the presence of mycobacterial tuberculosis. There were pulmonary and hepatic involvements as well. The patient was therefore diagnosed to have disseminated tuberculosis and was put on anti-tuberculous drugs.

Dr KC LAM
MBBS, FRCR
Department of Radiology, Queen Mary Hospital, Hong Kong

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1. IMS Health 2011
2. HKAPI data 2013
3. Data on file

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