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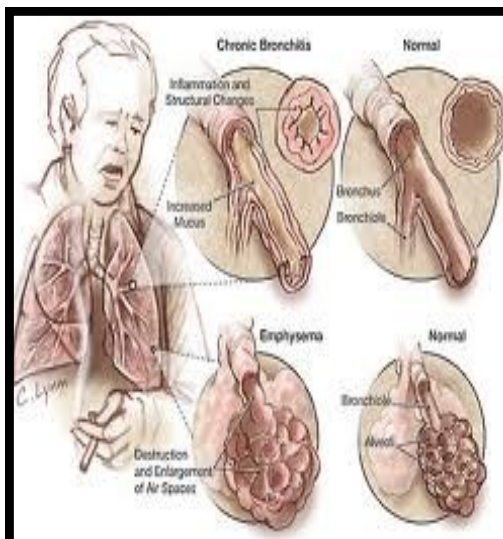
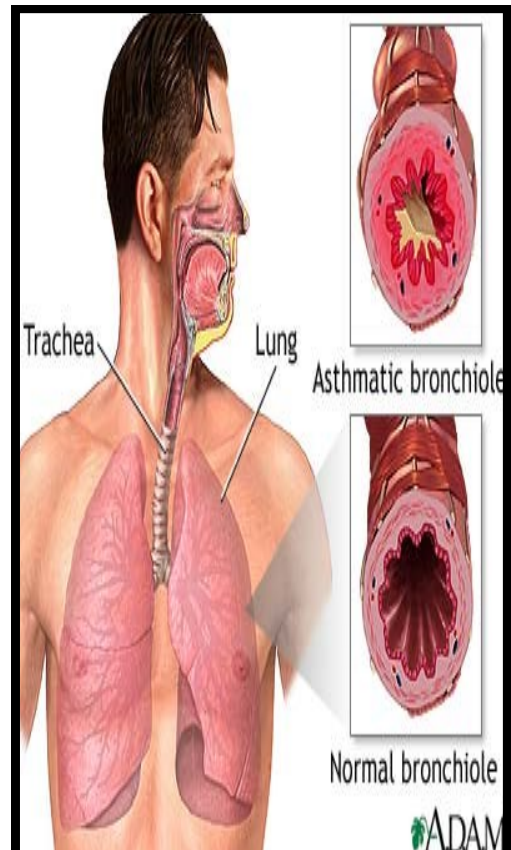
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ASTHMA AND COPD

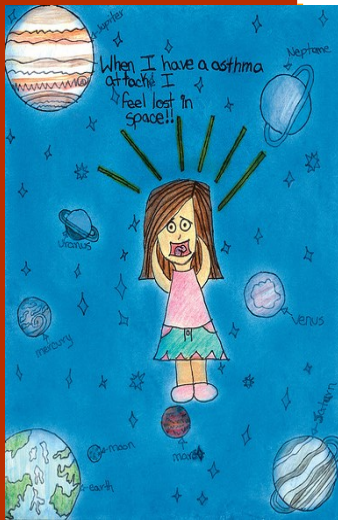
Asthma is a chronic inflammatory disorder of the airways. Chronically inflamed airways are hyperresponsive; they become obstructed and airflow is limited (by bronchoconstriction, mucus plugs, and increased inflammation) when airways are exposed to various risk factors.

Asthma is a common condition that gives rise to considerable morbidity and mortality. Its prevalence is increasing and a local study found 13.8% of primary school children in Kuala Lumpur to be asthmatic. It is underdiagnosed and often not managed optimally. In an ongoing surveillance of paediatric asthma deaths, 9 deaths have been reported in the past two years; and all of them have been due to inadequate assessment of the severity of the attack and hence under-treatment. There is an over reliance on symptomatic and oral therapy and an under-use of anti-inflammatory therapy leading to inadequate control and, in some cases, death. It is also recognised that disparities in management exist due to lack of access to appropriate information, drugs and resources.



Chronic obstructive pulmonary disease (COPD) is a syndrome characterized and defined by a single physiological parameter: limitation of expiratory airflow.

Although COPD and asthma have similar characteristics such as the signs of coughing and wheezing, they are two distinct conditions in terms of disease onset, frequency of symptoms and reversibility of airway obstruction.



“As well as pharmacologic therapy, nonpharmacology therapy can be managed with lifestyle changes”



Asthma

Definition

Asthma attacks all age groups but often starts in childhood. It is a disease characterized by recurrent attacks of breathlessness and wheezing, which vary in severity and frequency from person to person. In an individual, they may occur from hour to hour and day to day. This condition is due to inflammation of the air passages in the lungs and affects the sensitivity of the nerve endings in the airways so they become easily irritated. In an attack, the lining of the passages swell causing the airways to narrow

and reducing the flow of air in and out of the lungs.

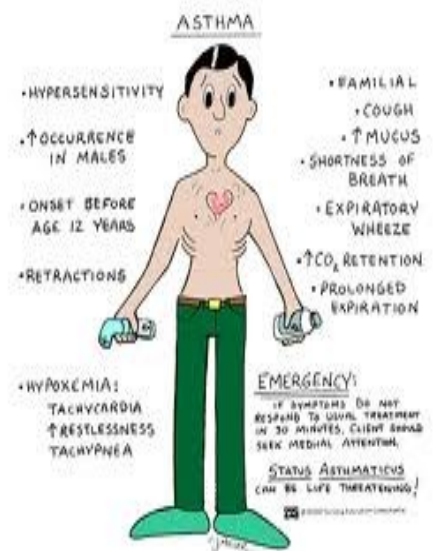
Diagnosis

There is currently not a precise physiologic, immunologic, or histologic test for diagnosing asthma. The diagnosis is usually made based on the pattern of symptoms (airways obstruction and hyperresponsiveness) and/or response to therapy (partial or complete reversibility) over time. The British Thoracic Society determines a diagnosis of asthma using a ‘response to therapy’

approach. If the patient responds to treatment, then this is considered to be a confirmation of the diagnosis of asthma. The response measured is the reversibility of airway obstruction after treatment. Airflow in the airways is measured with a peak flow meter or spirometer, and the following diagnostic criteria are used by the British Thoracic Society

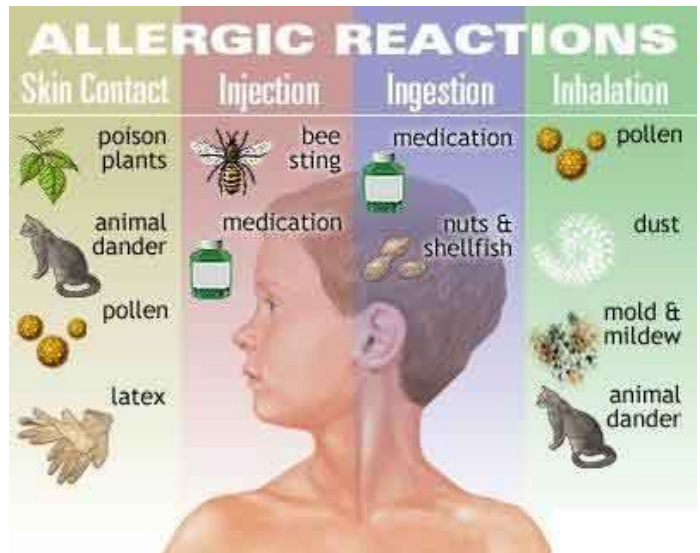
Clinical sign and symptoms

People with asthma experience symptoms when the airways tighten, inflame, or fill with mucus. Common asthma symptoms include coughing, especially at night, wheezing, shortness of breath, chest tightness, pain, or pressure. Still, not every person with asthma has the same symptoms in the same way



Nonpharmacologic therapy

Avoidance of known allergenic triggers can improve symptoms, and reduce medications use. Environmental triggers (e.g., animals) should be avoided in sensitive patients, and those who smoke should be encouraged to stop. Patients with acute severe asthma should receive supplemental oxygen therapy to maintain arterial oxygen saturation above 90% (above 95% in pregnant women and patients in heart disease).



Pharmacologic therapy

Intermittent asthma

Step 1

Preferred: Inhaled short-acting beta agonist, as needed

Persistent asthma: daily medication

Consult with asthma subspecialist if step 4 care or higher is required; consider consultation at step 3

Step 2

Preferred: Low-dose inhaled corticosteroid
nedocromil (formerly Tilade), or theophylline†
*Alternative:** Cromolyn (Intal), leukotriene receptor antagonist,

Step 3

Preferred: Low-dose inhaled corticosteroid, plus long-acting inhaled beta agonist
or
Medium-dose inhaled corticosteroid
*Alternative:** Low-dose inhaled corticosteroid, plus leukotriene receptor antagonist, theophylline†, or zileuton (Zyflo)‡

Step 4

Preferred: Medium-dose inhaled corticosteroid, plus long-acting inhaled beta agonist
*Alternative:** Medium-dose inhaled corticosteroid, plus leukotriene receptor antagonist, theophylline†, or zileuton‡

Step 5

Preferred: High-dose inhaled corticosteroid, plus long-acting inhaled beta agonist
and
Consider omalizumab (Xolair) for patients who have allergies

Step 6§

Preferred: High-dose inhaled corticosteroid, plus long-acting inhaled beta agonist, plus oral corticosteroid
and
Consider omalizumab for patients who have allergies

Each step: Patient education, environmental control, and management of comorbidities

Steps 2 to 4: Consider subcutaneous allergen immunotherapy for patients who have allergic asthma||

Quick-relief medication for all patients

Inhaled short-acting beta agonist as needed for symptoms. Intensity of treatment depends on severity of symptoms: up to three treatments at 20-minute intervals as needed. Short course of oral systemic corticosteroids may be needed

Use of inhaled short-acting beta agonist two or more days a week for symptom relief (not for prevention of exercise-induced bronchospasm) generally indicates inadequate control and the need to step up treatment

Step up if needed (first, check adherence, environmental control, and comorbid conditions)

Assess control

Step down if possible (and asthma is well controlled for at least three months)





Celebrities affected by lung disease



COPD

Definition

Chronic obstructive pulmonary disease (COPD) is a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible. The more familiar terms 'chronic bronchitis' and 'emphysema' are no longer used, but are now included within the

COPD diagnosis. COPD is not simply a "smoker's cough" but an underdiagnosed, life-threatening lung disease.

Prevalence

The WHO estimates that in 2000, 2.74 million people died of COPD worldwide. In 1990, a study by the World Bank and WHO ranked COPD

12th as a burden of disease; by 2020, it is estimated that COPD will be ranked 5th. According to the WHO, passive smoking carries serious risks, especially for children and those chronically exposed. The WHO estimates that passive smoking is associated with a 10 to 43 percent increase in risk of COPD in adults.

Diagnosis of COPD

Diagnosis

A COPD diagnosis is confirmed by a simple test called spirometry, which measures how deeply a person can breathe and how fast air can move into and out of the lungs. Such a diagnosis should be considered in any patient who has symptoms of cough, sputum production, or dyspnea (difficult or labored breathing), and/or a history of exposure to risk factors for the disease. Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools.



Clinical signs and symptoms

Clinical symptoms and signs, such as abnormal shortness of breath and increased forced expiratory time, can be used to help with the diagnosis. A low peak flow is consistent with COPD, but may not be specific to COPD because it can be caused by other lung diseases and by poor performance during testing.

Chronic cough and sputum production often precede the development of airflow limitation by many years, although not all individuals with cough and sputum production go on to develop COPD. The physical examination is normal in most patients who present in the milder stages of COPD.

When airflow limitation becomes severe, patient may have cyanosis of mucosal membranes, development of a 'barrel chest' due to hyperinflation of the lungs, an increased resting respiratory rate, shallow breathing, pursing of the lips during expiration, and use of accessory respiratory muscles.



Nonpharmacologic therapy

That COPD is an irreversible lung disease, speaks loudly for the importance of COPD treatment in slowing down the progression of the disease and improving quality of life.

While many medications are available to treat COPD, no drug has demonstrated effectiveness in halting the progression of the disease. Rather, the goal of drug therapy at this time is to maintain control of symptoms and prevent COPD exacerbation. **Smoking cessation** is the

Smoking cessation is the major ways reduce COPD

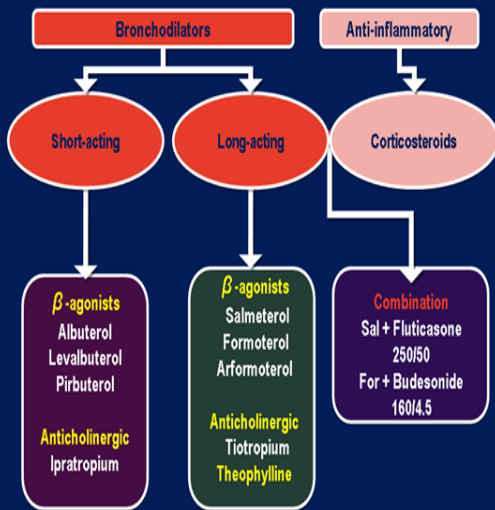
most effective strategy to reduce the risk of developing COPD and the only intervention proven to affect the long-term decline in FEV1 and slow the progression of COPD. **Pulmonary rehabilitation** programs include exercise training along with smoking cessation, breathing exercises, optimal medical treatment, psychosocial support, and health

education. Supplemental oxygen, nutritional support, and psychoeducational care (e.g., relaxation) are important adjuncts in a pulmonary rehabilitation program. **Annual vaccination** with the inactivated intramuscular influenza vaccine is recommended. One dose of the polyvalent pneumococcal vaccine is indicated for patients at any age.



Pharmacologic therapy

Existing Pharmacologic Treatment Options in COPD



Currently available pharmacologic treatments for COPD include bronchodilators and anti-inflammatory agents, as seen in Figure on left side. Bronchodilators are key for managing patients with symptomatic disease. These agents are given on a regular (long-acting agents) or as-needed (short-acting agents) basis. The main bronchodilator treatments for COPD are beta2 agonists, anticholinergic agents, and theophylline, and these drugs are used as monotherapy or in combination. Inhaled agents are generally preferred. Inhaled corticosteroids

are recommended as add-on therapy.

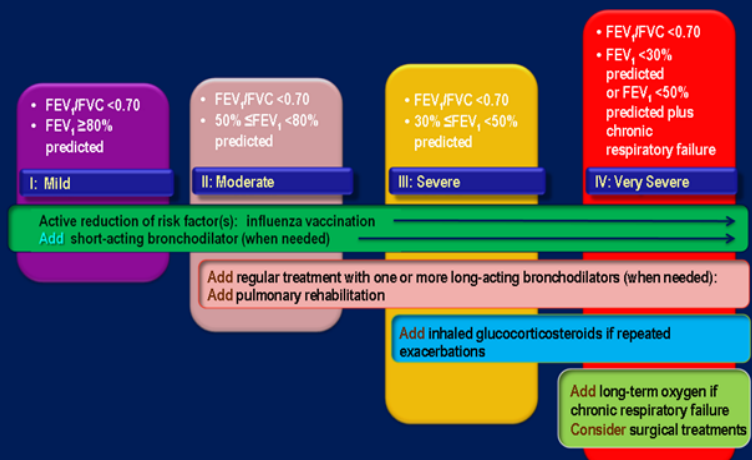
In patients with an FEV1 <50% predicted and those with repeated exacerbations, the combination of an inhaled glucocorticoid and a long-acting beta2 agonist is more effective than either therapy alone. Inhaled corticosteroid use has been associated with topical side effects, such as thrush and dysphonia. A meta-analysis of 11 randomized controlled trials looking at the safety of inhaled corticosteroids showed no increased risk of mortality at 1 year, but confirmed a significantly higher risk of pneumonia.

The GOLD guidelines

According to the GOLD guidelines, regular treatment with a long-acting bronchodilator is more effective and convenient than a short-acting bronchodilator for COPD management. Oxygen therapy is recommended for hypoxemic patients with very severe (stage IV) disease.

The goal of oxygen therapy is to obtain a SaO₂ of at least 90% or a PaO₂ to at least 60 mm Hg. Pulmonary rehabilitation should include education, exercise training, and nutrition counseling, and this program should be continued for at least 6 weeks to obtain optimal results. Finally, pulmonary rehabilitation is an important component of COPD management; however, its role is often under-recognized by patients and physicians.

GOLD Therapy at Each Stage of COPD



Common use of inhalers

RELIEVERS	CONTROLLERS	PREVENTERS
<p>1. Short-acting β_2-agonists</p> <p>Asthavent[®] MDI / DP-Haler[®] / Revolizer[®] (Salbutamol)</p> <p>Berotec[®] MDI (Fenoterol)</p> <p>Venteze[®] MDI (Salbutamol)</p> <p>Ventolin[®] MDI / Accuhaler[®] (Salbutamol)</p> <p>2. Anticholinergics</p> <p>Atrovent[®] MDI (Ipratropium Bromide)</p> <p>Ipvent-40[®] MDI (Ipratropium Bromide)</p> <p>Spiriva Handihaler[®] (Tiotropium)</p>	<p>Long-acting β_2-agonists</p> <p>Foratec DP-Haler[®] / Revolizer[®] (Formoterol)</p> <p>Oxis Turbuhaler[®] (Formoterol)</p> <p>Serevent[®] MDI / Accuhaler[®] (Salmeterol)</p>	<p>1. Inhaled Corticosteroids</p> <p>Alvesco[®] MDI (Ciclesonide)</p> <p>Beclate HFA[®] MDI (Beclomethasone)</p> <p>Budeflam DP-Haler[®] / Revolizer[®] (Budesonide)</p> <p>Budeflam HFA Gentle-Haler[®] (Budesonide)</p> <p>Flixotide[®] MDI / Accuhaler[®] (Fluticasone)</p> <p>Inflammid[®] MDI / Novolizer[®] (Budesonide)</p> <p>Pulmicort Turbuhaler[®] (Budesonide)</p> <p>QVAR[®] MDI (Beclomethasone)</p> <p>2. Leukotriene receptor antagonist</p> <p>Singular[®] tablets (Montelukast)</p>
<p>COMBINATIONS</p> <p>DP-Haler[®] / Revolizer[®] (Budesonide + Formoterol)</p> <p>Seretide[®] MDI / Accuhaler[®] (Fluticasone + Salmeterol)</p> <p>Symbicort Turbuhaler[®] (Budesonide + Formoterol)</p>		

How Does COPD Differ from Asthma?

1. The onset of asthma typically occurs during childhood or adolescence. COPD most often develops in smokers and former smokers who are in their mid-40s.
2. Exacerbations of asthma -- characterized by recurrent wheezing, shortness of breath, chest tightness and cough -- often have identifiable triggers such as allergens, cold air or exercise. However, exacerbations in COPD patients are commonly caused by respiratory tract infections.
3. With treatment, asthma patients have near-normal lung function and are symptom-free between exacerbations. COPD patients rarely experience a day without symptoms. Airflow obstruction in COPD sufferers is only partially reversible with smoking cessation and bronchodilator use.

Despite these distinctions, COPD is often misdiagnosed, and persons with COPD are treated instead for asthma. The first-line maintenance therapy for most patients with asthma is an inhaled corticosteroid, with the addition of a bronchodilator if needed to control symptoms. However, the reverse is true for the treatment of COPD. Bronchodilators are the first-line maintenance treatment for COPD.

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3. http://www.mts.org.my/resources/Guidelines_ChildhoodAsthma.html
4. <http://www.aarc.org/klein/versus.asp>
5. Asthma and COPD management and strategies for the primary care provider May 28, 2006.
6. COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. Mannino DM. Health, Centers for Disease Control and Prevention, 1600 Clifton Road, MS E-17, Atlanta, GA 30333, USA. dmmb@cdc.gov
7. <http://www.medscape.org/viewarticle/712114>

ADVERSE DRUG REACTION HOSPITAL SEGAMAT Jan– Mac 2013

No	Date	Drug	Adverse Reaction	Treatment	Reporter
1	1.2.2013	T. Simvastatin 20mg ON	Dizziness and alopecia	Medication was stopped	Miss Mira Marina Mahfodz (PF U41 PRP)
2	1.2.2013	C. phenytoin 300mg ON	Rash with itchiness and redness	Medication was stopped	Miss Soh Wei San (PF U41 PRP)
3	1.2.2013	IM Diclofenac sodium 75mg stat T. Diclofenac sodium 50mg TDS	Anaphylactic shock with low BP and rash	Medication was stopped Neb AVN 2:1:1 STAT IM adrenaline 1mg STAT IM hydrocortisone 200mg over 30 secs Slow IV Chlorpheniramine 10mg over 1 minute	Miss Soh Wei San (PF U41 PRP)

* No ADR reported in Jan and Mac 2013

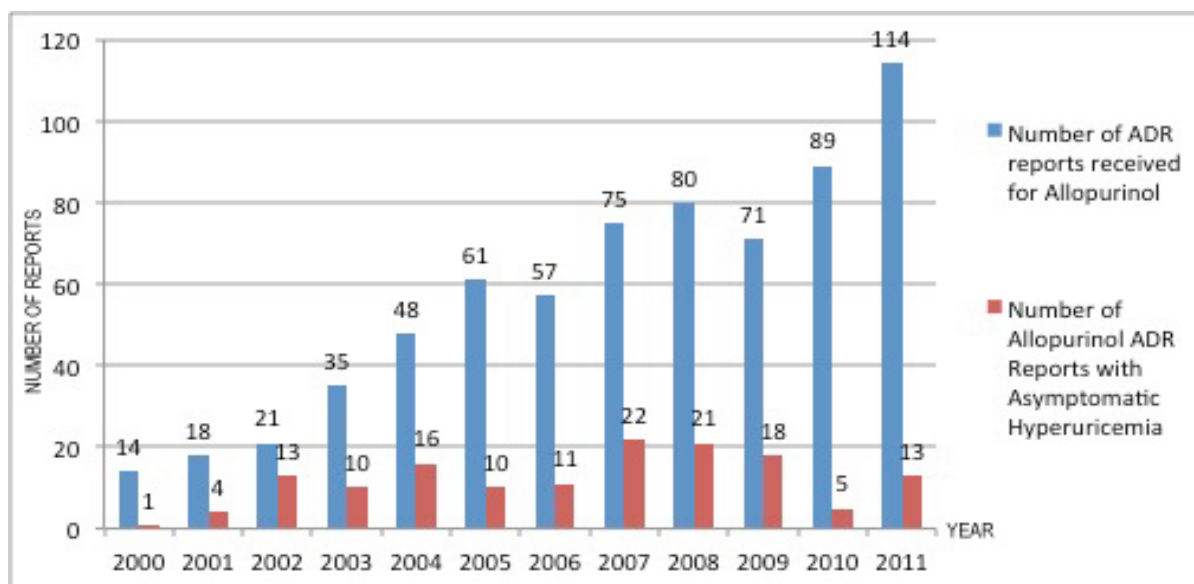
MADRAC UPDATES

ALLOPURINOL: AN UPDATE ON USAGE IN THE MINISTRY OF HEALTH FACILITIES AND RELATED ADVERSE CUTANEOUS DRUG REACTIONS By Vidhya Hariraj

Background In year 2010, CK Lee, A. Mohd Affandi et al. retrospectively reviewed all adverse cutaneous drug reactions (ACDR) reports associated with ingestion of allopurinol received by MADRAC from January 2001 till December 2009. Published in the MADRAC Bulletin in December 2010, the results showed a significant proportion of patients experienced serious ACDRs with a fraction of them succumbing to their complications. Allopurinol induced ACDRs were reported in 437 patients, accounting for 3.75% of the total ACDRs reported to MADRAC. The incidence in Malaysia was 15 cases per hundred thousand population years. The commonest ACDRs described were Steven Johnson Syndrome (27.3%), followed by maculopapular exanthem (23.1%) and Toxic Epidermal Necrolysis (5.7%).

Further analysis of data by MADRAC shows that 21% of total ADR reports received since year 2000 were due to use of allopurinol for asymptomatic hyperuricemia. **Chart 1** shows a breakdown from year 2000 till 2011 of ADR reports received for allopurinol and number of cases associated with asymptomatic hyperuricemia. An alarming 60% of ADR reports received for allopurinol in 2002 was related to its inappropriate use. In 2004, an advisory was disseminated to remind prescribers of allopurinol indication following which, a decline was seen in 2005 and 2006. However, from 2007 to 2009 an increasing trend of 25-30% was observed. The figures triggered a signal for risk minimisation actions which is explained further in the next section. Although the number of cases showed a decreasing trend and dropped to 5 (5.6%) and 13 (11.4%) cases in 2010 and 2011 respectively this still remain a cause for concern.

Chart 1: Number of adverse events reports received by NPCB associated with allopurinol 2000-2011



Source: National Centre for Adverse Drug Reaction Monitoring

Asymptomatic Hyperuricemia And Allopurinol Associated Adverse Cutaneous Drug Reactions (ACDRs)

Allopurinol is used for reducing uric acid formation in conditions where uric acid deposition has already occurred or is a predictable clinical risk. Almost 10% of adults are documented to have hyperuricemia at least once in their lifetime. There are many causes that lead to hyperuricemia involving overproduction, undersecretion or both. Asymptomatic hyperuricemia is common and does not in itself constitute a disease.

Allopurinol use in patients with asymptomatic hyperuricemia has been acknowledged as a suggested contributory risk factor for allopurinol hypersensitivity syndrome. Other factors include recent onset (several months) of allopurinol therapy, HLA-B58 allele in subjects of Han Chinese, European ancestry and chronic kidney disease, concomitant thiazide diuretic therapy and high allopurinol dose relative to renal function.

Risk Minimisation Actions of Allopurinol Associated ACDRs

MADRAC subsequently made recommendations on risk minimisation strategies and the following actions have been taken to curb the inappropriate use of allopurinol in light of these serious cutaneous adverse events:

In November 2008, the Director General of Health issued a circular to all State Health Directors to remind prescribers on allopurinol **prescribing indications** (KKM 87/91/19/1/0(12)).

In August 2011, another circular (KKM 87/P1/19/1/0(25)) was issued to inform all healthcare professionals that **uric acid test should no longer be included as part of routine renal function test but conducted only when patient presents with clinical symptoms of gout.**

Concurrently, in August 2011, the Ministry's Medicine List Review Panel Meeting (No.2/2011) **raised the category of allopurinol prescribers from Category B (by Medical Officer) to Category A/AKK (by Consultant/Specialist/Family Physician specialist only).**

The National Centre for Adverse Drug Reaction Monitoring will continue to monitor the adverse cutaneous drug reactions associated with allopurinol. Cutaneous drug reactions associated with inappropriate use of allopurinol need to be reduced and prevented as they cause significant morbidity and mortality. A progress report will be provided in 2013.

Reminder to healthcare professionals:

Primary prescribers are reminded to inform patients treated with allopurinol to **discontinue the drug at the first appearance of skin rash or other signs which may indicate an allergic reaction and seek immediate medical attention.**

Pharmacists should screen prescriptions for allopurinol to verify its indication.

All healthcare professionals are required to report any adverse event associated with allopurinol to NPCB.

Special Advice to Healthcare Professionals

No.	Registered Indications
1	Chronic gout/gouty arthritis
2	Uric acid nephropathy
3	Calcium oxalate renal calculi/uric acid renal calculi
4	Hyperuricemia 2° cancer chemotherapy/ radiation therapy
5	Hyperuricemia 2° blood dyscrasias
6	Hyperuricemia 2° enzyme disorders

1) Warnings and Precautions For the Use of Allopurinol are as follows:

WARNINGS AND PRECAUTIONS FOR USE OF ALLOPURINOL

- ✓ Allopurinol should be withdrawn **immediately** if a skin rash or other evidence of sensitivity occurs as this could result in more serious hypersensitivity reactions (including Stevens-Johnson syndrome and toxic epidermal necrolysis).
- ✓ Reduced doses should be used in patients with hepatic or renal impairment. Patients under treatment for hypertension or cardiac insufficiency, for example with diuretics or ACE inhibitors, may have some concomitant impairment of renal function and allopurinol should be used with care in this group.
- ✓ Asymptomatic hyperuricemia per se is generally not considered an indication for use of allopurinol. Fluid and dietary modification with management of the underlying cause may correct the condition.
- ✓ Patients with rare hereditary problems of galactose intolerance, the Lapp lactose deficiency or glucose-galactose malabsorption should not take this medicine.

Acute gouty attacks

Allopurinol treatment should not be started until an acute attack of gout has completely subsided, as further attacks may be precipitated. In the early stages of treatment with allopurinol, as with uricosuric agents, an acute attack of gouty arthritis may be precipitated.

Therefore it is advisable to give prophylaxis with a suitable anti-inflammatory agent or colchicine for at least one month. The literature should be referred for details of appropriate dosage and precautions and warnings.

If acute attacks develop in patients receiving allopurinol, treatment should continue at the same dosage while the acute attack is treated with a suitable anti-inflammatory agent.

2) The table below provides recommendations for diet, lifestyle modification and non-pharmacological management of gout:

Recommendations for diet, lifestyle modification and non-pharmacological management of gout:	
Encourage patients to:	Recommend patients to:
<ul style="list-style-type: none"> • Maintain an ideal weight • Consume low fat dairy, vegetable sources of protein and foods rich in vitamin C • Drink water > 2 L/day • Exercise moderately • Elevate and cool affected joints 	<ul style="list-style-type: none"> • Avoid (or limit) alcohol, particularly beer • Avoid (or limit) high purine foods such as red meat, shellfish, oily fish, liver, kidney, yeast extracts, sucrose and fructose containing soft drinks • Avoid high protein, low carbohydrate diets • Avoid dehydration • Avoid intense exercise and joint trauma

References:

CK Lee, A Mohd Affandi, YY Lee, CC Chang and R Baba. Allopurinol Induced Adverse Cutaneous Drug Reactions (ACDRs): A Review of MADRAC (Malaysian Adverse Drug Reaction Advisory Committee) Report from 2000-2009.

HE Dincer, AP Dincer, DJ Levinson, et al. Asymptomatic hyperuricemia: To treat or not to treat. Cleveland Clinic Journal Of Medicine August 2002; Volume 69, 8: 594-608.

Jeannie Chao and Robert Terkeltaub. A Critical Reappraisal of Allopurinol Dosing, Safety, and Efficacy for Hyperuricemia in Gout. Current Rheumatology Reports 2009; 11:135–140.

Zyloric Package Insert. Malaysia. [September 2011]

Bpac nz. The Management of Gout Revisited. Best Practice Journal, August 2011; 37: 34-40. <http://www.bpac.org.nz/magazine/2011/august/gout.asp>

Program Pemulangan Ubat

Pengenalan

- Kepatuhan pengambilan ubat adalah sangat penting bagi memastikan kesan rawatan yang optimum.
- Bagi ubat yang tidak digunakan, anda digalakkan untuk memulangkannya ke farmasi

Faktor Yang Menyumbang kepada Ubat yang Tidak Digunakan Lagi

- Pertukaran regimen ubat
- Pesakit telah berhenti rawatan ubat
- Pesakit tidak patuh terhadap rawatan ubat-ubatan
- Pesakit telah meninggal dunia
- Ubat diperolehi dari pelbagai sumber.

Mengapa ubat tidak patut dibuang begitu sahaja?

- Ubat-ubatan yang tidak digunakan lagi biasanya dibuang melalui mangkuk tandas, sinki atau dibuang bersama sampah domestic. Tindakan ini menjadi titik permulaan pencemaran alam sekitar.
- Kita perlu mengubah sikap bagi memastikan alam sekitar kita selamat untuk generasi masa depan

▶ Berikut adalah perkara yang anda perlu lakukan... Ia Sangat Mudah

LANGKAH 1

Kemaskan tempat simpanan ubat di rumah, asingkan ubat berlebihan & yang telah tamat tarikh luput.

LANGKAH 2

Bawa ubat tersebut ke unit farmasi di hospital / klinik kesihatan kerajaan yang berhampiran.

LANGKAH 3

Serahkan ubat tersebut ke farmasi hospital dan klinik kesihatan kerajaan untuk pelupusan yang betul.

LANGKAH 4

Beritahu kepada rakan dan saudara mara anda berkaitan Program Pemulangan Ubat.

▶ Fikir dahulu sebelum anda membuang



Jangan buang ubat ke dalam mangkuk tandas



Jangan buang ubat ke dalam tong sampah domestik



Jangan buang ubat ke dalam sinki

**PULANGKAN UBAT
BERLEBIHAN DAN TAMAT
TARIKH LUPUT KE FARMASI**

WHO Drug Information 2012– Safety and Efficacy Issues

Antipsychotics in children and adolescents: cardiometabolic reactions

Canada — Health Canada has received 29 reports of cardiometabolic adverse reactions suspected of being associated with second-generation antipsychotics (SGAs) in children and adolescents under 18 years of age. In Canada, no SGAs are authorized for use in children or adolescents, with one recent exception authorized for use only in adolescents of 15 to 17 years old for the treatment of schizophrenia.

Excess weight and obesity in the population are increasing problems throughout the Western world, and this rise has also been observed in children and adolescents (1). Weight gain and obesity are known to be associated with diabetes, dyslipidaemia and hypertension (2). In addition, weight gain is a well-established adverse reaction to second-generation antipsychotics (SGAs) (1).

In Canada, there are seven marketed second-generation antipsychotics: clozapine, risperidone, olanzapine, quetiapine, paliperidone, ziprasidone and aripiprazole. Recently, aripiprazole (Abilify®) was authorized for the treatment of schizophrenia in adolescents 15 to 17 years old (3). Previously, there were no authorized indications for the use of SGAs in children or adolescents under 18 years of age in Canada. Paediatric drug use, in many circumstances, has been based primarily on information extrapolated from studies involving adults, as well as from other types of scientific evidence, including case reports, open studies of clinical experience and controlled clinical trials (4, 5). Use of these drugs in the paediatric population has increased substantially over the last decade (6–8). According to one estimate, antipsychotic drug prescriptions for children and youth in Canada increased by 114% from 2005 to 2009 (4). Despite his increased use, data regarding their safety are limited (2).

The cardiometabolic effects of SGAs in paediatric patients, including age inappropriate weight gain, obesity, hypertension, and lipid and glucose abnormalities, are of concern (8). Furthermore,

children and adolescents with mental health problems often have multiple cardiovascular risk factors, including poor nutrition, inadequate exercise, substance abuse and lack of adequate healthcare monitoring (2, 9). Some studies have shown that youth using antipsychotic agents may be at a higher risk of weight gain and metabolic effects than adults who use the same drugs (2, 7, 10). If weight gain is established in youth, it tends to persist into adulthood (10). Because of differences in absorption, distribution and metabolism of antipsychotics in the paediatric population, higher doses per weight are required than in adults to achieve similar efficacy (2). Cardiometabolic effects are problematic during childhood because they tend to be predictors of adult obesity, metabolic syndrome, hypertension, cardiovascular morbidity and malignant disease (2, 7, 8).

Adverse effects such as weight gain have been found to vary significantly by SGA agent. Clozapine and olanzapine seem to be associated with the highest risk of clinically significant weight gain in children and adults (1, 2, 7). Risperidone and quetiapine generally show modest risk, whereas ziprasidone and aripiprazole are associated with the lowest risk. Limited data are available for paliperidone (4). The risk of lipid elevation and increased blood sugar appears to be greatest with olanzapine (11).

Extracted from Canadian Adverse Reaction Newsletter, Volume 22, Issue 1, January 2012

Quetiapine: information updated United Kingdom

— The manufacturer of quetiapine and quetiapine prolonged release has informed healthcare professionals of an update to the special warnings and precautions section of the summary of product characteristics concerning weight gain, hyperglycaemia and metabolic risk. Weight gain has been reported in patients who have been treated with quetiapine, and should be monitored and managed as clinically appropriate in accordance with antipsychotic guidelines. Hyperglycaemia and/or development or exacerbation of diabetes occasionally associated with ketoacidosis or coma has been reported rarely, including some fatal cases. In some cases, a prior increase in body weight has been reported which may be a predisposing factor. Patients treat-

treated with any antipsychotic agent including quetiapine, should be observed for signs and symptoms of hyperglycaemia, (such as polydipsia, polyuria, polyphagia and weakness) and patients with diabetes mellitus or with risk factors for diabetes mellitus should be monitored regularly for worsening of glucose control. Weight should also be monitored regularly. Given the observed changes in weight, blood glucose and lipids seen in clinical studies, there may be possible worsening of the metabolic risk profile in individual patients, which should be managed as clinically appropriate.

Reference: Medicines and Healthcare Products Regulatory Agency (MHRA). Communication from AstraZeneca UK at <http://www.mhra.gov.uk/Safetyinformation/Safetywarningsalertsandrecalls/Safetywarningsandmessagesformedicines/index.htm>

Ondansetron: QT prolongation United States of America — The Food and Drug Administration (FDA) has informed healthcare professionals and the public that preliminary results from a recently completed clinical study suggest that a 32 mg single intravenous dose of ondansetron (Zofran®, ondansetron hydrochloride, and generics) may affect QT interval prolongation. The updated label will state that no single intravenous dose should exceed 16 mg and that ondansetron can continue to be used in adults and children with chemotherapy-induced nausea and vomiting at the lower intravenous dose recommended in the drug label of 0.15 mg/kg administered every 4 hours for three doses. The new information on QT prolongation does not change any of the recommended oral dosing regimens for ondansetron. It also does not change the recommended lower dose intravenous dosing of ondansetron to prevent postoperative nausea and vomiting. Electrolyte abnormalities (e.g., hypokalaemia or hypomagnesaemia) should be corrected prior to the infusion of ondansetron.

Reference: FDA Drug Safety Communication, 29 June 2012 at <http://www.fda.gov/Drugs/DrugSafety/ucm310190.htm>

Serotonergic medicines: cerebral vasoconstriction syndrome New Zealand

— A recent Medsafe review confirmed that cases of reversible cerebral vasoconstriction syndrome (RCVS) have been reported in association with the use of the serotonergic medicines. Cases have been reported with duloxetine, sertraline, citalopram, paroxetine, fluoxetine and sumatriptan. However, data is currently inadequate to confirm a causal association. RCVS should be considered in the differential diagnosis of thunderclap headaches when other causes have been excluded. RCVS is thought to be under-reported for many reasons including lack of awareness of the condition and difficulties in confirming the diagnosis. The current data on RCVS comes primarily from case series conducted in France, Taiwan, and USA (1–3). The case series include 262 patients who experienced RCVS. RCVS is a unifying term used to describe a diverse range of conditions characterized by recurrent thunderclap headaches and reversible segmental cerebral arterial vasoconstriction on angiogram. Conditions include Call-Fleming syndrome, benign angiopathy of the CNS, postpartum angiopathy or idiopathic thunderclap headache. RCVS classically presents with sudden-onset and severe headaches that recur over a 1–3 week period (4). The headaches may be accompanied by nausea, vomiting and photophobia. Neurological complications occur in up to 50% of patients. Complications include seizures, cortical subarachnoid, haemorrhage, ischaemic stroke and intracerebral haemorrhage. The majority of patients recover fully. However, neurological deficits were found to be permanent in 3–9% of patients in a prospective case series (5).

Extracted from Prescriber Update 2012; 33(2) June 2012 at http://www.medsafe.govt.nz/profs/PUArticles/PDF/PrescriberUpdate_



ANNOUNCEMENT

WELCOME TO PHARMACY DEPARTMENT

1. MISS KOMALA DEVI MARIAPPAN

PHARMACIST U41

2. PN FAZENON OTHMAN

PHARMACY ASSISTANT U36



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"Instead of prescribing 100 capsules, how about just taking one large one?"