An Update on Prostate-Specific Antigen and Early Detection of Prostate Cancer

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Medical Management of renal calculi

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Point-of-care diagnostic test for seasonal influenza

by Dr. TAM Yat Hung
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My dearest HKMA colleagues, the most beautiful season in Hong Kong, Autumn is coming.

In the recent months, the whole medical field is very busy (if not chaotic!) Let us enjoy the quotes about the art of busy and relaxation together.

"Beware the barrenness of a busy life."
~ Socrates (classical Greek philosopher, 470-399BC)

"Technology can be our best friend, and technology can also be the biggest party pooper of our lives. It interrupts our own story, interrupts our ability to have a thought or a daydream, to imagine something wonderful, because we're too busy bridging the walk from the cafeteria back to the office on the cell phone."
~ Steven Spielberg (American director, producer, screenwriter and editor, 1946-)

"Being busy does not always mean real work. The object of all work is production or accomplishment and to either of these ends there must be forethought, system, planning, intelligence, and honest purpose, as well as perspiration. Seeming to do is not doing."
~ Thomas A Edison (1847-1931, American inventor and businessman)

"If you want to conquer fear, don’t sit home and think about it. Go out and get busy."
~ Dale Carnegie (American writer and lecturer, 1888-1955)

"Together with a culture of work, there must be a culture of leisure as gratification. To put it another way: people who work must take the time to relax, to be with their families, to enjoy themselves, read, listen to music, play a sport."
~ Pope Francis (the 266th and current Pope, 1936-)

"I worry from the moment I take a job. I worry about how I'm going to do it, if I can do it... Then I walk on set and the director says, "Roll", and all of a sudden, all of it disappears and it's all going to do it, if I can do it... Then I walk on set and the director interrupts our ability to have a thought or a daydream, to imagine something wonderful, because we're too busy bridging the walk from the cafeteria back to the office on the cell phone."
~ Jack Nicholson (American actor and film maker, 1937-)

"With every year of playing, you want to relax one more muscle. Why? Because the more tense you are, the less you can hear."
~ Yo-Yo Ma (Chinese-American cellist, 1955)

"I am very lucky because when I come home, I have a completely normal life. I can relax, playing golf, fishing – doing what I want. I know when I finish a tournament, I am going to relax at home."
~ Rafael Nadal (Spanish professional tennis player, currently world No.4, 1988)
An Update on Prostate-Specific Antigen and Early Detection of Prostate Cancer

What is Prostate-Specific Antigen (PSA)

PSA is a protease (human kallikrein peptidase 3) primarily produced by prostate luminal epithelial cells but leaks into the blood stream in very small amount. It liquefies semen after ejaculation and facilitates reproduction. Its production is androgen regulated. Serum PSA level rises slightly for about 2 days after each ejaculation and its measurement should be done more than 2 days after latest ejaculation (1). Ectopic expression of PSA have been reported in malignant breast cancer tissue, adrenal and renal carcinomas and is not clinically relevant.

History of PSA in Detection of Prostate Cancer (CAP)

Although PSA was discovered in 1979, its use as a tumor marker was only explored in late 1980s and 1990s. Since PSA is organ specific, its elevation in serum may signify prostate diseases. However, PSA is not disease specific. It is thought that disruption of prostate architecture causes release of PSA into blood stream and it can happen in various prostate diseases such as benign prostate hyperplasia (BPH), prostatitis and prostate cancer (CAP). It is also elevated after vigorous prostate manipulation such as prostate massage, needle biopsy of prostate and urethral instrumentation such as cystoscopy. It may also be elevated in urinary tract infection (UTI) and retention of urine. Gentle digital rectal examination (DRE) of the prostate usually would not result in significant elevation.

Historically, persistently raised serum PSA level is associated with increased risk of prostate cancer and normal serum concentration in men 50-80 years old without prostate disease ranges between 1.0-4.0 ng/ml (2). On a per-gram basis, cancerous prostate tissue would not produce higher concentration of PSA than non-cancerous tissue. However, elevation of PSA in CAP is thought to be due to the PSA produced by cancerous tissue is more resistant to serum degradation and uncontrolled growth of CAP eventually would result in bigger cell mass with more PSA production. It is also likely that more PSA is being released from CAP tissue due to more serious disruption of its internal architecture.

Contribution of PSA in Early Detection of CAP

Before the PSA era, CAP was detected by DRE. The old serum marker of prostatic acid phosphatase was only elevated in late stage and was not sensitive enough for early detection although it was very disease specific. DRE alone only can detect palpable tumors resulting in most cases being advanced or metastatic when diagnosed. Different imaging techniques were rather nonspecific. With PSA-based detection, nowadays, most cases of CAP are diagnosed much earlier when the lesion is still clinically nonpalpable (stage T1c) and organ confined. More cases are thus amendable to curative treatment such as surgery or radiotherapy. There has been 40% reduction in CAP specific mortality rate (3).

Limitation of PSA in CAP Detection

Historically, PSA level of 4 ng/ml is regarded as the cutoff point of normal (1). Trans-rectal ultrasound directed prostate needle biopsy (TRUS-Bx) is triggered when there is abnormal serum PSA and/or suspicious DRE. PSA screening is indicated in man > 50 year of age when significant increase in CAP risk starts (Hong Kong Cancer Registry 2013). People with positive family history should have their first serum PSA checked about a decade earlier. However, since PSA is not disease specific and that prostate pathology especially BPH is also very common in that target age group, there is a large overlap between benign and malignant conditions. Using PSA alone as an indication would result in over employment of TRUS-Bx resulting in increased cost and procedure related morbidity and even mortality. In general, PSA trigger at 4 ng/ml would result in at least 40% of false positive. On the contrary, with more and more cores being taken during TRUS-Bx (from previously 6 cores up to presently 10 cores or more), taking 4 ng/ml as the trigger point would result in significant number.
of false negative missing some of the more dangerous high risk CAP especially in high CAP prevailing countries such as the United States. Therefore, It is said that for a single PSA value, there is no really absolutely safe cutoff point although the lower the safer. Meanwhile, different countries and different medical centers may use different cutoff point and in the U.S., PSA value of 2.5-3 ng/ml is generally used as a threshold to recommend prostate biopsy (4). In view of these not yet satisfactory sensitivity and specificity results of using PSA alone, there have been calls for more accurate screening tools and more refined diagnostic trigger points.

Variations in Serum PSA Level

To compound the diagnostic problem more, interpretation of a single PSA value is affected by variations in PSA value in different clinical situations. Malignant disease-free PSA level varies with size of the prostate especially when it is affected by BPH. Higher PSA level may just indicate bigger prostate size. The implication for this is for a similar PSA level, smaller prostate carries higher chance of CAP. Level of PSA also varies with different ethnic groups. In the U.S., African-American has the highest baseline normal PSA level. Aging would increase the PSA level gradually and age adjustment is recommended although there is no exact consensus on any cutoff point for different age group in Chinese. Body built can also affect its level. More obese patients may have lower PSA level due to haemodilution.

Improvement Measures to Increase PSA Efficacy in Detecting CAP

1. PSA Density

Bigger prostate in general has higher baseline PSA. Cross-sectional study suggested that PSA increases 4% per ml of prostate volume (5). PSA level divided by prostate volume is termed PSA Density (PSAD). PSAD of 0.15 or greater was recommended for TRUS-Bx in men with PSA 4-10 ng/ml with normal DRE (PSA > 4 ng/ml with normal DRE and PSA > 10 ng/ml would need a biopsy usually) (6). However, different studies differ in their PSAD cutoff point. Some workers recommended using the transitional zone volume of the prostate alone instead of the whole prostate volume since it is the part mostly affected by BPH (7). Meanwhile, there is still no common consensus on exact PSAD cutoff point.

2. Age Related PSA and PSA Velocity (PSAV)

Older age is associated with higher PSA level (5). It was found that 5% variance on PSA level could be attributed to age alone. In men without BPH, the rate of change in PSA is 0.04 ng/ml per year compared with 0.07 to 0.27 ng/ml per year in men with BPH who are 60-85 years old (8). Change in PSA value with time is called PSAV. Meanwhile, PSAV > 0.75 ng/ml/year over a period of at least 18 month is used to consider biopsy. However, actual cutoff value of PSAV has not been verified in Chinese populations.

3. Free Prostate-Specific Antigen (fPSA)

PSA in blood appears as bound (to serum protein) (complexed PSA) and free (fPSA) forms. There are thus in fact 3 major types of PSA assays: total PSA (tPSA), fPSA and complexed PSA. Total PSA is the most common assay and is usually just referred to as PSA. CAP cells are found to produce more bound form resulting in less percentage of fPSA. Ratio of fPSA/tPSA has been explored as a tool to detect possibility of CAP in patients with PSA value < 10 ng/ml (9). However, there is still no universally accepted fPSA cutoff point and most studies would recommend level between 15-25%.

4. PSA Isoforms

PSA is initially produced in a precursor form called proPSA (pPSA). pPSA is being activated in prostatic lumen to become its active form (active PSA). Due to various cleavage processes caused by protease enzymes, PSA leaked into the blood stream comprises of different isoforms such as intact pPSA; truncated forms of pPSA at different cleavage sites such as [-7] pPSA, [-2] pPSA; BPH-associated PSA (BPSA) which is found to be associated with BPH. Investigations have been performed to study value of various isoforms as additional tumor markers. It was found that CAP in deed can produce significantly higher levels of these truncated forms of proPSA. Meanwhile, study had shown that %[-2] PSA might outperform tPSA and fPSA/tPSA in predicting presence of CAP at biopsy. Prostate Health Index (PHI) is a FDA approved test which applies a mathematical formula to calculate an index to indicate chance of CAP after measuring serum levels of tPSA, fPSA and [-2] PSA. It is indicated for men 50 years or older with tPSA 4-10 ng/ml. In addition, PHI value was also found to be associated with improved prediction rate of high grade (more malignant) CAP that need to be treated. In a recently published local study (10), 569 Chinese men with PSA 4-10 ng/ml and non-suspicious DRE with TRUS-Bx done were retrospectively studied for PSA-based and PHI-based models. Patients were also stratified according to different prostate volume according to DRE and TRUS. Overall, in this selected
Prostatitis although can cause elevation of PSA, antibiotic trial for patients with elevated PSA but without any prostatitis symptoms is not recommended (American Urological Association 2013).

**Issues on CAP Screening**

PSA-based CAP screening has resulted in earlier detection of organ-confined and potentially curable disease. However, asymptomatic CAP is common with advancing age and many of these may not affect the patient even in the long run. Overall, the single most common mortality for all CAP men in fact is not CAP but heart disease. There has been concern on overtreating the much less aggressive cases which may not affect the patients even if left undiagnosed. Screening of CAP in the general population thus poses concern of overdiagnosis. There were 2 major PSA-based screening programs which had given rise to different results. The U.S.-based Prostate, Lung, Colorectal, and Ovarian (PLCO) Cancer Screening Trial had shown no benefit on prostate cancer-specific survival resulting in the U.S. Preventive Services Task Force (USPSTF) to conclude in 2008 that the evidence was insufficient to assess the balance of benefit and harm of screening among men 75 years of age or younger and that screening was not recommended for men 75 years or older (12). However, the European Randomized Trial of Prostate Cancer Screening (ERSPC) had on the contrary reported 21% reduction in prostate cancer-specific mortality amongst those screened (13). Recently, it was found that the patient sample taken in the controlled arm of the U.S. study was somehow “contaminated” resulting in possible statistical fraud making the decision of USPSTF more revocable by the urology community (AUA Annual Meeting 2016). Meanwhile, the American Cancer Society recommends that men with at least a 10-year life expectancy should have an opportunity to make an informed decision about CAP screening beginning at age 50 and before age 50 for higher risk people such as those with positive family history.

There is at present no CAP screening program of any sort in Hong Kong. According to Hong Kong Cancer Registry (2013), CAP was the 3rd commonest cancer in the male but only ranked 5th in cancer mortality signifying the less lethal nature of the disease. Crude mortality rate for CAP was 11.2/100,000 compared to 74.8, 33.7 and 32.6/100,000 for cancers of lung, liver and colorectal respectively. In the same Registry, 1655 new cases of CAP were registered and the youngest patient for CAP was in the 25-35 age group (1 patient). 12 patients were before age of 50 and the rest were > 50 years of age. Number of patients in the 50-55 age group climbed steeply to 41. It is obvious that to our individual patients, PSA screening...
should be considered on individual bases and our patients should be given an informed decision especially when they are 50 years or older including patients who have less than 10 years of life expectancy and they should not be totally ruled out in the screening process. Those with family history should be considered at an earlier age up to a decade younger.

It has to be noted that for the first PSA, no single value is absolutely cancer-free since the initially low PSA may rise with time if there is CAP inside the organ. The question is how often it has to be repeated. If the patient would want to have yearly blood check anyway and if he has no concern about the cost, this author would not discourage the patients to have their PSA checked yearly especially for those with borderline PSA of near 4 ng/ml. For high CAP prevailing countries such as Sweden, PSA has predictive value in that PSA measurements in the 40 years age group had in fact been found to predict the risk of CAP mortality 25 years later (Malmo Preventive Project). 

**CAP Risk According to PSA Levels and DRE Findings in Hong Kong**

Risk stratification according to specific PSA level in any population can only be answered by a general screening program. Such program is basically lacking in Hong Kong and in other Chinese populations. The closest approximation is the study done by Jeremy YC Teoh et al (2015) (14). In their retrospective analysis of 2606 local Chinese men who were referred for TRUS-Bx between year 2000 to 2013, for those who had normal DRE, cancer rates were 8.6%, 13.4%, 21.8%, 41.7% and 85.2% and for those with abnormal DRE, the cancer rates were 12.4%, 30.2%, 52.7%, 80.6% and 96.4% for PSA < 4, 4-10, 10.1-20, 20.1-50 and > 50 ng/ml respectively. It has to be noted that in their study, there were 8.6% biopsy proven CAP even for those whose PSA was < 4 ng/ml. They had found that older age, smaller prostate volume and larger number of biopsy cores, presence of abnormal DRE and higher PSA level were associated with increased risk of CAP detection. They had also concluded that local Chinese men appeared to have lower CAP detection rates when compared to western population. They recommended an individualized approach to the decision of TRUS-Bx.

**Multiparametric MRI (mp-MRI)**

Imaging techniques in any form are not for screening. Recently, MRI using multiparametric technique which includes T2-weighed imaging (T2W), dynamic contrast-enhanced (DCE) imaging, diffusion-weighted imaging (DWI) and magnetic resonance spectroscopy (MRS) have proven to be major improvement with sensitivity and specificity had been reported up to 93% and 98% in detecting and excluding high-grade cancers greater than 0.5 mL in volume (Ukimura et al AUA Annual Meeting 2012). Mp-MRI can detect and grade the suspicious areas in the prostate which would help to decide whether to proceed to TRUS-Bx or not in PSA-suspicious cases and the MRI images can guide the sites of biopsy. Although MRI-guided biopsy had been reported, it is still limited by lack of facilities and high cost and has not been widely practiced.

**Fusion Biopsy**

For practical purpose, most if not all prostate biopsies are still guided by TRUS. In order to make use of the very useful information of mp-MRI, the MRI images can be imported into the TRUS machine by specially designed software so that images of both investigations can be superimposed during the biopsy procedure to target at the more suspicious areas. This is especially useful for the second or third biopsy after previously negative biopsy and for tumors in the more obscured sites of the prostate such as the apex and the anterior lobe. At least 4 such programs are currently approved by the U.S. FDA (15).

**Conclusion**

PSA has revolutionized early detection and treatment of CAP. More organ confined diseases were found for curative treatment. As a result, there has been major refinement in local curative treatment such as open and robotic surgeries and radiotherapy. These had resulted in much reduced CAP specific mortality rate. Current issues would include how to improve the PSA-based diagnostic process, how to avoid unnecessary biopsy procedures, how to stratify risk according to PSA level in different ethnic groups especially Chinese, and how to define less aggressive diseases. Major advance is to be expected in coming years for both PSA-based and non-PSA-based screening and diagnoses. As CAP incidence in Hong Kong had been doubled in recent decade (Hong Kong Cancer Registry), clinicians are expected to update their knowledge on various issues concerning CAP and PSA screening and diagnosis.

**References**

Please indicate whether the following statements are true or false.

1. PSA is an useless substance produced by the prostate.
2. PSA > 4 ng/ml always needs a prostate biopsy.
3. For PSA > 4 ng/ml, multiparametric MRI would be diagnostic.
4. Prostate biopsy is CT (computerized tomography) guided.
5. Alpha-blockers used in BPH reduce PSA value by half at the end of one year.
6. PSA may be elevated in retention of urine.
7. At present, PHI seems to be better than tPSA in avoiding unnecessary biopsy.
8. fPSA/tPSA may be useful in deciding whether to proceed to TRUS-Bx in PSA > 10 ng/ml.
9. PSA < 4 ng/ml carries negligible risk of CAP.
10. Swedish study had shown PSA checked in younger age can predict CAP death 25 years later.

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**Q&A Answers to August 2016**

Spotlight 1 – Obstructive sleep apnoea (OSA) in adult – an overview for general practitioners

Spotlight 2 – PCSK9 Inhibitors – a new treatment option for hyperlipidemia

Medical Management of renal calculi

Introduction
Renal calculi disease, or nephrolithiasis, is common. In the United States, it affects 15% of men and 8% of woman. In Hong Kong, Chan et al reported a random telephone survey on 1,010 people with 25 of them reported history of renal stones, giving a local prevalence of 2% in 2008 (1). Its prevalence seems to be increasing and it might be related to diet and obesity. There is evidence that renal calculi are associated with obesity and coronary heart diseases. Stone recurrence is common and the life time recurrence rate is 60-80%, according to different reports. Since many of the renal calculi migrated to the ureter to be passed out later, we used the general term urinary calculi in this communication.

Formation of urinary calculi
The normal daily urine output is around 2 litres and all the solutes generated from the body metabolism have to be passed out in the 2 litres of urine. The urine so formed is supersaturated, that is, it holds more solute than a saturated solution can normally do. There is thus a natural tendency for solutes such as calcium oxalate to crystallize in the urine. Once a crystal is formed, it acts as a nidus and further crystallization will occur at an accelerated pace. Some studies suggested that calcium oxalate crystals first form in the renal tubules and attach to the anionic sites in the renal tubular cells. Stone formation is kept in check by natural inhibitors in the urine as a citrates and glycoproteins. They form soluble complexes with the calcium oxalate and keep them in solution.

The chemical composition of the urinary calculi is summarized as in table 1. It can be seen that the majority of the stones is calcium oxalate stones.

<table>
<thead>
<tr>
<th>Composition</th>
<th>Frequency, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium Oxalate</td>
<td>60</td>
</tr>
<tr>
<td>Calcium oxalate and calcium phosphate</td>
<td>10</td>
</tr>
<tr>
<td>Calcium phosphate</td>
<td>10</td>
</tr>
<tr>
<td>Magnesium ammonium phosphate (struvite)</td>
<td>3-10</td>
</tr>
<tr>
<td>Uric acid</td>
<td>3-10</td>
</tr>
<tr>
<td>Cystine</td>
<td>1</td>
</tr>
<tr>
<td>Others</td>
<td>1</td>
</tr>
</tbody>
</table>

The magnesium ammonium phosphate stone (struvite stone 燕窩石), deserves special mention. It is often associated with urinary stasis and urinary tract infection with urease producing organisms such as Proteus, Klebsiella or Serratia species. The urease breaks down urea into ammonia and carbon dioxide. Because of the ammonia production, the urine is alkaline with pH >7. The chemical composition is magnesium ammonium phosphate (struvite). This chemical was first recovered from medieval sewer systems in Hamburg Germany in 1845 and named after the geographer and geologist Heinrich Christian Gottfried von Struve (Wikipedia). Since this chemical is also found in bird droppings, it is called ‘bird droppings stone’ 鳥糞石 in Chinese literature.

Renal calculi with repeated urinary tract infections should raise the suspicion of struvite stones. Similarly, the discovery of the magnesium and ammonium content in a urinary stone sample would alert the physician to the possibility of urinary tract stasis and urinary tract infection as the cause of the stone. Checking the pH of the urine would be useful beside test to substantiate the suspicion of struvite stone (Figure 1).

The treatment of the struvite stone includes the complete surgical removal of the stone and the treatment of the urinary tract infection. A urease inhibitor, acetohydroxamic acid, had been tried in some cases where complete stone clearance was not achieved.

The role of physicians include:
1. Diagnosis of the urinary stone disease
2. Investigations for the cause of the renal calculi
3. Expedient management and referral to urologists for surgical intervention
4. Dietary and other advice to the patients
5. Pharmacological treatment for stone recurrence

Figure 1: Testing the pH of the urine by the bedside
**Diagnosis of urinary calculi**

Urinary calculi usually present as ureteric colic with loin pain and haematuria. However, they can be silent and only detected as incidental findings on medical check. Silent renal stones can present as its complications like urinary tract infection or obstruction. The whole renal pelvis might be occupied by the stone as in the case of staghorn calculi (Figure 2).

The simplest investigation is the plain X-ray (KUB) but they cannot detect radiolucent stones and it could not reveal the state of the urinary tract. This causes problems because it may be difficult to distinguish between a lower ureteric stone and calcification in the veins in the pelvis.

The renal ultrasound is safe, effective and can be performed at the bedside. Apart from the renal stones, the kidneys and the presence of hydronephrosis can be detected. However, ureteric stones cannot be easily visualized.

The intravenous urogram (IVU) used to be the gold standard in the urinary calculi investigation as it can show the urinary tract as well as the stones (Figure 3). It also showed the different stages of excretion of the contrast. The disadvantage is that it depends on the renal excretion of the radiocontrast. It would be problematic if there is severe obstruction when the excretion is hampered by the back pressure from the obstruction, thus affecting the quality of the image. This can be partially compensated by taking a delayed film up to 48 hours to wait for the accumulation of the contrast (Figures 4A, 4B). Another situation is when the renal function is impaired and hence the contract excretion was low. In the past this was partially compensated by increasing the dose (‘double dose IVU’ and ‘drip infusion IVU’) or a nephrotomogram, but the nephrotoxic effect of the contrast is an increasing concern.

With the improvement in the computerized tomography (CT) imaging technique, non-contrast CT urogram is now available and the relation of the urinary stone to the urinary tract can be studied without the use of radio-contrast (Figures 5A, 5B). The cost of the non-contrast CT urogram has been lowered and is increasingly used. However, a renal ultrasound plus KUB would be sufficient in the majority of cases. If a patient presents a gross haematuria, it might be advisable to order a contrast CT urogram to exclude causes of haematuria such as urological malignancy.

**Causes of renal stone formation**

In most cases, the cause of the renal calculus for an individual patient is not known for certain. Causes for renal stone formation include:

1. Primary hyperparathyroidism, renal tubular acidosis and sarcoidosis;
2. Increased urinary excretion of calcium (idiopathic hypercalcicuria), uric acid excretion and cystine excretion;
3. Malabsorption such as small bowel inflammation or small bowel resection. It is believed that the malabsorption result in the presence of fat in the colon. The fat increases the absorption of oxalates from the large bowel and increases the risk of oxalate stones;
4. Anatomical abnormalities of the urinary tract such as urinary reflux, polycystic kidneys diseases and medullary sponge kidneys. Urinary stasis would increase the chance of urinary tract infection. Some organisms produce urease which breakdown the urea into ammonia and cause the formation of struvite stones.
Simple tests such as renal function test, serum calcium, alkaline phosphate, uric acid level are useful. In recurrent cases, the 24 hour urine calcium excretion (normal value: 0.1 mmol/kg/day) or uric acid excretion (4.5 mmol/day for women and 4.8 mmol/day for men) might be carried out to detect the presence of hypercalciuria and hyperuricosuria. However, review of the literature showed that there is no evidence that such approach does not reduce stone recurrence (2).

Dietary approach to urinary calculi disease
In the 1980s, the usual dietary advice was as follows:
1. Drink large amount of water to ensure the passage of 3 litres of urine
2. Since the commonest stone is calcium oxalate, avoid high calcium diet and high calcium milk
3. Avoid taking large amount of strong tea because of the oxalate content
4. A urinary calculus may be available, such as those stones which passed out spontaneously, those removed by surgery or those stone fragments passed out after ESWL. In that case, send the stone for chemical composition so that the dietary advice may be tailored accordingly (Figures 6A, 6B)

Some of these dietary approaches are challenged by recent findings (2, 3). The current approach is as follows:

Fluid intake
It is now generally agreed that the most effective method to prevent renal stone formation is to ensure the passage of 2.5 litres of urine per day, although the evidence in support of it is of low quality only (2). Note the guideline refers to the amount of urine passed, not the amount of fluid ingested. This is because in a hot environment, one might lose a lot of water through sweating and the urine would be very concentrated despite a large amount of water intake. The urine output is thus chosen as the ‘end-point’. Increasing the urine volume will decrease the concentration of the urine, thus reducing the risk of crystallization. Some advised people not to take mineral water because of its mineral content; however, studies had shown that there is no difference in renal stone formation rate between the groups who take mineral water and tap water. The volume of the fluid intake is the most important consideration, not the nature of the drink.

Although a person may pass 2.5 litres of urine in a day in total, he might be exposed to hot environment during certain part of the day and the urine might be concentrated during that period. It is important to note that water loss in the form of sweating should not be underestimated, especially during sports in a hot environment. People were advised to look at their urine colour and to ensure the passage of dilute urine. If the urine colour was dark, he should drink more water immediately. Urine colour charts are now available for people to check their urine concentration and guide their water intake (Figure 7).

The tea story
In the past, strong tea was thought to increase stone formation due to the oxalate content and people were asked to avoid strong tea. Later studies showed that for people taking green tea regularly, the incidence of stone formation was in fact reduced. It was postulated that the phenol in the green tea causes the calcium oxalate crystals formed in the urine to become flatter and hence less likely to aggregate to form stones. Similarly, recent studies showed that moderate intake of tea, coffee or wine would reduce, rather than increase, stone risk. It was postulated that such drinks inhibit the excretion of anti-diuretic hormone from the hypothalamus, thus causing the passage of dilute urine.

This is in agreement with the concept that the volume of the fluid taken is more important than the nature of the drink.
**Dietary calcium**
For a long time, it was held that calcium intake should be reduced in patients with renal stone diseases because calcium is the main component of urinary stones. Studies in osteoporotic women who took calcium supplements revealed that they have reduced incidence of stone formation. The mechanism for such stone protective effect is that the calcium in the food form a complex with the oxalate in the diet and would pass out in the faeces. In this way, the calcium acts as an ‘oxalate binder’ in the gut and hence the oxalate absorption is reduced.

The current advice is that high oxalate food should be taken with a calcium rich diet. The oxalate will be bound by the calcium and will pass out un-absorbed.

Calcium restriction is therefore not necessary. The recommended calcium intake is 800 mg/day while one cup of milk is 300 mg/day.

**Dietary oxalate**
In the urine, the amount of calcium is much larger than the oxalate, hence an increase in oxalate excretion in the urine would have a bigger effect in calcium oxalate stone formation. Oxalate are found in fruits and vegetables, nuts and seeds, grains, legumes, and even chocolate and tea. High oxalate foods include: peanuts, rhubarb, spinach, beets, chocolate and sweet potatoes. However, dietary oxalate contributes only about 20-50% of urinary oxalate. Only moderate dietary oxalate restriction is needed and it is best to be taken with calcium rich foods (See dietary calcium above).

**Urinary citrates**
Citrate in urine binds with substances like oxalate and keeps them in solution instead of crystallizing. It is thus a stone inhibitor. It also prevents the crystals which had been formed to aggregate to become stones. Many substances crystallize in acid urine and citrates increases the urinary pH (making it less acidic) and the tendency for stone formation is reduced. An example is the uric acid stone prevention. Citrates, by its alkalinizing effect in the urine, have a greater effect in uric acid stone prevention than allopurinol, which decreases uric acid formation.

Apart from exogenous source, citrates are also produced by the body in the Krebs’s cycle. Citrate secretion in the urine reduced in acidosis and hypokalaemia, thus accounting for the increased formation of renal stones in renal tubular acidosis.

To increase the citrate in urine, potassium citrate can be given. However, lemonade and other citrate juice are just as effective. It was found that orange juice has greater alkalinizing power and greater citrate excretion in the urine.

**Three unlikely culprits: sodium, animal protein and sugar**
Since 1980s, three dietary factors were found to be associated with urinary stone formation.

1. **Sodium**
It was found that high sodium in the diet is associated with stone formation. It was likely that the increased sodium excretion causes increased calcium excretion, thus increasing the chance of stone formation. It is recommended that the dietary salt should be restricted to 5 g/day, which is equivalent to one teaspoonful of salt (The average salt intake of Hong Kong people is 10 g/day). Note that 5 gram of salt (sodium chloride) is equivalent to 2 g of sodium because 40% of sodium chloride is sodium. In practice, most of the dietary sodium comes from pre-packed food such as ham and sausages. Reading the food labels is essential in managing dietary sodium intake.

2. **Animal protein**
Increased intake of animal protein is associated with urinary stone formation. The postulated mechanism is that the purine content in the meat broke down into uric acid which is excreted in the urine. It also increases the calcium excretion and decreases the citrates excretion. It is likely that the increases meat consumption is one of the factors for the increased stone incidence in affluent countries.

3. **Dietary sugar**
Dietary sugar increases blood glucose which causes glomerular hyperfiltration, which in turn causes increased sodium delivery and increased calcium excretion. Food items with simple sugars should be restricted. Coca-Cola has high sugar content and high phosphoric acid (which accounts for the brown colour). Both the sugar and the phosphate content increase stone risk. One study showed that in people who drink more than 160 ml of Coke has reduced chance of renal stones if they stop the intake (Figure 8).

**Pharmacological intervention**
For patients with recurrent renal stones despite dietary interventions, physicians can consider the drug approach.
1. **Thiazides**
Apart from being mild diuretics, thiazides decrease urinary calcium excretion in the urine. One study showed that after three years of thiazides administration, the risk of stone formation was reduced by 20%. The effect is therefore not marked. Drugs used include hydrochlorothiazide 50mg or indapamide 2.5mg om together with potassium supplement. Amiloride, a potassium sparing diuretics, can also be used without the need for potassium supplement.

2. **Xanthine Oxidase Inhibitors**
Xanthine oxidase inhibitors decrease uric acid production and reduce urinary uric acid excretion in the urine. The commonest agent used is allopurinol. In patients with uric acid stone, allopurinol can be used for stone prevention. However, since uric acid stones will form in an acidic environment, making the urine less acidic is also an important approach in its prevention. For patients with the common calcium oxalate stone together with high uric acid excretion, allopurinol administration is also useful.

For patients with allopurinol hypersensitivity, febuxostat can be considered.

3. **Citrates**
Urinary citrates bind to calcium and decrease the tendency for urinary stone formation. It also reduces urinary acidity. Potassium citrate can be administered although some studies indicated that oral citrate fruits intake can be just as effective.

Some studies seemed to suggest that combination therapy of the above is not more effective than monotherapy.

**Conclusion**
Although there are effective therapies to treat urinary stones, prevention of recurrence is still an important issue. Water intake and dietary manipulation are probably more important than pharmacological intervention. There are still controversies and unsolved questions in the ‘renal stone’ diet, it would be advisable to discuss with the dietitian to work out an individualized diet for the patient based on the modern understanding and the patient characteristics.

**Renal stone pamphlets**
To facilitate patient communication, the Hong Kong Nephrology Group has published some renal stone pamphlets in Chinese (腎結石患者須知). Interested readers may contact the author for free copies (Figures 9A, 9B).

**References**

**Declaration of Interest**
This article is based on the talk presented by the author in the Uro-nephrology meeting on 12 August, 2016 in the Hong Kong Baptist Hospital. The renal stone pamphlet project was kindly sponsored by Astellas. There was no other interest to declare.

**Q&A Self-Assessment Questions:**
Complete this course and earn 1 CME Point
Answer these on page 21 or make an online submission at: www.hkmacme.org. Please indicate whether the following statements are true or false.
1. The most common chemical composition of the urinary calculi is calcium oxalate.
2. If urine is supersaturated, there is a tendency for crystallization in the urine causing stones.
4. The investigation of choice for ureteric calculi in a patient with renal impairment is intravenous urogram (IVU).
5. Drink large amount of water to ensure the passage of 2.5 litres of urine is the best way to avoid renal stone.
6. Renal calculi patients should avoid strong tea because of the oxalate content.
7. Citrate is a stone inhibitor because it binds with substances like oxalate and keeps them in solution instead of crystallizing.
8. High sodium in diet, increased intake of animal protein and increased blood glucose are three dietary factors that are associated with urinary stone formation.
9. Coca-Cola has high sugar content and high phosphoric acid will increase stone risk.
10. Thiazides decrease urinary calcium excretion in the urine thus reduce the risk of stone formation.
Point-of-care diagnostic test for seasonal influenza

Dr. TAM Yat Hung
MBChB (CUHK), FHKAM (Community Medicine), Specialist in Community Medicine

SPOTlight-3

Introduction

As part of the clinical decision and management process, development of diagnostic tests has gone through a long history, from the basic history taking and physical examination, to the increasingly sophisticated biochemical tests. Advanced technology has made most of the diagnostic tests centralised in medical laboratory. In relation to infectious diseases, identification of aetiological agents has been the mainstay of diagnosis in most clinical setting. While microbiological and virologic supports could be readily available in hospital setting or hospital based out-patient clinics, they are much less accessible to community clinics. Apart from the logistic difficulty in collecting suitable specimens in clinics and sending them to medical laboratory, community-based laboratories usually provide only limited microbiological tests than hospital-based laboratories which can afford to equip automated instruments because of their high throughput and economy of scale. As the results may take hours to days to be available, clinicians generally rely on laboratory tests mainly for non-acute clinical management. While highly accurate diagnostic tests are always desired by both clinicians and patients, readily accessible and affordable tests could be shown useful to guide diagnosis when clinical presentation has narrowed down to several possible diagnoses.

Nature of point-of-care diagnostic tests

Point-of-care diagnostic test (POCT) can be defined as medical testing at or near the site of patient care by specially trained healthcare professionals, generally outside the physical facilities of clinical laboratories. The POCT can be performed in various settings including primary care, community care or even mobile field station and may involve blood, urine, stool or other body secretions. It often requires only instruments ranging from simple testing strips for urinalysis, transportable, portable and handheld instruments like meters for blood glucose or international normalized ratio, to bench analysers for blood gas and haemoglobin level.1 In general, POCT allows more convenient test and immediate results for both clinicians and patients, enabling more timely diagnosis, monitoring and treatment. Moreover, its point-of-care nature may improve patient’s experience by being more participatory in the healthcare process, making compliance to clinical treatment and disease preventive advice potentially higher.

POCT has continued to evolve in terms of technology and diseases covered. Many POCT are originated from simplified laboratory-based tests. Before immunology-based technology was used in POCT, there had been urine test strip based on spot analysis for professional use since 1950s and the over-the-counter home testing kit for pregnancy since 1976.2 Immunologically or serologically based tests aiming at specific biomarkers or antigens later developed including beta-human chorionic gonadotropin, C-reactive protein, cardiac troponins, D-dimer, glycated haemoglobin A1c, prostatic specific antigen, thyroid stimulating hormone, faecal occult blood, etc.3

Most of the immunologically based POCT are using lateral flow designs in which the separation of analyte takes place as the sample moves along a solid phase. As the analyte moves, it is bound with colour particles dissolved by sample fluid and then captured by analyte-specific antibody immobilised in a stripe. Colour change occurs as more particles brought with the passing fluid accumulate and can be detected by spectrophotometer or naked eye depending on different products.4

The market of POCT has been growing for more than four decades. While it is largely made of self-monitoring of blood glucose and pregnancy test (both over-the-counter tests and professional tests), infectious disease testing is the area of fastest growing.4

Point-of-care diagnostic tests for infectious diseases

The range of infectious agents can be detected by POCT has increased rapidly. It covers different groups of common respiratory, enteric, sexually transmitted, vector-borne and blood-borne pathogens by testing on different kinds of clinical specimens using disposable reagent kit or card. Some examples are summarised in Table 1.

Many of the POCT have been shown highly valuable in developing countries lacking of facilities and especially in disaster setting using handheld devices, where cholera and other water-borne disease outbreak occurred.5 POCT for common respiratory pathogens such as influenza A and B, group A streptococci, Legionella pneumophila and pneumococci have been available in developed countries and adopted in primary care settings for many years.
predictive value, in regard to true and false infections defined.

Specificity, positive predictive value and negative predictive value for a POCT for an infectious disease

Sensitivity is defined as the proportion of patients tested truly positive by the screening test among those who have the infection according to the gold standard. Some false negative results are inevitable in any test, with its relative proportion of true positive results among those infected depending on the level of analyte that the assay required to be reactive and cross-reactivity of the assay to other analytes. On the contrary, specificity is defined as the proportion of patients tested truly negative by the screening test among those who do not have the infection. Similarly, some false positive results are inevitable. Sensitivity and specificity can therefore be regarded as the intrinsic properties of a screening test. The relationship between sensitivity and specificity can be seen in Figure 1 below.

On the other hand, positive predictive value (PPV) and negative predictive value (NPV) are not only affected by the intrinsic properties of the test but also the prevalence of infection in the population being tested. PPV is defined as the proportion of patients actually infected among those who are tested positive by the screening test. It can be seen in Figure 1 that the proportion of true positive among all patients tested positive by the test is directly related to the prevalence. NPV, which is defined as the proportion of patients actually not infected among those who are tested negative by the test is similarly affected by prevalence.

The relationship between these four performance indicators of screening test is essential in choosing appropriate POCT for the population being screening. For example, in a country with HIV prevalence of 25%, it can be expected that 25000 individuals be infected in 100000 population. If a POCT with sensitivity of 88% and specificity of 95% is used to screen these 100000 population, 25750 individuals would have positive results and 14.6% of them were false positive. On the other hand, if the POCT is used to screen 100000 population of another country with much lower HIV prevalence of 0.1%, only 5083 individuals would have positive results but 98.1% of them were false positive.

As PPV and NPV are particularly relevant to clinical practice when direct interpretation of positive or negative results is required, knowledge of the prevalence of the concerning infection in the population being screening is particularly important. It can be easily done by making use of the positive likelihood ratio (LR+), negative likelihood ratio (LR-) and converting prevalence to pre-test odds.

LR+ is often mentioned in the product information as a summary indicator of test sensitivity and specificity. It is defined as the ratio between the probability of obtaining a positive test result in an infected person and the probability of obtaining a positive test result in an uninfected person. It can be calculated by the formula:

\[
LR^+ = \frac{Sn}{(1-Sp)}.
\]
Pre-test odds can be converted from prevalence (Pv) by the formula:

\[
\text{Pv}/(1-\text{Pv})
\]

The product of LR+ and pre-test odds is the post-test odds (pO), which can be transformed to PPV by the formula:

\[
pO/(1+pO)
\]

Taking the previous POCT for HIV as example, the test’s LR+ is 17.6. As HIV prevalence of 25% is equivalent to pre-test odds of 0.34, the post-test odds can be calculated as 0.28, which can be readily transformed to PPV of 85.4%. Alternative PPV can therefore be estimated quickly by assuming different prevalence in the population. 1-NPV can be estimated similarly based on LR- instead of LR+ using the formula:

\[
\text{LR-} = (1-\text{Sn})/\text{Sp}
\]

Performance indicators of point-of-care diagnostic tests for infectious agents

Performance indicators of POCT vary between the tests for different infectious agents (Table 2). For instance, sensitivity is generally lower among POCT for respiratory agents than other infectious agents. Specificity of all POCT is generally high, making them useful as confirmatory test to rule in the suspected infection. Their short processing time allows their results to be read in the same clinical consultation.

Table 2. Performance indicators and processing time of selected point-of-care diagnostic tests

<table>
<thead>
<tr>
<th>Infectious agents</th>
<th>Processing time, mins</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
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</thead>
<tbody>
<tr>
<td>Influenza</td>
<td>15-30</td>
<td>23-74</td>
<td>98.2-100</td>
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<tr>
<td>Group A streptococci</td>
<td>8-10</td>
<td>86</td>
<td>94</td>
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<tr>
<td>Respiratory syncytial virus</td>
<td>15</td>
<td>80</td>
<td>97</td>
</tr>
<tr>
<td>Legionella pneumophila</td>
<td>15</td>
<td>31.5-88.9</td>
<td>86.6-100</td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td>15</td>
<td>74-75</td>
<td>80-99</td>
</tr>
<tr>
<td>Chlamydia trachomatis</td>
<td>20-40</td>
<td>17.1-98.7</td>
<td>53.1-100</td>
</tr>
<tr>
<td>Trichomonas vaginalis</td>
<td>20-40</td>
<td>38-98</td>
<td>88.8-100</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>20-40</td>
<td>12.5-100</td>
<td>88.9-100</td>
</tr>
<tr>
<td>Treponema pallidum</td>
<td>20-40</td>
<td>99.6-100</td>
<td>95.7-100</td>
</tr>
<tr>
<td>Plasmodia spp.</td>
<td>20</td>
<td>82.8-95</td>
<td>99.4-98.5</td>
</tr>
<tr>
<td>Dengue</td>
<td>15-20</td>
<td>21-99</td>
<td>77-100</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>20</td>
<td>75.4-99.5</td>
<td>94-99.8</td>
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</table>

Influenza

Duration* ≤48 h

<table>
<thead>
<tr>
<th>Infectious agents</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>PPV, %</th>
<th>NPV, %</th>
</tr>
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<tbody>
<tr>
<td>Group A streptococci</td>
<td>80</td>
<td>99</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>Population</td>
<td>Children 29</td>
<td>99</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>Influenza</td>
<td>Adult 37.2-92</td>
<td>98.2-100</td>
<td>94.7-96</td>
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<tr>
<td>Population</td>
<td>Children 53.9</td>
<td>98.6</td>
<td>93-97</td>
<td></td>
</tr>
<tr>
<td>Type</td>
<td>A 64.6</td>
<td>99.8</td>
<td>93-93</td>
<td></td>
</tr>
<tr>
<td>B 52.2</td>
<td>99.8</td>
<td>94.6-100</td>
<td>94.9-96</td>
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<tr>
<td>Duration*</td>
<td>≤48 h</td>
<td>39-92</td>
<td>96-100</td>
<td></td>
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<tr>
<td>&gt;48 h</td>
<td>25-74.2</td>
<td>87-99</td>
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<tr>
<td>Respiratory syncytial virus (RSV)</td>
<td>15</td>
<td>97</td>
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<td></td>
</tr>
<tr>
<td>Population</td>
<td>Children 41.2-97</td>
<td>93-100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td>74-75</td>
<td>80-99</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type</td>
<td>Bacteraemic 77-92</td>
<td>89-97</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-bacteraemic</td>
<td>72-78</td>
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</table>

* Duration of symptoms at the time of testing

Guiding use of POCT for seasonal influenza by performance indicators

In spite of these difficulties, most parameters involved in assessing performance of POCT for seasonal influenza could be reasonably estimated. Given that during influenza epidemic the prevalence of seasonal influenza A in the community can increase from the inter-epidemic baseline of 1% to 20%, it can be estimated that the PPV would increase from 42.0% to 84.7%, while the NPV would decrease from 99.6% to 19.5%. The substantial increase in PPV but minor decrease in NPV strongly supports that the POCT for seasonal influenza should be used mainly during influenza epidemics.

On the other hand, the low sensitivity of the POCT can be improved by combining with another screening test in series. Clinical practice itself can be regarded as a combination of screening tests. With a possible diagnosis in mind, each question to the patient and each physical examination is a test on a hypothesis as a POCT. Clinical case definition is a simple checklist of signs and symptoms which, when systematically applied, can serve as a screening tool for specific disease before confirmatory test is performed. For influenza, there are different case definitions designed for influenza surveillance but can also be used to aid clinical diagnosis. Influenza-like
illness (ILI) is the case definition adopted by the World Health Organization consisting of sudden-onset fever (> 38°C) with cough or sore throat, in the absence of other diagnoses, with sensitivity of 70% and specificity of 53% in detecting seasonal influenza.34 Applying such ILI definition to symptomatic patients during the early phase of influenza epidemic with 3% prevalence can provide a PPV of 4.4%. Even applying the POCT during this period can only provide PPV of 68.9%. However, if POCT is applied only to those fulfilling the ILI definition, its PPV could be boosted up to 76.8%.

Since the sensitivity of the POCT for influenza is rather low, symptoms suspicious of influenza and with onset within 48 hours should be carefully assessed before performing the test. While a positive test result during epidemic supports a reasonable chance of true influenza infection, a negative test result could not exclude influenza infection because an NPV of 91.8% means 8.2% of patient tested negative are actually false negative. Clinical symptoms, contact history and risk factor to influenza complications should be considered when establishing or excluding a diagnosis and deciding management.

**Key messages**

Point-of-care diagnostic test for seasonal influenza is a useful tool during influenza epidemic in primary care setting.

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**References**


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**Self-Assessment Questions:**

Complete this course and earn 1 CME Point

Answer these on page 21 or make an online submission at: www.hkmacme.org.

Please indicate whether the following statements are true or false.

1. Point-of-care tests generally require installation of bench analyser in the clinic.
2. Point-of-care tests can improve patient’s experience by being participatory in the healthcare process.
3. The processing time of point-of-care tests for infectious diseases is usually 60 minutes.
4. Some point-of-care tests for respiratory pathogens are designed to test on urine specimens.
5. Clinical performance of a point-of-care test is decided by its sensitivity and specificity only.
6. Sensitivity of a screening test is defined as the proportion of patients actually infected among those who are tested positive by the screening test.
7. Positive predictive value and negative predictive value of a screening are affected by the prevalence of the targeted disease in the population.
8. Only the positive likelihood ratio of a screening test and the prevalence of the disease being screened are required for estimating the positive predictive value.
9. Only the population being screened and type of infectious agent should be considered when choosing the appropriate point-of-care diagnostic test.
10. Apart from the point-of-care test result for influenza, patient’s clinical symptoms, contact history and risk factor to complications should be considered when deciding management.
Drug-induced ECG changes

A 60-year-old gentleman with diabetic neuropathy complained of bilateral lower limb numbness. He was prescribed amitriptyline for symptom control. He did not have any prior cardiovascular diseases. His pre-treatment ECG is shown in Figure 1.

After 2 weeks of treatment, patient’s symptoms improved. Another ECG was performed which is shown in Figure 2. He did not report any cardiac symptoms such as chest pain, shortness of breath, palpitation or syncope. His family history was unremarkable regarding cardiovascular diseases and sudden cardiac death.

Q&A

Please indicate one answer to each question

1. What is shown in the ECG in Figure 2?
   A. Right bundle branch block
   B. ST-segment elevation myocardial infarction
   C. Prolonged QT interval
   D. Type I Brugada ECG pattern
   E. Atrial fibrillation

2. The following drugs have been reported to induce similar ECG changes except:
   A. Flecainide
   B. Cocaine
   C. Procainamide
   D. Digoxin
   E. Fluoxetine

3. What is the appropriate management for this gentleman?
   1. Withdraw amitriptyline and consider alternative treatment
   2. Electrophysiology study
   3. Amiodarone
   4. Implantable cardioverter-defibrillator (ICD)

   A. 1 only
   B. 1 and 3 only
   C. 1, 2 and 3 only
   D. 1, 2 and 4 only
   E. All of the above
August Answers

1) C  2) D  3) B  4) A

Discussion
The differential diagnosis of a patient with cutaneous purpura and myocardial infarction can be challenging. However, with the addition of other organ involvement including the kidney, and joints, one should suspect a systemic cause, in particular vasculitis of small to medium sized vessels. Although Henoch-Schönlein Purpura (HSP) is predominantly a pediatric disease, it can affect adult too, and should not be overlooked in patients passed adolescent year but presenting with typical triad of symptoms. HSP commonly affects the kidney, causing hematuria and IgA type nephropathy. Extra-renal involvements are rare but can be life threatening if missed. Cardiac involvement has been described only in a handful of case reports. Atrioventricular block, arrhythmias, autoimmune myocarditis and STEMI were some of the reported cardiac manifestations of HSP.

The prompt normalization of ST segment change in our patient suggested of transient ischemic of the myocardium, and one possible mechanism can be coronary artery spasm precipitated by vasculitic process which damages the coronary vessels endothelium and releases vasoconstrictive substances from those cells, such as endothelin-1 which is known to have potent vasoconstrictive actions. Such phenomenon was observed in several other vasculitic conditions. Another postulated mechanism of the myocardial ischemia in our patient could be related to the acute arterial thrombosis that was associated with the presence of antiphospholipid antibodies that were sometimes found in patients with HSP. The antiphospholipid antibodies, however, were negative in our patient.

In our patient, coronary angiogram was performed to exclude coronary artery stenosis as our patient has multiple risk factors for atherosclerosis, and the angiogram demonstrated coronary artery spasm relieved by nitrate. Other causes of acute coronary syndrome with normal coronary arteries are summarized in Table 1.

<table>
<thead>
<tr>
<th>Causes of ACS with normal coronary arteries</th>
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<tr>
<td>Coronary Artery spasm</td>
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<td>Coronary Artery embolism</td>
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<td>Coronary Artery anomaly</td>
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<td>Pericarditis</td>
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<td>Myocarditis</td>
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<td>Cardiomyopathy</td>
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<td>Aortic stenosis</td>
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<tr>
<td>Aortic dissection</td>
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<td>Pulmonary embolism</td>
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<td>Syndrome X</td>
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While skin biopsy or tissue biopsy of the organ involved is often required to establish the final diagnosis, this can be time consuming and should not delay treatment if clinical suspicion is high. As with most vasculitic conditions, the initial treatment for patients suffering from HSP with multi-organ involvement is high dose steroid followed by immunosuppressive therapy.

Reference

The content of the August Cardiology Series is provided by:
Dr. TAN GuangMing MBChB, MRCP, FHKCP, FHKAM (Med), Specialist in Cardiology
Dr. CHEUNG Shing Him, Gary MBBS, MRCP, FHKCP, FHKAM (Med), Specialist in Cardiology

八月臨床心臟科個案研究之內容承蒙譚廣明醫生及張誠謙醫生提供。
Q&A

Please answer ALL questions

Answer these on page 21 or make an online submission at: www.hkmacme.org

1. What is this medication?
2. What is the mechanism of action?
3. What are the medical diseases that can be treated by this medication?
4. What are the cosmetic diseases that can be treated by this medication?
5. What are the possible complications?

The below figure shows a medication commonly used in a dermatology clinic.

August Answers

1. The diagnosis is longitudinal melanonychia where thumbs and big toes are the most commonly affected areas for both males and females. Differential diagnoses could be melanoma, subungual haematoma and onychomycosis.

2. Longitudinal melanonychia is caused by the deposition of melanin on nail matrix resulted from melanocytic activation or hyperplasia. There might be other varying causes like melanocytic naevi, trauma, inflammatory skin disease such as psoriasis or lichen planus, endocrine disorders, drug-related (e.g. chemotherapy), specific syndrome associated, fungal infections or subungual melanoma.

3. Relevant laboratory tests should be conducted accordingly if specific secondary causes are suspected. In suspicious cases, dermoscope may help the diagnosis of various skin lesions. But a skin biopsy is recommended where there is a periungual extension of pigment onto the proximal and lateral nail folds (Hutchinson’s sign), which suggests the possibility of subungual melanoma.

4. No treatment is necessary if longitudinal melanonychia is attributed to a benign cause. However, treatment of the underlying condition or discontinuation of the offending drug is required if it is secondary to a systemic disease or drug. If lesions are suggestive of melanoma or there are observant changes during follow up, a biopsy should be performed.
Please answer ALL questions and write the answers in the space provided.

**SPOTlight - 1**  
Complete Spotlight and earn **1 CME point**

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**SPOTlight - 2**  
Complete Spotlight and earn **1 CME point**

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**SPOTlight - 3**  
Complete Spotlight and earn **1 CME point**

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Complete BOTH Cardiology & Dermatology cases and earn **0.5 CME point**

**Cardiology**

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**Dermatology**

1

2

3

4

5
HKMA Structured CME Programme with HKS&H Session IX:

Current Management of Asthma in Adults

Date: 13 October 2016 (Thursday)
Time: 2:00–3:00 p.m. [Light lunch starts at 1:15 pm]
Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21–22 Connaught Road Central, HK

Speaker: Dr. LAM Chung Mei, Jamie
MBBS(UNSW), MD(HK), MRCP(UK), FRCP(Glasg), FRCP(Edin), FHKCP, FHKAM (Medicine)
Honorary Clinical Assistant Professor (HKU)
Specialist in Respiratory Medicine

This symposium is co-organized with Hong Kong Sanatorium & Hospital.

Registration:
Please fill in and return the Registration Form together with a cheque of adequate amount made payable to “The Hong Kong Medical Association” to 5/F Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong. Each lecture will carry 1 CME point under the MCHK/HKMA CME Programme (unless otherwise stated). Accreditation from other colleges is pending. (The Secretariat fax no.: 2865 0943)

Reply Slip

I would like to register for the following CME lecture:

HKMA Structured CME Programme with HKS&H

13 October 2016 (Thursday) Current Management of Asthma in Adults

HKMA Member HK$50 CME Participants HK$80

I enclose herewith a cheque of HK$

Name:

HKMA Membership No. or HKMA CME No.:

Mobile No.: Signature:

Fax No.: Date:

Data collected will be used and processed for the purposes related to the MCHK/HKMA CME Programme only. All registration fees are not refundable or transferable.
Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?

Co-organized by
HKMA Shatin Doctors Network
and Primary Care Office of the Department of Health

Date: Wednesday, 19 October 2016

Speaker: Dr. FOK Oi Ling, Annie
Acting Senior Medical Officer, Family Health Service Head Office, Department of Health

Time: 1:00 – 2:00 p.m. Registration & Lunch
2:00 – 2:45 p.m. Lecture
2:45 – 3:00 p.m. Q&A Session

Venue: Jasmine Room, Level 2, Royal Park Hotel,
8 Pak Hok Ting Street, Shatin, Hong Kong

Moderator: Dr. MAK Wing Kin
CME Convenor, HKMA Shatin Doctors Network

Deadline: Friday, 7 October 2016

Fee: Free-of-charge

Capacity: 60. Registration is strictly required on a first come, first served basis. Priority will be given to doctors practising in Shatin district.

Enquiry: Ms. Candice TONG, Tel: 2527 8285
*Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.

CME Accreditation: Pending

REPLY SLIP

HKMA Shatin Doctors Network
Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?

Fax: 2865 0943

[ ] I would like to register for the above lecture. Please “✓” as appropriate

Name: [ ] HKMA No.: [ ]
Mobile No.*: [ ] Fax No.: [ ]

*Please fill in your updated mobile number so that you can be notified of your application via SMS. If you do not have a mobile phone, the Secretariat will still issue a confirmation letter to you.

Practising location: In Shatin (Please specify *: [ ])
Others (Please specify: [ ])

* Null entry will be treated as non-Shatin member registration.

Signature: [ ] Date: [ ]

Data collected will be used and processed for the purposes related to this event only.
CME Lectures in October 2016

Organizer: HKMA New Territories West Community Network
HKMA Kowloon West Community Network

Date: Thursday, 6 October 2016
Tuesday, 18 October 2016

Topic: Ambulatory Blood Pressure Monitoring
Update on the Management of Chronic Hepatitis B

Speaker: Dr. YAN Chun Ting, Fergus
Specialist in Cardiology
Dr. FUNG Tang Tat, Konrad
Specialist in Gastroenterology & Hepatology

Time: 1:00 – 2:00 p.m. Registration & Lunch
2:00 – 2:45 p.m. Lecture
2:45 – 3:00 p.m. Q&A Session

Venue: Atrium Function Rooms, Lobby Floor, Hong Kong Gold Coast Hotel,
1 Castle Peak Road, Gold Coast, Hong Kong
(Crystal Room IV-V, 3/F., Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, N.T.)

Moderator: Dr. CHEUNG Kwok Wai, Alvin
Chairman,
HKMA NT West Community Network
Dr. CHAN Ching Pong
Committee Member,
HKMA Kin West Community Network

Deadline: Monday, 26 September 2016
Friday, 7 October 2016

Fee: Free-of-charge

Capacity: 50

Registration is strictly required on a first come, first served basis. Priority will be given to doctors practising in NT West districts (for the lecture on 6 Oct)/Kowloon West districts (for the lecture on 18 Oct).

Enquiry: Miss Hana YEUNG, Tel: 2527 8285
*Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.

Sponsor: SANOFI
Bristol-Myers Squibb Pharma (HK) Ltd.

CME Accreditation: Pending

REPLY SLIP

HKMA NTW and KW Community Networks
CME Lectures in October 2016
Fax: 2865 0943

I would like to register for the following lecture(s):

6 October 2016 (NTW) 18 October 2016 (KW)

Please “✓” as appropriate

Name: ____________________________ HKMA No.: ____________________________
Mobile No.: ____________________________ Fax No.: ____________________________
* Please fill in your updated mobile number so that you can be notified of your application via SMS. If you do not have a mobile phone, the Secretariat will still issue a confirmation letter to you.

Practising location: In New Territories West districts (Please specify *):
In Kowloon West districts (Please specify *):
Others (Please specify):

* Null entry will be treated as non-New Territories West or non-Kowloon West member registration.

Signature: ____________________________ Date: ____________________________

* Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.

Data collected will be used and processed for the purposes related to these events only.
CME Lectures in October 2016

Date: Thursday, 6 October 2016
Topic: New Treatment for Heart Failure Patients
Speaker: Dr. LEUNG Kwok Fai
Specialist in Cardiology
Time: 1:00 – 2:00 p.m. Registration & Lunch
2:00 – 2:45 p.m. Lecture
2:45 – 3:00 p.m. Q&A Session
Venue: Lei Garden Restaurant (利苑酒家),
Shop no. L5-8, apm, Kwun Tong,
No. 418 Kwun Tong Road, Kowloon
Moderator: Dr. AU Ka Kui, Gary
Chairman,
HKMA Kowloon East Community Network
Fee: Free-of-charge
Capacity: 48. Registration is strictly required on a first come, first served basis. Priority will be given to doctors practising in the Kowloon East district.
Deadline: Monday, 26 September 2016
Enquiry: Miss Hana YEUNG, Tel: 2527 8285
*Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.
Sponsor: Boehringer Ingelheim
CME Accreditation: Pending

Date: Thursday, 20 October 2016
Topic: Update on Type 2 Diabetes Management – Focus on SGLT2 inhibitors
Speaker: Dr. LAU Wing Yan, Winnie
Specialist in Endocrinology, Diabetes & Metabolism
Time: 2:00 – 2:45 p.m. Lecture
2:45 – 3:00 p.m. Q&A Session
Venue: V Cuisine, 6/F., Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tseung Kwan O (將軍澳唐德街3號香港九龍東智選假日酒店6樓彩雲軒)
Moderator: Dr. MA Ping Kwan, Danny
Vice-chairman,
HKMA Kowloon East Community Network
Fee: Free-of-charge
Capacity: 48. Registration is strictly required on a first come, first served basis. Priority will be given to doctors practising in the Kowloon East district.
Deadline: Friday, 7 October 2016
Enquiry: Miss Hana YEUNG, Tel: 2527 8285
*Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.
Sponsor: Boehringer Ingelheim
CME Accreditation: Pending
Pearls for the Early Identification of Inflammatory Arthritis

Date: Saturday, 15 October 2016
Speaker: Dr. LEE Ka Wing, Gavin
Specialist in Rheumatology
Time: 1:30 – 2:00 p.m. Registration & Lunch
2:00 – 2:30 p.m. Lecture by Dr. Gavin LEE
2:30 – 3:20 p.m. Clinical Workshops:
   Workshop 1: Bio Markers by Dr. Gavin LEE
   Workshop 2: RA/Back Pain by Dr. YIP Man Lung, Ronald
3:20 – 3:30 p.m. Q & A Session
Venue: Plentiful Delight Banquet, 1/F., Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long
Moderator: Dr. CHUNG Siu Kwan, Ivan
Vice-Chairman, HKMA NT West Community Network,
Deadline: Friday, 30 September 2016
Fee: Free-of-charge
Capacity: 24. Registration is strictly required on a first come, first served basis. Priority will be given to doctors practising in NT West district.
Enquiry: Miss Hana YEUNG, Tel: 2527 8285
*Please call and confirm that your facsimile has been successfully transmitted to the HKMA Secretariat if you do not receive confirmation 14 days before the event.
CME Accreditation: Pending

This lecture is sponsored by Pfizer Hong Kong Corporation Limited

REPLY SLIP

HKMA New Territories West Community Network
Pearls for the Early Identification of Inflammatory Arthritis
Fax: 2865 0943

I would like to register for the above event. Please "✓" as appropriate

<table>
<thead>
<tr>
<th>Name:</th>
<th>HKMA No.:</th>
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<td>Mobile No.*:</td>
<td>Fax No.:</td>
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* Please fill in your updated mobile number so that you can be notified of your application via SMS. If you do not have a mobile phone, the Secretariat will still issue a confirmation letter to you.

Practising location: In New Territories West (Please specify *: )
Others (Please specify: )

* Null entry will be treated as non-New Territories West member registration.

Signature: Date:

Data collected will be used and processed for the purposes related to this event only.
The HKMA Central, Western and Southern Community Network (CW&SCN) ~ Dr. YIK Ping Yin

Three CME lectures were being held in August 2016. Dr. CHOW Chun Kuen, Specialist in Otorhinolaryngology, presented on “Current Management of Meniere’s Disease” on Wednesday, 10 August 2016. On Friday, 19 August 2016, Dr. TSANG Tat Chi, Consultant of Accident & Emergency Department of Queen Mary Hospital gave a talk on “Clinical Wisdom in Managing Patient Presenting with Acute Chest Pain” while Dr. CHAN Hon Wah, Raymond, Consultant Cardiologist & Chief (Clinical Services) of Cardiac Division of the Department of Medicine of Queen Mary Hospital, presented on “Management Update on Acute Coronary Syndrome”. On Wednesday, 24 August 2016, Dr. FU Chi Kin, Jackie, Specialist in Psychiatry, delivered a lecture on “Precision Medicine in Clinical Practice”.

Dr. CHAN Chun Yin, Johnny, Specialist in Dermatology & Venereology, delivered a luncheon lecture on “Biologic Therapies in the Management of Psoriasis” on Thursday, 11 August 2016 at the HKMA Central Premises. Dr. HAU Kwun Cheung kindly acted as the moderator for the event.

Dr. LAM Chung Mei, Jamie, Specialist in Respiratory Medicine, will give a talk on “Current Management of Asthma in Adults” on Thursday, 13 October 2016. Interested members please refer to the announcement on p.22 for details and enrolment.

The HKMA Yau Tsim Mong Community Network (YTMCN) ~ Dr. LAM Tzit Yuen, David

Dr. LEE Cheuk Fun, Justin, Specialist in Cardiology, delivered a talk on “Review of Cardiovascular Outcome Trials of Oral Glucose-lowering Drugs” on Tuesday, 9 August 2016.
The HKMA New Territories West Community Network (NTWCN) ~ Dr. CHEUNG Kwok Wai, Alvin

Dr. LI, Ernest Han Fai, Specialist in Gastroenterology & Hepatology, gave a talk on “Latest Update in GERD Management” on Thursday, 11 August 2016.

Dr. CHAN Chun Chung, Ray, Specialist in Geriatric Medicine, will deliver a talk on “Ambulatory Blood Pressure Monitoring” on Thursday, 6 October 2016. Interested members please refer to the announcement on p.25 for details and enrolment.

On Saturday, 15 October 2016, Dr. LEE Ka Wing, Gavin, Specialist in Rheumatology, will present on “Pearls for the Early Identification of Inflammatory Arthritis”. There will be two clinic workshops hosted by Dr. Gavin LEE and Dr. YIP Man Lung, Ronald respectively after the talk. Interested members please refer to the announcement on p.27 for details and enrolment.

The HKMA Shatin Doctors Network (SDN) ~ Dr. FUNG Yee Leung, Wilson and Dr. MAK Wing Kin

Dr. CHAN Tak Yan, Norman, Specialist in Paediatrics, will present on “Rotavirus Infection in Children: Disease Burden and Prevention” on Friday, 7 October 2016. Interested members please contact Mr. David YIM at 8226 9592 for registration and enquiry.

Besides, a CME lecture on “Seminar on Management of Common Breastfeeding Problems: What Primary Care Doctors Need to Know and Practice?” which is co-organized by the Network and Primary Care Office of the Department of Health (DH) will be presented by Dr. FOK Oi Ling, Annie, Acting Senior Medical Officer of Family Health Service Head Office of DH, on Wednesday, 19 October 2016. Interested members please refer to the announcement on p.24 for details and enrolment.

The HKMA Hong Kong East Community Network (HKECN) ~ Dr. CHAN Nim Tak, Douglas

The final session of the “Certificate Course on Diabetes Mellitus” titled “Management of DM Complications” was presented by Dr. TING Zhao Wei, Rose, Specialist in Endocrinology, Diabetes & Metabolism, on Thursday, 11 August 2016. Moreover, Dr. CHAU Mo Chee, Elaine, Specialist in Cardiology, delivered the lecture on “The Latest Updates on the Management of Atrial Fibrillation – Real Life Evidence on NOACs” on Thursday, 18 August 2016.

Dr. YUNG Wai Ming, Miranda, Specialist in Respiratory Medicine, will present on “Update on Prevention of COPD Exacerbations” on Thursday, 6 October 2016. Dr. CHOW Wing Cheong, Louis, Specialist in General Surgery, will present on “Breast Cancer Prevention for General Women Aged >45 and Chemoprevention for High Risk Group” on Thursday, 20 October 2016. Interested members please refer to the announcement on p.23 for details and enrolment.
The HKMA Kowloon East Community Network (KECN) ~ Dr. AU Ka Kui, Gary

Dr. HO Ka Keung, Specialist in Dermatology & Venereology, gave a talk on “Update Management on Psoriasiform Dermatosis in Clinical Practice” on Thursday, 11 August 2016. The third session of the “CME Course for Health Personnel 2016” titled “Management of Blurred Vision” was delivered by Dr. CHUNG Chung Yee, Derek, Associate Consultant of the Department of Ophthalmology of United Christian Hospital, on Saturday, 20 August 2016.

Dr. LEUNG Kwok Fai, Specialist in Cardiology, will give a talk on “New Treatment for Heart Failure Patients” on Thursday, 6 October 2016. Dr. LAU Wing Yan, Winnie, Specialist in Endocrinology, Diabetes & Metabolism, will present on “Update on Type 2 Diabetes Management – Focus on SGLT2 inhibitors” on Thursday, 20 October 2016. Interested members please refer to the announcement on p.25 for details and enrolment.

The HKMA Kowloon West Community Network (KWCN) ~ Dr. TONG Kai Sing

Dr. YIP Wai Chun, Andrew, Specialist in Urology, presented on “New Challenges of Erectile Dysfunction Management” on Tuesday, 9 August 2016. Dr. FUNG Lai Ming, Specialist in Endocrinology, Diabetes & Metabolism, gave a talk on “Update on Type 2 Diabetes Management – Focus on SGLT2 inhibitors” on Tuesday, 23 August 2016.

Dr. FUNG Tang Tat, Konrad, Specialist in Gastroenterology & Hepatology, will present on “Update on the Management of Chronic Hepatitis B” on Tuesday, 18 October 2016. Interested members please refer to the announcement on p.25 for details and enrolment.

HKMA Tai Po Community Network (TPCN) ~ Dr. CHIU Sik Ho, Bonba

Dr. TSE Lung Fung, Specialist in Orthopaedics & Traumatology, will present on “Fragility Fracture: Medical and Surgical Treatment” on Tuesday, 4 October 2016. Interested members please contact Mr. Nathan YEUNG at 8199 8834 for enrolment.
HKMA CME Bulletin

September 2016

16 Sep 2016    HK Sanatorium & Hospital – Surgery Centre
1:00 – 3:00 pm  Joint Surgical Pathology Meeting with Hong Kong Baptist Hospital; St. Teresa’s Hospital
Lecture Room 7A, 7/F, Li Shu Pu Block, Phase I, HKSH; Chapel, 8/F, Block C, Hong Kong Baptist Hospital; 9/F, Conference Room, Main Block, St. Teresa’s Hospital
Ms. Ng Mei Han – Tel: 2835 8998

17 Sep 2016    HK Sanatorium & Hospital – Breast Care Centre
HK Sanatorium & Hospital – Comprehensive Oncology Centre
Multidisciplinary Breast Conference (Every Saturday)
7/F, Nursing Administration, Li Shu Pu Block, HKSH
Ms. Lam Kiey Yung – Tel: 2875 9206

17 Sep 2016    Hong Kong Medical Association
2016 HKMA Dragon Boat Team CME Lecture cum Celebration Dinner – Cardiology Update 2016 for everyday clinical practice
HKMA Dr. Li Shu Pu Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong
Miss Denise Kews – Tel: 2557 9826

19 Sep 2016    Union Hospital – Department of Paediatrics
Paediatrics Departmental Round
8:30 – 9:30 am
New Seminar Room 2, 2/F, Hospital Building, Union Hospital
Ms. Anthony Ma – Tel: 2609 3850

19 Sep 2016    HKU – Department of Medicine
HKU Generic Grand Round
12:00 – 1:00 pm
K2, Queen Mary Hospital; C7, Tung Wah Hospital; C3, Tung Wah Hospital; 5/F, KTHS Centre, Grantham Hospital; 5/F, Fung Yiu King Hospital
Ms. Jane Yau – Tel: 2255 4769

19 Sep 2016    HKU – Department of Medicine
HKU Generic Academic Meeting
1:00 – 2:00 pm
K2, Queen Mary Hospital; C7, Tung Wah Hospital; C3, Tung Wah Hospital; 5/F, KTHS Centre, Grantham Hospital; 5/F, Fung Yiu King Hospital
Ms. Jane Yau – Tel: 2255 4769

20 Sep 2016    Hong Kong Medical Association – Kowloon West Community Network
Diagnosis and Management of Attention Deficit Hyperactivity Disorder (ADHD) in Children
Crystal Room IV – 3/F, Panda Hotel, 3 Tsuen Wan Street, Tsuen Wan, NT
Miss Hana Yung – Tel: 2572 8285

21 Sep 2016    HK Baptist Hospital – Radiotherapy & Oncology Ctr
Urology Cancer Grand Round (Monthly; Wednesday of Second last week of each month)
Multifunction Room, 9/F, Block D, Hong Kong Baptist Hospital
Tel: 2309 8503

21 Sep 2016    HA – Our Lady of Maryknoll Hospital
Grand Round Journal Club (Wednesday Educational Meeting July-Sep 2016)
Conference Room A, 7/F, OPD Block, Our Lady of Maryknoll Hospital
Ms. Clara Tsang – Tel: 2354 2440

21 Sep 2016    Hong Kong Medical Association – Shatin Doctors Network
Recent Advancement in Cancer Immunotherapy
Jasmine Room, Level 2, Royal Park Hotel, Sha Tin
Ms. Zoe Ma – Tel: 9971 2930

21 Sep 2016    Hong Kong Medical Association – Central, Western & Southern Community Network
Certificate Course on Dermatology
Session 2: Common Superficial Fungal Infections
HKMA Central Premises, Dr. Li Shu Pu Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong
Miss June Wu – Tel: 2572 8285

21 Sep 2016    HA – GEH – Multi-disciplinary Trauma Service
Multi-Disciplinary Trauma Service Monthly & Morbidity Meetings 2016
Lecture Room-J000, Ground Floor, Block J, GEH
Ms. Chan Po Shan, Lily – Tel: 3506 2399

22 Sep 2016    HK Sanatorium & Hospital – Orthopaedic & Sports Medicine Ctr
Academic Professional Development Meeting 2016
HK Sanatorium & Hospital, Miss Cheung Yau Yan – Tel: 2883 7990

22 Sep 2016    Hong Kong Medical Association – Kowloon East Community Network
Rotavirus Infection in Children: Disease Burden and Prevention
V Cuisine, 6/F, Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tsuen Wan
Miss Hana Yung – Tel: 2572 8285

22 Sep 2016    HA – OMH – Dept of Neurosurgery
Neuroscience Working Group Meeting
Lecture Theatre, 5/F Professional Block, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong
Ms. Claire Chan – Tel: 2255 4181

22 Sep 2016    Hong Kong Society of Musculoskeletal Pain
Multidisciplinary Musculoskeletal Pain Grand Round and Case Discussions 2016
HK Pain Medicine Centre, Room 1301, SBI Centre, 54-59 Des Voeux Road Central, Hong Kong
Ms. May Cheung – Tel: 2988 8003

22-23 Sep 2016    Hong Kong Medical Association – Kowloon East Community Network
New Paradigms in Gout & Hyperuricemia Treatment
5/F, Duke of Windsor Social Service Buildings, 15 Hennessy Road, Wan Chai
Ms. Candice Tong – Tel: 2527 8285

25 Sep 2016    HK Sanatorium & Hospital – Surgery Centre
Moody’s & Mortality (Adult) Meeting
Lecture Room 7A, 7/F, Li Shu Pu Block, Phase I, HKSH
Ms. Ng Mei Han – Tel: 2835 8998

26 Sep 2016    HKU – Carol Yu Centre for Infection
Infectious Diseases Rounds for Year 2016
Conference Room, Room 405, Clinical Pathology Building, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong
Secretariat – Tel: 2255 5555

23 Sep 2016    Hong Kong Medical Association – You Tsun Mong Community Network
Update on Prevention of COPD Exacerbations
1:00 – 3:00 pm
Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon
Ms. Candice Tong – Tel: 2527 8285

23 Sep 2016    Hong Kong Medical Association – Kowloon City Community Network
Update on New Player in Cancer Therapy (Immunotherapy) – Long Plentiful Delight Banquet, 1/F, Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long
Ms. Ms. Candice Tong – Tel: 2527 8285

23 Sep 2016    Hong Kong Medical Association – Shatin Doctors Network
One Step Forward in BPH management: Early LUTS Identification and Treatment
Star Seafood Floating Restaurant, Sha Tin
Ms. Suk Chan – Tel: 2688 6053

24 Sep 2016    HK Sanatorium & Hospital – Breast Care Centre
Multidisciplinary Breast Conference (Every Saturday)
7/F, Nursing Administration, Li Shu Pu Block, HKSH
Ms. Lam Kiey Yung – Tel: 2875 9206

24 Sep 2016    Hong Kong Medical Association – Kowloon East Community Network
Update on New Player in Cancer Therapy (Immunotherapy)
1:00 – 3:00 pm
Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon
Ms. Candice Tong – Tel: 2527 8285

26 Sep 2016    HKU – Department of Medicine
HKU Generic Grand Round
12:00 – 1:00 pm
K2, Queen Mary Hospital; C7, Tung Wah Hospital; C3, Tung Wah Hospital; 5/F, KTHS Centre, Grantham Hospital; 5/F, Fung Yiu King Hospital
Miss June Wu – Tel: 2572 8285

26 Sep 2016    HKU – Department of Medicine
HKU Generic Academic Meeting
1:00 – 2:00 pm
K2, Queen Mary Hospital; C7, Tung Wah Hospital; C3, Tung Wah Hospital; 5/F, KTHS Centre, Grantham Hospital; 5/F, Fung Yiu King Hospital
Ms. Jane Yau – Tel: 2255 4769

28 Sep 2016    HK Baptist Hospital – Radiotherapy & Oncology Ctr
Lung Cancer Clinic – pathological conference (Monthly; Wednesday of Second last week of each month)
Multifunction Room, 9/F, Block D, Hong Kong Baptist Hospital
Tel: 2309 8503

28 Sep 2016    HA – Our Lady of Maryknoll Hospital
Grand Round Journal Club (Wednesday Educational Meeting July-Sep 2016)
Conference Room A, 7/F, OPD Block, Our Lady of Maryknoll Hospital
Ms. Clara Tsang – Tel: 2354 2440

29 Sep 2016    Hospital Authority – United Christian Hospital
Hong Kong College of Family Physicians
HKU Medical Association – Kowloon East Community Network
Certificate Course for GPs 2016 – Management of Arrhythmia
Conference Room, 5/F, Block K, United Christian Hospital
Tel: 2865 3435

29 Sep 2016    Hong Kong Medical Association – Information Technology Committee
HKMA.A.T. Seminar in New Territories West District – HKMA Clinic Management System (CMS) Yuan Wang (YW) IT Seminar
Pearl Delight Banquet, 1/F, Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long
Ms. Candice Tong – Tel: 2527 8285

29 Sep 2016    Hong Kong Medical Association
Improving asthma management – should we challenge our long-standing assumptions
Shangri-La Room, Cordis Hotel, 555 Shanghai Road, Mongkok, Kln, H.K.
HKMA CME Dept. – Tel: 2527 8452

HKMA CME Bulletin

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