



1. How to follow-up with patients taking amiodarone

? Amiodarone seems to be prescribed more often for cardiac arrhythmias. Could you please provide a practical method for following patients on amiodarone (i.e., which tests should be done and how often should they be done)?

Submitted by:
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This month's topics:

1. How to follow-up with patients taking amiodarone
2. The scoop on bacterial vaginosis
3. Allergies to OTC vitamins
4. Can ACE inhibitors prevent diabetes progression?
5. What's the treatment for LBD?

Amiodarone often causes adverse effects, which can increase in frequency and severity during long-term administration. The following questions illustrate a simplified approach to elicit adverse effects caused by amiodarone. Please note, this is not a complete list of all amiodarone's adverse effects. Careful surveillance of all amiodarone-treated patients is essential.

1. *"Have you noticed any change in your handwriting?"*
Amiodarone frequently causes subtle neurologic symptoms, often manifested as a deterioration in handwriting quality, tremor, and difficulty with balance.
2. *"Have you noticed change in your quality of sleep?"*
Patients on amiodarone occasionally report vivid dreams, difficulty falling asleep, insomnia, or nightmares.
3. *"Has there been a change in your appetite or bowel habits?"*
Amiodarone may cause lack of appetite, nausea, and, occasionally, constipation.
4. *"Have you noticed any change in your vision?"*
Amiodarone causes corneal microdeposits, but only occasionally causes visual disturbance. This is most often experienced as visual haloes, especially at nighttime (around street lights or car lights). A few patients also have dry, itchy, and "gritty" eyes.
5. *"Have you noticed any sensitivity to sunlight?"*
Patients on amiodarone frequently report easy sunburning, sometimes with redness or "prickly" feeling in the skin. They should all be instructed to use sunscreens and to wear protective clothing.
6. *"Have you noticed any other symptoms? Do you think you are having any side-effects?"*
Some possible adverse effects include pulmonary toxicity, thyroid toxicity, liver toxicity, bradycardia, and drug interactions. Thyroid and liver functions should be measured every six months.

Answered by:
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2. The scoop on bacterial vaginosis

? Is bacterial vaginosis a sexually transmitted disease?

Submitted by
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The long and the short of it is that we don't really know. Bacterial vaginosis (BV) is caused by an overgrowth of *Gardnerella* in the vagina resulting in malodorous discharge. Diagnosis is made by Nugent criteria of an alkalotic vaginal pH (> 5.0), a positive whiff test, the evidence of clue cells in a saline wet mount, and an absence of irritation of the vaginal or vulvar epithelium. BV is found in 10% to 25% of patients in general obstetrics and gynecology clinics and in up to 64% of patients visiting sexually transmitted disease clinics.¹ Sexually transmitted infections (STIs) are increased in patients with BV and the latter is increased in patients with some risk factors for STIs.² Evidence remains controversial with respect to treating partners of those testing positive for BV, but some physicians feel this is helpful in cases of recurrent BV. Of significance is the fact that BV is seen in patients who are not currently sexually active, or who have never been sexually active. *Gardnerella* is also found in people who are asymptomatic. Hence, BV should probably be considered an infectious process that is transmitted through various means, including sexual contact, that becomes problematic under favourable conditions

References

1. Hacker NF, Moore JG: *Essentials of Obstetrics and Gynecology*, Third Edition. WB Saunders Company, Philadelphia, 1998.
2. Morris MC, Rogers PA, Kinghorn GR: Is bacterial vaginosis a sexually transmitted disease? *Sex Transm Infect* 2001; 77(1):63-8.

Answered by:

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3. Allergies to OTC vitamins

? Are there any known gastrointestinal allergies to over-the-counter multivitamins?

Submitted by:
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A search of the PubMed database did not reveal any case reports of allergic reactions to over-the-counter vitamins resulting in gastrointestinal manifestations. However, many vitamins are now formulated with other naturopathic supplements which may be highly allergenic in sensitized individuals. These include echinacea, royal jelly, bee pollen, and St. John's Wort. Niacin in high doses (50 mg to 100 mg) often causes transient cutaneous flushing. Less commonly, niacin may also cause abdominal pain or diarrhea.

Answered by:
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4. Can ACE inhibitors prevent diabetes progression?

? If a patient has impaired glucose tolerance with no documented hypertension, would there be a role to start an ACE inhibitor to prevent progression to overt diabetes?

Submitted by:
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As the progression from impaired glucose tolerance to overt Type 2 diabetes is between 5% and 10% annually, there is understandably a lot of impetus for preventing/delaying the onset of Type 2 diabetes. Studies using angiotensin-converting enzyme (ACE) inhibitors, such as captopril (CAPPP) and ramipril (HOPE), have demonstrated a 21% to 33% relative risk reduction in onset of Type 2 diabetes. Similarly, in the LIFE study, there was a 25% relative risk reduction in patients randomized to the angiotensin II receptor blocker (ARB), losartan. The mechanism of this effect is hypothesized to be due to either an improvement in glucose disposal secondary to increased skeletal muscle blood flow and/or an improvement in insulin sensitivity. These trials, however, were not designed to address this specific question, and diabetes prevention was neither the primary nor, in some cases, the secondary outcome of these studies. Several other studies, designed specifically to examine diabetes prevention with lifestyle modification (DPP, DPS), metformin (DPS), troglitazone (TRIPOD), acarbose (STOP-NIDDM), orlistat (XENDOS), *etc.*, have also demonstrated a 25% to 58% reduction in onset of diabetes. Two large prospective studies of diabetes prevention using the combination of ramipril/rosiglitazone and valsartan/nateglinide are currently underway. Until more evidence is available and questions regarding optimal dosage, duration of therapy, *etc.*, are answered, I would not recommend pharmacotherapy for diabetes prevention. However, I would emphasize the importance of increased physical activity and dietary modification aiming at 5% to 7% weight loss, as these have consistently been shown to reduce the risk of developing Type 2 diabetes.

Answered by:
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5. What's the treatment for LBD?

? Can Lewy body dementia co-exist with Parkinson's disease dementia, and what is the current suggested treatment for Lewy body dementia?

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Parkinson's disease with dementia is diagnosed when an individual is diagnosed with Parkinson's disease, and at least 12 months later begins to reveal dementiform changes. The one-year period of preserved cognition is critical in the current diagnosis formulation.

In Lewy body dementia (LBD), cognitive impairment, involving memory and attention, is often the earliest observed symptom. Parkinsonian findings (usually no resting tremor), visual hallucinations, and sensitivity to neuroleptics frequently accompany the presentation. The best treatment for LBD is the cholinesterase inhibitor, rivastigmine. Its effectiveness was clearly shown in the classic study published in the *International Journal of Geriatric Psychiatry*.¹

L-dopa compounds usually do not help significantly and can actually heighten the delirious features of this disorder. Much smaller studies have demonstrated the efficacy of the other two cholinesterase inhibitors.

Some experts believe the distinction between Parkinson's and LBD is quite artificial, and that the diagnostic criteria requires adjustment. [Dx](#)

Reference

1. Del Ser T, McKeith I, Anand R, et al: Dementis with lewy bodies: Findings from an international multicentre study. *Int J Geriatr Psychiatry* 2000; 15(11):1034-1045.

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