1. Clues to bipolarity diagnosis

In a depressed person, what clues can lead to a diagnosis of bipolarity? Can a trial of selective serotonin reuptake inhibitors lead to a manic episode?

Submitted by:
Waguih Tannous, MD
Montreal, Quebec

The presentation of depressive symptomatology is the same for a depressed patient with or without bipolarity. The diagnosis of bipolarity depends on careful history taking. This involves exploring the patient's personal history for previous episodes of mania or hypomania, or any history of mood swings that could suggest Type II bipolarity. In taking the patient's history, it is important to explain to the patient and/or his family, what we mean by manic or hypomanic. Also, explain the difference between a complaint of insomnia, which is very common and the lack of sleep due to excessive energy and a high mood, in which case the patient enjoys being up all night without feeling tired or sleepy.

Another very important aspect of history taking relates to exploring the family history for any family member or relative that was previously diagnosed with a bipolar or manic depressive disorder. Such a history if positive, makes the physician suspicious that the current depressive episode may be part of an underlying bipolar disorder.

Finally, it is true that selective serotonin reuptake inhibitors (SSRIs) could trigger a manic episode in a patient who is predisposed genetically to have a bipolar disorder. If there is any concern that the current depressive episode is part of a bipolar disorder, then the depression should still be treated with an SSRI, but under close observation and the physician may decide to add a mood stabilizer such as lithium or olanzapine (atypical anti-psychotic with mood stabilizer properties), as a protection against triggering or switching to a manic episode.

Answered by:
Dr. Hany Bissada

2. Role of corticosteroids in viral bronchitis

What is the role of inhaled corticosteroids in acute viral bronchitis?

Submitted by:
Sakina Raj, MD
Calgary, Alberta

Acute viral bronchitis is a clinical syndrome lasting less than three weeks, characterized by cough with or without sputum production. It should be distinguished from an acute exacerbation of an underlying condition (*i.e.*, chronic obstructive pulmonary disease or asthma), pneumonia, or an acute upper respiratory tract infection (*i.e.*, common cold or sinusitis). The cause of the cough in acute bronchitis is thought to be due to acute bronchial mucosal injury and inflammatory response. Cough is often associated with clinical and physiological evidence of transient reversible airflow obstruction and bronchial hyperresponsiveness. As symptoms related to these changes are usually self-limited, there is no need to treat except with short-term antitussive therapy. Persistent cough (*i.e.*, more than three weeks) should lead to a search for an alternative diagnosis rather than empiric therapy with inhaled corticosteroids.

References
1. Braman SS: Chronic cough due to acute bronchitis. ACCP evidence-based clinical practice guidelines. CHEST 2006; 129(1 Suppl):95S-103S.

Answered by:
Dr. Paul Hernandez

3. Can a lung tumour raise blood glucose?

Can a lung tumour produce a substance that can increase blood glucose values?

Submitted by:
Andrea Coholic, MD
Timmins, Ontario

Yes, hyperglycemia may be seen in tumours of the lung in association with the ectopic secretion of corticotropin (ACTH). This is most commonly observed in the lung tumour subtypes of small cell carcinoma or bronchial carcinoids. In addition to hyperglycemia, this paraneoplastic syndrome of ectopic ACTH secretion may also be associated with clinical manifestations of edema, hypertension, hypokalemia and muscle wasting.

Answered by:
Dr. Sharlene Gill

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4. Why test for seasonal allergens?

What is the advantage of allergy testing for seasonal allergens when we have so many nasal sprays and eye drops?

Submitted by:
Peter C. Noble, MD
Oshawa, Ontario

With more effective topical and oral antihistaminic and anti-inflammatory therapies currently available, more patients are able to experience significant relief of their seasonal allergic rhinitis symptoms than ever before. However, there are several advantages to identifying the specific allergen to which the patient is sensitized. Firstly, when the exact culprit allergen is known (*e.g.*, tree pollen), patients can prepare in advance by premedicating with nasal corticosteroids, according to the seasonal patterns of allergen exposure in the local geographical region. They can also take some precautions when relevant (*i.e.*, wearing protective filtration masks when cutting grass). Some seasonal allergies can continue on an indoor basis (*i.e.*, mould allergies) where further prevention measures may be helpful. More importantly, despite the complaint of mainly seasonal symptoms, many patients may experience perennial rhinitis and identification of these allergens is important (*i.e.*, dust mite allergy), as treatment that takes this into account will also serve to reduce their seasonal symptoms. Finally, when prevention measures and medical treatment fails to ameliorate symptoms, identifying the patient's specific allergies is critical for application of effective allergen immunotherapy treatment.

Answered by:
Dr. Tom Gerstner

5. Prostatic calcification

What is the clinical significance of prostatic calcification on ultrasound?

Submitted by:
Hany Aeta MD
Cumberland, Ontario

Prostatic calcification (formerly called hyperechoic lesion) frequently seen on ultrasound is a nonspecific and a benign finding of unknown origin (perhaps sequela of former chronic prostatitis). No specific follow up is required. On the other hand, prostate cancer confirmed on transrectal ultrasound biopsies are either isoechoic or hypoechoic lesions.

Answered by:
Dr. François Péloquin

6. Gabapentin for chronic pain

Why and how should gabapentin be used for chronic pain?

Submitted by:
Gaétan Lavoie, MD
Matane, Quebec

Gabapentin is a drug known to have an effect on chronic pain, as opposed to acute pain.[1] As a group, anticonvulsant drugs have been used in the management of pain since the 1960s. Carbamazepine has also been commonly used for chronic pain, but the ease of titration, the relatively mild side effects of gabapentin, not to mention its efficacy, have made it another choice.

Dosing is simple with an initiation of 300 mg three times a day, with an increase of 300 mg to 600 mg per day at three day intervals, to a maximum of 1800 mg per day. Doses higher than that, up to 2400 mg, have not been shown to have a greater clinical effect.

Care should be taken with the elderly or those with impaired renal function, as gabapentin plasma clearance is reduced. Its normal elimination half life is about five hours to seven hours and it is unaltered by further multiple dosing. Morphine too can enhance gabapentin concentration, so in concomitant opioid therapy, gabapentin dosing should be reduced accordingly.

References
1. Wiffen PJ, McQuay HJ, Edwards JE, et al: Gabapentin for acute and chronic pain. Cochrane Database Syst Rev 2005; (3):CD005452.

Answered by:
Dr. Mo Verjee

7. DEXA scan for osteoporosis

Who should be screened for osteoporosis by a DEXA scan? How long should treatment for osteoporosis continue once the T-score is better than 2.5?

Submitted by:
Michael Yachnin, MD
Ottawa, Ontario

The Osteoporosis Society of Canada recommends screening everyone (men and women) after the age of 65 with a dual energy X-ray absortiometry (DEXA) scan. The screening age can be younger if risk factors are present (Table 1).

Table 1

Factors identifying those who should be assessed

Major risk factors	Minor risk factors
• Age ≥ 65 years	• Rheumatoid arthritis
• Vertebral compression fracture	• Past history of clinical hyperthyroidism
• Fragility fracture after age 40	• Chronic anticonvulsant therapy
• Family history of osteoporotic fracture (especially maternal hip fracture)	• Weight < 57 kilograms
• Systemic glucocorticoid therapy for more than three months	• Weight loss > 10% of weight at age 25
• Malabsorption syndrome	• Smoker
• Primary hyperparathyroidism	• Excess alcohol intake
• Propensity to fall	• Excess caffeine intake
• X-ray appearance of osteopenia	• Low dietary calcium intake
• Hypogonadism	• Chronic heparin therapy
• Early menopause (before age 45)	

The question of how long to treat is somewhat controversial. Previously, many recommended periodic drug holidays, but now that good long term bone quality data is available for bisphosphonates (seven years to 10 years) it may not be necessary to stop drug therapy. Practically, if the patient has modifiable risk factors and eventually falls into a lower risk category with treatment, then one might stop treatment temporarily, but continue close follow up and observation.

References
Brown JP, Josse RG: CMAJ 2002;167(10 suppl):S1-S34.

Answered by:
Dr. Michael Starr

8. Wait and see approach with osteopenia?

Should women with moderate to severe osteopenia be treated with a bone metabolism regulator or be watched until osteoporosis develops? And if treated, at what dosage?

Submitted by:
Gayle Garber, MD
Conception Bay South, Newfoundland

The T-score describes a patient's bone density, in terms of the number of standard deviations above or below the young adult mean normal reference range. A T-score of ≤ -2.5 confirms the diagnosis of osteoporosis in postmenopausal women and men over 50. Bone densities that are relatively well-preserved and are between -1 and -2.5 fall into the definition of osteopenia. Individuals with this degree of bone density should be further evaluated to ensure that there are no secondary causes for bone loss.

If an individual has already experienced a fragility fracture or has had height loss indicative of vertebral compression fractures, then antiresorptive drug therapy should be initiated, even at T-scores in the osteopenia range, as the risk of further fractures is considerable. According to the new Osteoporosis Canada guidelines, the 10-year absolute risk for fragility fracture is determined by age and bone mass density (BMD) T-score and could fall into one of three categories:

1. Low risk = < 10% chance of a fragility fracture in the next 10 years
2. Moderate risk = 10% to 20%
3. High risk = > 20%

After determining the fracture risk category based on age and BMD, two other key risk factors for fracture are also integrated in the fracture risk assessment:

1. Prior fragility fracture after the age of 40
2. Previous or current glucocorticoid use for more than three months

If either of these factors are present, the risk category is increased to the next level. Treatment is recommended for those in the high risk of fracture and individualized therapy is advised for those in the moderate risk of fracture. Preventive lifestyle changes, as well as calcium and vitamin D supplementation, with appropriate followup, is advised for those in the low fracture risk category.[1]

References
1. Siminoski K, Leslie WD, FrameH, et al: Recommendations for bone mineral density reporting in Canada. Can Assoc Radiol J 2005; 56:178-188.

Answered by:
Dr. Aliya Khan

9. Hyperparathyroidism

How would you treat a 39-year-old man with hypercalemia due to primary hyperparathyroidism as well as nephrolithiasis, testicular calcinosis and reduced glomerular filtration rate? He is not responding to pamidronate and zoledronic acid. There is a six- to eight-month waiting list before he can have his parathyroidectomy done.

Submitted by:
Iram Anees, MD
St-John's, Newfoundland

Surgery or destruction of the parathyroid adenoma is the only definitive treatment for primary hyperparathyroidism. This patient needs earlier surgery if it can be arranged.

Answered by:
Dr. Vincent Woo

10. Routine PSA screening for men over 50?

Should all males over age 50 be screened for PSA? What are the current recommendations?

Submitted by:
Balbina Russillo, MD
Town of Mount Royal, Quebec

This issue of prostate specific antigen (PSA) screening remains controversial. Serum PSA is of unknown value as a population screening test. Although there is evidence that it increases the rate of detection of earlier stage prostate cancers, there is little evidence to date that such early detection leads to reduced mortality.

Thus, it is recommended that a digital rectal examination (DRE) should be done annually in fit men between the ages of 50 and 70 or if obstructive or other urinary tract symptoms are present. Serum PSA is recommended as a diagnostic adjunct in men with lower urinary tract symptoms or suspicious DRE findings.

Men with at least 10 years of life expectancy between the ages of 50 and 70 can be made aware of the availability of PSA as a detection test for prostate cancer. They should be aware of the potential benefits and risks of early detection so they can make an informed decision as to whether to have the test performed.

References
1. BCCA Cancer Management Guidelines: Genitourinary/Prostate/Screening & Early Detection. BC Cancer Agency: (http://www.bccancer.bc.ca/ HPI/CancerManagementGuidelines/Genitourinary/Prostate/ScreeningEarlyDetection.htm); website accessed June 14, 2006.

Answered by:
Dr. Sharlene Gill

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11. Impact of NSAIDs on asthma

What is the impact of NSAIDS on asthma?

Submitted by:
Kenneth Armstrong, MD
Niagara Falls, Ontario

A significant but ill-defined percentage of asthma patients are intolerant to acetylsalicylic acid (ASA) and other non-steroidal anti-inflammatory drugs (NSAIDS). A subset has been well described for decades with the classic triad of asthma, rhinosinusitis with nasal polyps and ASA sensitivity. Intolerance to these drugs can manifest variably from mild, acute bronchoconstriction to poor, chronic asthma control to life-threatening anaphylactoid reactions. It is best to avoid the use of these drugs in patients with asthma, or to involve an asthma or allergy specialist if use of such medication is clinically necessary to treat other conditions.

Answered by:
Dr. Paul Hernandez

12. Role of drug modifiers in rheumatic disease

What is the role of drug modifiers in inflammatory rheumatic disease?

Submitted by:
Alfred Ernst, MD
Rosetown, Saskatchewan

Most patients with inflammatory rheumatic diseases—rheumatoid arthritis, psoriatic arthritis and ankylosing spondylitis—will receive disease modifying anti-rheumatic drugs (DMARDS) at some time in the course of their disease and usually for prolonged periods. DMARDS can be divided into two formulations: traditional (*e.g.*, methotrexate, leflunomide, sulfasalazine or hydroxychloroquine) and biological (*e.g.*, etanercept, infliximab or adalimumab).

The goal of DMARD or remittive therapy is to reduce inflammation, control symptoms and improve function and quality of life. However, the ultimate goal is to slow and hopefully arrest disease progression, thereby reducing joint deformity and preventing radiographic damage. With the availability of new, more targeted treatments for inflammatory arthritis, there is growing evidence that earlier treatment leads to significantly better outcomes and that disease remission is achievable. Therefore, early referral for suspected inflammatory arthritis should be encouraged to assist the primary care physician in the diagnosis and early management of these conditions.

Answered by:
Dr. Michael Starr

13. Management of hypocondriasis

What is the best approach to deal with a hypocondriac patient that we can't reassure?

Submitted by:
Samen Hasswani, MD
Montreal, Quebec

The management of hypochondriasis has typically been in the domain of the primary care physician. At least initially, these patients strongly resist psychiatric referral. Their mental model of the problem is that they have a covert physical illness and so they do not believe they need to see a psychiatrist. With that frame of mind, they do not respond favourably to counseling or traditional psychotherapy. One pragmatic approach that has heuristic merit is the use of paradoxical intention, telling the patient that you recognize the suffering but that you are not certain that you can help very much. When little is expected, sometimes small gains are appreciated and there is some improvement.

Hypochondriasis patients usually accept referral for treatment of a comorbid psychiatric condition, such as obsessive-compulsive disorder, panic disorder, or a depressive disorder. When these conditions are treated pharmacologically and with the appropriate support and acknowledgement of the patient's suffering, the hypochondriasis often will improve.

Although there are no controlled clinical trials on which to base rational treatment for hypochondriasis, an open trial of high dosage of fluoxetine on hypochondriasis patients not meeting criteria for co-morbid depressive disorders showed much promise, with 10 of 16 patients showing much improvement at the end of a 12-week trial. Several other trials with selective serotonin reuptake inhibitors with primary hypochondriasis have also shown some positive results.

Answered by:
Dr. Hany Bissada

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14. Left bundle branch block

What is the significance of a left bundle branch block in an asymptomatic patient?

Submitted by:
Samen Hasswani, MD
Montreal, Quebec

This electrocardiographic finding is not to be overlooked and is usually associated with underlying heart disease. It is associated with significantly reduced long-term survival, with 10-year survival rates as low as 50%, probably reflecting the severity of the underlying heart disease. I would proceed with a complete history and physical examination in search of underlying heart disease, particularly related to the presence of obstructive coronary disease and/or systolic dysfunction. Pharmacologic stress testing with dobutamine or adenosine is usually warranted and may be more specific than exercise nuclear imaging in diagnosing left anterior descending artery coronary stenosis.

Answered by:
Dr. Igal A. Sebag

15. Cardiovascular recommendations

A 63-year-old male has no lifestyle risk factors for coronary heart disease other than age and a low HDL-C of 0.83 mmol/L; lipids are otherwise normal; total cholesterol is 4.77 mmol/L and LDL-C is 3.6 mmol/L. What would you recommend?

Submitted by:
Herb Domke, MD
Victoria, British Columbia

In addressing the issue of LDL-cholesterol and total cholesterol/HDL-C ratios, the risk category for this patient[1] is at least moderate (*i.e.*, 10-year risk estimate of cardiovascular death is > 10% and < 20%). In this case, diet and therapeutic lifestyle changes are recommended. If target levels are not achieved within three months (*i.e.*, LDL-C < 3.5 mmol/L and total cholesterol/HDL-C < 5 mmol/L), then statin therapy should be initiated.

The second and perhaps more important issue to address in this case is the low HDL-C (< 1.0 mmol/L). One should consider the possibility of the metabolic syndrome—abdominal obesity, triglyceride (TG) levels, BP and fasting glucose must be determined. In this case, increased aerobic exercise, increased intake of monounsaturated fats, moderate alcohol intake (only if the TG level is within normal limits) and weight loss are beneficial. A fibrate should also be considered.

Suggested Reading
1. Genest J, Frohlich J, Fodor G, et al: The Working Group on Hypercholesterolemia and Other Dyslipidemias: Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease, 2003 update. CMAJ 2003; 168(9):921-4.

Answered by:
Dr. Igal A. Sebag

16. Confirming a drug allergy

What is the safest and most accurate way of confirming or refuting a supposed drug allergy in which there is no documented allergic reaction and the patient has no memory of the reaction they had?

Submitted by:
Declan Boylan, MD
Sudbury, Ontario

This is obviously a difficult thing to do, given that establishing a diagnosis of a drug allergy in most cases is solely dependant upon the history. The ability to establish the mechanism of a reaction (*i.e.*, Type 1 immunoglobin E, vs. Type 3 antibody-antigen complex formation, vs. Type 4 delayed type hypersensitivity) depends almost completely on detailing the nature and time course of the reaction. Given that a drug allergy bears little relationship to the patient's overall atopic status, one cannot ascribe risk based on other allergic features in the patient or family (except perhaps other drug allergies).

If the drug is a ß-lactam antibiotic, then intradermal testing for the major and minor determinants may be carried out. If negative, then a drug challenge may be considered, or simply start the necessary course of treatment under close surveillance for delayed eruptions. For most other drugs, the relevant haptens and immunogenic metabolites are either unknown, or too vast in number to identify reliably with *in vivo* testing at this time. *In vitro* techniques (*i.e.*, lymphocyte transformation testing) exist, but are not currently available for general clinical use.

If the drug is deemed necessary (*i.e.*, there are no alternatives available), then a careful test dosing procedure (graded challenge) may be carried out, preferably in a hospital setting. For example, 1/100th, then 1/10th of the dose is given at 30 minute intervals, followed by the remainder of the dose with continued observation. This differs from a desensitization protocol (in a patient with a *known* drug allergy), where smaller increments are given over a longer period of time (four hours to six hours) until the full dose is reached. Following test dosing, the patient still requires close surveillance during the treatment course to observe for the development of a delayed response (*i.e.*, rash, mucosal lesions).

Answered by:
Dr. Tom Gerstner

17. Sunburn and melanoma

We know that intermittent sunburns predispose to later melanoma development, but is this risk restricted to the previously burned skin only, or does the increased risk apply equally to all skin surfaces?

Submitted by:
Wayne Parsons, MD
Stratford, Ontario

While a causal relationship has not been confirmed, it is generally well accepted that ultraviolet sunlight exposure is associated with melanoma. As noted, melanomas are particularly associated with intermittent, intense exposure and sunburns.[1] An increased risk may be observed with this pattern of exposure at any age although the acquired risk is believed to be higher if the exposure is in childhood or adolescence.

While it is reasonable to presume that the risk is likely to be greatest in previously burned skin, the biologic mechanism for increased melanoma risk with intermittent sunburns is not well elucidated; thus, it is unknown whether the risk is restricted to previously burned skin or applies equally to all surfaces. However, in practical terms, high risk individuals should be familiar with skin self-examination and undergo an annual total body skin examination by a physician. Dx

References
1. Elwood JM, Jopson J: Melanoma and sun exposure: An overview of published studies. Int J Cancer 1997; 73(2):198-203.

Answered by:
Dr. Sharlene Gill