Consultant's Corner

Practical Answers To Your Everyday Questions

Chlamydia Retreatment

1.

How long after chlamydia and retreatment can the patient be retested?

Question submitted by:
Dr. Omayma Fouda
Toronto, Ontario

Generally, test-of-cure for chlamydia treatment is not necessary unless the patient is pregnant or less than 14-years-old, compliance is a concern, symptoms persist, reinfection may have occurred, or the site of infection is the nongenital tract. If a test of cure is necessary, it should be done three to four weeks after completion of treatment if a nucleic acid amplification test is used or five to seven days after treatment if culture is used. Testing sooner may provide inaccurate false positive results.

Many women and men who have had chlamydia are at increased risk of repeated infections; thus, it is suggested that chlamydia testing be conducted three to six months after treatment or within a year at their next annual visit if they do not return sooner. Most new infections result from lack of treatment in the partner and reinfection or exposure to a new infected partner. As chlamydia can cause pelvic inflammatory disease, resulting in infertility, it is important to be vigilant with these patients.

Answered by:
Dr. Cathy Popadiuk

Bleeding Risk with Dabigatran and Melatonin Supplement

2.

Is there an increased risk of bleeding with dabigatran and melatonin supplement?

Question submitted by:
Dr. S. Shore
Langley, British Columbia

To the author's knowledge, there is no current evidence to support or refute an interaction of melatonin with dabigatran.

Answered by:
Dr. Cyrus Hsia and
Dr. Kang Howson-Jan

Eyelid Dermatitis

3.

What are the common causal factors for eyelid dermatitis? What is the pharmacological treatment and management?

Question submitted by:
Dr. N. Aptekar
Brampton, Ontario

The eyelids are a vulnerable area for the development of dermatitis. They are subject to environmental exposure — wind, dryness, and airborne allergens (such as pollens). The eyelid skin is delicate and has a thin barrier that is easily disrupted, which can lead to irritant dermatitis and flares of atopic dermatitis. Also, common allergens can contact the area directly, such as substances transferred from the hands (nail polish components, nickel, hair dyes), which may lead to allergic contact dermatitis. In short, persistent eyelid dermatitis needs a meticulous work-up looking for endogenous factors like atopy, icthyosis, and seborrhea, as well as exogenous factors, such as irritants and allergens. We use bland moisturizing lotions or creams, mild steroids, and, where appropriate, topical calcineurin inhibitors, therapeutically.

Answered by:
Dr. Scott Murray

Possible Gastrointestinal Hemorrage with SSRI Use

4.

Please comment on the risk of gastrointestinal hemorrhage with SSRI use. Who is at risk?

Question submitted by:
Dr. Heather Sylvester
Stratford, Ontario

Several recent reports have implicated SSRI usage in increased risk of upper gastrointestinal (GI) hemorrhage. The impairment of the integral role of serotonin in hemostasis through platelet aggregation dysfunction has been identified as a possible cause of GI bleeds, although the specific mechanism has not been clearly defined.[1] The significance of bleeding risk associated with SSRI usage has been variable, with the greatest risk being identified in patients with concomitant NSAID and SSRI use in some studies.[2-7] Usage of concomitant PPI therapy has been found to diminish this elevated bleeding risk in recent data, although the significance of the protective effect has not been clarified.[7,8] In the context of the existing data, it is reasonable to consider SSRI usage as conveying some risk of upper GI hemorrhage, and caution is warranted in patients with risk factors for peptic ulcer disease. Furthermore, reversible risk factor modification and the use of concomitant PPI therapy is indicated in patients with significant risk.

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Answered by:
Dr. Theodore Xenodemetropoulos

Treating High LDL and Muscular Dystrophy

5.

A patient with high LDL also has muscular dystrophy. Besides diet, how can her LDL be reduced? What would be the pharmacological method?

Question submitted by:
Dr. Salman Augla, Carbonear, Newfoundland

Statins are by far the most efficacious drugs to reduce LDL cholesterol. There are case reports of patients whose muscular or neurologic diseases were "unmasked" by statins. These include myotonic muscular dystrophy types 1 and 2.[1] To my knowledge, no form of muscular dystrophy is a contraindication to statin therapy. Serious muscle damage, while rare, varies among the statins; lovastatin and simvastatin are somewhat more likely to cause muscle damage.[2] Other aggravating factors include reduced kidney or liver function, hypothyroidism, diabetes, vigorous exercise, and drug-drug interactions, (*e.g.* with gemfibrozil).[2] We recommend that patients who are prescribed statins report new onset muscle pain or weakness. Creatine kinase should then be measured. If it's 10 times or more than the upper limit of normal, stop the statin.

Surveillance of your patient may be more complicated if muscle weakness currently is a prominent symptom. Remember that the absolute benefit of statins in primary prevention, particularly for the short term (less than five years), is modest. Other alternatives include ezetimibe and colesevelam, but there are few studies showing that these reduce morbidity or mortality.

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Answered by:
Dr. Thomas W. Wilson

Estrogen and Progesterone Replacement

6.

What is the appropriate dosage of estrogen and progesterone replacement for a woman with premature ovarian failure, and what prescription is available?

Question submitted by:
Dr. Bernadette Yuan
Richmond, British Columbia

Premature ovarian failure (POF) may occur spontaneously or as a result of therapeutic treatments, such as a surgical oophorectomy, chemotherapy, or directed radiation. Estrogen is required to prevent osteoporosis and relieve symptoms of hot flashes, night sweats, and vaginal atrophy. If the uterus is still in place, then progesterone must be part of the regimen for hormone replacement therapy (HRT) to protect the endometrial lining from hyperplasia or cancer. There is no one best regimen for HRT in POF. There are a plethora of preparations now available for this purpose in various forms, such as oral pills, transdermal patches and gels, and vaginal pills and rings. Treatment depends upon patient preference, side effects, cost, and physician familiarity with the regimens. In a young woman with a uterus, a low-dose oral contraceptive pill with ethinyl estradiol and progesterone taken continuously, may be satisfactory and easy.

The minimum dose of estrogen effective for maintenance of bone health is 0.625 mg of conjugated estrogen (CE) or estropipate, 0.5 mg of micronized estradiol, or 50 mg of transdermal estradiol per day. Progesterone can be continuously or cyclically given and is available in various oral and patch preparations, some of which combine the estrogen and progesterone for ease of administration. The minimum dose of progesterone to protect the endometrium for a 0.625 mg dose of CE is 2.5 mg medroxyprogesterone acetate (MPA) daily or 10 mg dose of MPA cyclically for 10 days (monthly).

If the patient is experiencing menopausal symptoms, the dose of estrogen can be increased with a commensurate increase in progesterone if taken daily. For women who don't want to take oral preparations, the transdermal patch may be more appealing, allowing a more physiologic, continuous infusion of estrogen. The patch, however, may cause local irritation or fall off; thus, estrogen gel applied daily or the vaginal ring, changed every three months, may be more acceptable alternatives. The treatment must be tailored to each individual patient and is usually discontinued when natural menopause would be reached at age 50 or 51.

Answered by:
Dr. Cathy Popadiuk

How Should the SMART Trial be Interpreted?

7.

How should the Symbicort Maintenance and Reliever Therapy (SMART) trial be interpreted? Do we still have to give our patients short acting bronchodilators (SABAs)?

Question submitted by:
Dr. Steve Coyle
Winnipeg, Manitoba

The SMART strategy for treating asthma depends upon the patient using Symbicort both as maintenance therapy, for example two doses twice each day, and, in addition, using single doses of budesonide/formoterol as required to relieve symptoms. To be effective, it requires careful adherence to the maintenance dose and the exclusive use of budesonide/formoterol as a reliever. Although there has been a tendency to continue the use of short-acting β2-agonists before exercise, the current belief is that pre-exercise bronchodilators should not be needed in those with well-controlled asthma. Budesonide/formoterol would thus be a more appropriate pre-exercise medication in those using SMART. In short, we should not still have to give our patients on SMART a short-acting β2-agonist.

Answered by:
Dr. Robert Cowie

Treatment for Severe Chemotherapy Induced Mucositis

8.

Is an effective treatment for severe mouth ulcers in a patient with chemotherapy induced mucositis available?

Question submitted by:
Dr. George Linn
Kingston, Ontario

Oral mucositis, including the development of oral ulcers, is a common adverse effect of systemic chemotherapy due to mucosal injury via direct cellular effects, generation of reactive oxygen species, and production of proinflammatory cytokines. In mild cases of chemotherapy-induced mucositis, this may be managed with nonpharmacological mouth rinses, such as club soda or a saline and baking soda solution (*e.g.,*. 1 tsp of salt and 1 tsp of baking soda in 1 L of water).[1] Although evidence is lacking, in patients with refractory, severe mucositis, a "magic mouthwash" cocktail is commonly used. Multinational association of supportive care in cancer (MASCC) guidelines state that "topical anesthetics or other agents should be considered to promote oral comfort."[2-3] Various formulations for "magic mouthwash" exist with commonly used ingredients, including xylocaine viscous, diphenhydramine, magnesium hydroxide/aluminum hydroxide, nystatin, and corticosteroids.[4-5] In Alberta, for example, Dr. Akabutu's mouthwash consists of 60 cc of xylocaine viscous (2%), 50 ml of nystatin suspension (100,000 U/ml), 125 ml of normal saline, 60 mg of hydrocortisone (three tablets of 20 mg), and 3 to 5 ml of glycerine to yield a total volume of 240 ml. Instructions are to swish 30 ml of solution around the mouth and throat for one minute and to spit out the excess, to be used every four to six hours as needed. Whenever possible, patients should also avoid eating or drinking for approximately 30 minutes after its use. Infrequently, in severe cases, systemic opioids, such as oral morphine or parenteral opioids may be required.

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Answered by:
Dr. Roger Y. Tsang

Vaccinations for Multiple Sclerosis Patients

9.

Should patients with multiple sclerosis avoid stimulating their immune system with vaccinations?

Question submitted by:
Dr. Eric Teboul
St-Laurent, Québec

There is no evidence that vaccinations are unsafe in multiple sclerosis (MS) patients or that they cause relapses. There is only one study to date that addressed this issue by Confavreux *et al.* that found the relative risk of relapse after immunization was 0.71 (95% CI 0.40 to 1.26). The exception is live attenuated virus vaccines — although no evidence supports the risk in MS patients, there is a hypothetical risk, as it is well known that infections can increase disease activity in MS patients.

Answered by:
Dr. Sarah A. Morrow

Reference
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RBBB Finding in a Healthy 18-year-old Athlete

10.

Incomplete right bundle branch block (RBBB) in a healthy 18-year-old athlete is found. Is there a need for further testing?

Question submitted by:
Dr. Len Grbac
Etobicoke, Ontario

A right bundle branch block (RBBB) is diagnosed based on electrocardiographic criteria of a QRS greater than or equal to 120 msec, RSR1 pattern in V1, and a slurred S wave in leads I and V6. The diagnosis of an incomplete right bundle branch block (iRBBB) is present when the same electrocardiographic criteria as above are present, but the QRS width is between 100 and 120 msec. iRBBB is commonly seen as a normal finding in healthy individuals, athletic or not, and, although more common in younger individuals, it can be a normal variant at any age.

In an asymptomatic individual with a good exercise capacity and no history of syncope or abnormal cardiac physical examination findings, it should not be a cause for concern or prompt further testing.

Answered by:
Dr. Jennifer Rajala and
Dr. Brett Heilbron

Latest Canadian Guidelines for Lipid Reduction

11.

What are the latest Canadian guidelines for lipid reduction in reference to stroke prevention?

Question submitted by:
Dr. Paul Stephan
Scarborough, Ontario

Recommendations for CV risk reduction include thromboembolic stroke prevention. The Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of CVD have been issued recently and can be found online.[1] These guidelines represent a simplified, yet more aggressive approach to risk stratification and dyslipidemia management. In particular, patients found to be at intermediate risk, based on the Framingham Heart Study (Framingham 10-year risk score between 10 to 20%), have been added to the high-risk category.[2]

It is recommended that intermediate-risk patients be considered for lipid-lowering treatment if their LDL cholesterol levels exceed 3.5 mmol/L, if total cholesterol/HDL ratio is over 5.0, or if they have a positive family history of premature CVD (age < 60 years). The primary LDL cholesterol targets for patients with established CVD or diabetes mellitus, as well as for patients with a Framingham 10-year risk score of 10% or above, are an LDL cholesterol level below 2.0 mmol/L or a relative reduction in LDL cholesterol of at least 50%.

In patients felt to be low-risk (those with a Framingham 10-year risk score below 10%), lipid lowering treatment is recommended if their LDL cholesterol level exceeds 5.0 mmol/L; the treatment target is a relative reduction in LDL cholesterol of at least 50%.

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Answered by:
Dr. Theodore K. Fenske

Has CD24 Been Released for Clinical Use?

12.

Has the biomarker for cluster of differentiation 24 (CD24) oncogene been released for clinical use in detecting colon cancer, and is it a valuable tool?

Question submitted by:
Dr. R. Dlin
Edmonton, Alberta

CD24 has recently been identified as a gene implicated in cell-to-cell interaction as well as cellular proliferation and adhesion functions.[1,2] It has been implicated in the acceleration of neoplastic growth and metastasis through a variety of complex intracellular pathways and is highly expressed in colon cancer cells at an early stage of carcinogenesis.[1,2] As such, the use of the CD24 oncogene as a target for early detection and therapy for colon cancer has been proposed, although no translational applications of this knowledge/technology have been released in clinical medicine, to date.

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Answered by:
Dr. Theodore Xenodemetropoulos

Detecting the Existence of Pernicious Anemia

13.

If a patient has macrocytic anemia with low B12 serum levels, what tests should be done to prove the existence of pernicious anemia?

Question submitted by:
Dr. Constantine Vitou
Town of Mount Royal, Québec

To prove the existence of pernicious anemia, a Schilling test is required. However, this test is no longer available in most centres, and there are no good alternatives to the Schilling test. Some have advocated intrinsic factor antibody measurement (poor sensitivity), serum gastrin or pepsinogen I testing (poor specificity), or a combination of these tests for pernicious anemia. Nonetheless, these tests are expensive, difficult to obtain, and do not help to definitively diagnose the underlying cause.

Please note that a Schilling test is ideally used to determine the etiology of B12 deficiency, not to confirm it. There are many other causes of a macrocytic anemia that should be ruled out. Furthermore, a low serum B12 level does not mean that there is a functional B12 deficiency. The reason for this is that serum B12 level measures total B12 bound to transcobalamin proteins I, II, and III. Only a small fraction of vitamin B12 bound to transcobalamin II is physiologically important for cellular function. To confirm B12 deficiency, one should have at least two separate values of low serum B12 levels or a low serum B12 value with an elevated methylmalonic acid level. An elevated fasting homocysteine level will also be present.

Answered by:
Dr. Cyrus Hsia and
Dr. Kang Howson-Jan

Management of COPD Patients

14.

What is your recommended management of patients with chronic obstructive pulmonary disease (COPD) colonized with pseudomonas?

Question submitted by:
Dr. S. Giles
Sioux Lookout, Ontario

It may be difficult to determine that a patient with COPD has been colonized with pseudomonas. In many instances, patients with COPD might transiently have positive sputum cultures for pseudomonas, and, in the context of a clinical exacerbation, the use of antibiotics appropriate for treating pseudomonas is recommended. In general, colonization with pseudomonas, especially mucoid strains in patients with COPD, is associated with a worse prognosis and with more frequent acute exacerbations of COPD. What is less certain is whether attempts to eradicate the organism can be successful and whether that has a significant impact on the course of the disease. The process of eradicating pseudomonas found to be colonizing the airways is controversial and complex.

Answered by:
Dr. Robert Cowie

Is Depression an Independent Risk Factor for CV Death?

15. Is depression an independent risk factor for CV death?

Question submitted by:
Dr. L. Grbac
Etobicoke, Ontario

An independent association between depression, or depressive symptoms, and mortality from CVD has been described in the literature.[1,2] This association appears to be strongest for those experiencing depressive symptoms while admitted to the hospital for myocardial infarctions, unstable angina, or heart failure. Moreover, the severity of the depressive symptoms may be correlated with the severity of the recent CV event. For many patients, depression will resolve during follow-up care.[1]

Patients with depression have an increased risk of metabolic changes that may place them at increased risk of CV complications.[3,4] These include increases in weight, smoking, nonadherence with regards to taking their medication(s) — and to potentially having a negative reaction to the medications used in the treatment of depression, as they may cause ECG changes (*e.g.* citalopram, fluoxetine, paroxetine, and venlafaxine).[3,5] However, the exact mechanism has not been completely described.[3]

Of the ten studies exploring the possible association between depression and mortality after myocardial infarction (completed prior to a study by Stewart, *et al.*) eight studies found no statistically significant association between depression and mortality after adjustment for potential confounding factors.[1]

More recent studies examining the possible association between depressive symptoms and CV have demonstrated a clear association.[2, 6-7] Therefore, more recent evidence in the literature suggests that depression may be an independent predictor of all-cause mortality and CV death. As such, additional studies evaluating whether treatments for depression may improve survival and/or reduce future hospitalizations are warranted.[2]

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Answered by:
Dr. Joel Lamoure

Drug Interaction between Paroxetine and Tamoxifen?

16.

Is there a significant drug interaction between paroxetine and tamoxifen in breast cancer treatment?

Question submitted by:
Dr. Philip K. Ng
Port Coquitlam,
British Columbia

There is a significant drug interaction between fluoxetine and tamoxifen that results in an increased risk of death from breast cancer if these medications are taken over several years. Fluoxetine is among the antidepressants that inhibit the CYP2D6 liver enzyme that converts tamoxifen to its active tumour-fighting form endoxifen. A large population study showed that the longer women on tamoxifen took fluoxetine, the higher the likelihood of tumour recurrence. There was one extra breast cancer death for every 20 women treated over five years of follow-up with this antidepressant-tamoxifen combination.[1] Other antidepressants that inhibit CYP2D6 include sertraline hydrochloride and paroxetine, but these studies have been of smaller size and shorter duration, resulting in less conclusive outcomes, particularly as a greater proportion of women take fluoxetine.[2] Venlafaxine and citalopram have less inhibitory effects on CYP2D6 and are thus considered better alternatives for antidepressant treatment in breast cancer patients taking tamoxifen.

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Answered by:
Dr. Cathy Popadiuk

Treatment Options for Syringomas

17.

What are the treatment options for syringomas and their side effect profile?

Question submitted by:
Dr. Gerald Boey
Vancouver, British Columbia

Syringomas are benign, but they are a cosmetic nuisance. Treatment modalities have included dermabrasion and various methods of excision, such as cryosurgery, electrodesiccation, and ablative laser surgery, all of which can raise the risk of scarring or dyspigmentation. Topical and oral retinoids can be tried, but they have a mild effect at best in my experience. There are some reports of topical atropine being useful for the rare case of pruritic eruptive syringomas. The variable response in individual cases requires a careful assessment of possible therapies.

Answered by:
Dr. Scott Murray

Treating Pediatric Anxiety

18.

Why do physicians treat pediatric anxiety much less readily than adult anxiety? Is there a role for medications in pediatric treatment?

Question submitted by:
Dr. Julie Begin
Vaudreuil-Dorion, Québec

The biology of anxiety in children is noticeably less understood than in adults, and thus, the precipitants of anxiety and interventions for anxiety are much less well understood. Second, physicians tend to be much less likely to treat anxiety in children with pharmacotherapy than in adults for two very good reasons.

First, the efficacy of many drugs used in adults for anxiety has not been established in children, and, given the potential for differences in the basic neurobiology of anxiety between adults and children, it is entirely possible that there may be marked differences in efficacy.

Second, the risk of adverse events for many commonly used medications is likely to be greater among children than in adults, especially the potential for cognitive effects on children of school age.

The approach to anxiety in children needs to be focused on identifying and dealing with the source(s) of anxiety. Given the many unanswered questions with respect to treatment of anxiety in children, starting pharmacotherapy is not in the realm of the primary care physician; it should only be conducted after a specialist with expertise in the evaluation and management of anxiety in children has performed an assessment on the child.

Answered by:
Dr. Michael Rieder

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