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Case Studies in Type 2 Diabetes

- The legacy effect of early and intensive achievement of glycemic targets has a long term legacy effect in reducing both micro and macrovascular complications.
- Treatment must be started early in the natural history of TD2M, if progressive beta cell failure is to be prevented.
- Combination therapy will be required in virtually all diabetic patients at some stage of their disease. This reflects the multiple pathophysiologic defects present in the development of hyperglycemia. Medications with complementary but different mechanisms of action should be chosen based on an individualized rather than an algorithmic basis.
- Hypoglycemia is both the most common and most serious side effect of diabetes therapy, and may occur in over 20% of sulfonylurea treated patients. Because of the increased risk of hypoglycemia in the elderly, therapies that minimize this side effect should be considered.
- According to the American Diabetes Association in 2010, the general A1C goal for nonpregnant adults is <7%. However, epidemiologic studies have suggested a small, but incremental benefit in microvascular outcomes to lowering A1C from 7% into the normal range. Accordingly, the A1C goal for selected individual patients should be lower than the general goal of <7%, if this can be achieved without significant hypoglycemia. Patients for which this goal might be appropriate include those with a short duration of diabetes, long life expectancy, and no significant CVD.
- For those with a history of severe hypoglycemia, shortened life expectancy, advanced microvascular or macrovascular complications, extensive comorbid conditions or long-standing diabetes in whom the general goal is difficult to attain, less stringent A1C goals than the general goal (<7%) should be considered. Specific goals for people with these conditions should be individualized.
- Injectable therapies for diabetes are often underutilized in management of hyperglycemia. Insulin is a very effective and usually necessary therapy for patients with A-1c greater than 9% and should be considered much earlier in the treatment course for patients with poorly controlled diabetes.
- Glp-1 analogues and Dpp-4 inhibitors provide a unique mechanistic approach to reducing blood glucose without attendant hypoglycemia. The use of Glp-1 injectable therapy may be useful in certain patients before turning to or as an adjunct to insulin therapy.
Atrial Fibrillation 2012: New Developments in Risk Stratification and Antithrombotics to Prevent Stroke

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- Persistence with oral agents is poor
- Stroke risk reduction with warfarin is substantial (±66%)
- Risk of ICH with warfarin is low (<1%/year)
- The CHADS2 scoring system remains the preferred risk stratification method for patients with non-valvular atrial fibrillation. The new ATP9 guideline suggests any CHADS2 score of 1 or greater deserves antithrombotic and not aspirin therapy. As a reminder:
  - C – Congestive Heart Failure – 1 point
  - H – Hypertension – 1 point
  - A – Age >= 75 – 1 point
  - D – Diabetes – 1 point
  - S – History of Stroke or TIA – 2 points
- While novel anticoagulants offer some important advantages over warfarin (all reduce risk of stroke or systemic embolism and lessen the risk of intracerebral bleed), whether the newer agents should replace warfarin in patients with stable INRs that are usually in therapeutic range (TTR above 70%) continues to be debated
- Important differences among the newer agents exist, and they have not been compared in a head-to-head trial, making any comparison about the “best” newer agent more difficult
- AT9 Guidelines offer new Rx directions, but these directions are not agreed upon by all guideline committees
- This changing field will be watched closely over the next year or two as new approved antithrombotic agents and updated guidelines will continue to change the landscape of those who treat patients with non-valvular atrial fibrillation
Evolving Issues and Therapies in Inflammatory Bowel Disease: A Primary Care Update

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**Pearls for Managing Active Therapy of IBD**

- Understanding the diagnosis is important for effective management.
  - A significant number of patients with presumed active IBD have no evidence of inflammation upon investigation
  - Active inflammation responds to immunosuppressive medications, IBS does not
- Encourage adherence to effective therapies for IBD patients.
  - Once daily dosing with mesalamine
  - Patient education regarding benefits, risks of untreated disease
- Monitor response to medications with a measurement tool such as the Harvey Bradshaw Index
- Remission should be achieved successfully before transitioning to maintenance therapy
- Steroids: effective for short-term but use should be minimized by steroid-sparing agents
- Beware of disease mimickers and look for infections (especially when not appropriately responding to therapy)
  - C. difficile infection more common in IBD, requires vancomycin therapy
- 5-ASA therapy should be dosed and delivered to the area of disease
- Anti-TNF therapy is beneficial for UC and CD (esp. luminal and fistulizing disease)
  - Combination therapy appears to have synergy when the patient is naïve to both immunomodulator and anti-TNF therapy

**Important Preventative Strategies**

- Appropriate vaccinations
  - Avoid live attenuated vaccines for patients with prednisone, immunomodulators or anti-TNF therapy
- Calcium/vitamin D supplementation (especially when on prednisone), screen for vitamin D deficiency and osteopenia/porosis
- Colonoscopic surveillance at 8 years of disease- then every 1-3 years
- Annual Pap smear for females affected by IBD
Who has Pulmonary Arterial Hypertension and How to Best Help Them?

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- Be mindful of patient with shortness of breath where no ischemic cardiac or pulmonary etiology is found: Get an echocardiogram.
- When the echocardiogram claims patient to have Pulmonary Hypertension, multiple questions need to be asked:
  - What kind of Pulmonary Hypertension (PH)?
  - Is it left sided heart disease manifesting itself in elevated pulmonary pressure?
  - Which PH group does the patient belong to?
  - Is the elevated pressure truly elevated?
- All PH patients need a right heart catheterization to accurately assess pressures and evaluate the patient from a hemodynamic perspective and to help assign PH group
- PH: Increased mean Pulmonary Artery Pressure mPAP ≥ 25
- PAH (Pulmonary Arterial Hypertension): mPAP ≥ 25 and Normal Pulmonary Capillary Wedge Pressure PCWP ≤ 15
- There are five groups of PH:
  - I: PAH where the problem is in the pulmonary vasculature,
  - II: Cardiac dysfunction related,
  - III: Lung Disease Related
  - IV CTEPH (related to pulmonary Embolism)
  - V: Miscellaneous
- Therapy best started at PAH Centers
- No Trials of Calcium Channel Blockers without evidence of reversibility on right heart catheterization
- Patients need close follow-up, especially with advanced/accelerated disease
- Therapy should be escalated aggressively. No one should die with monotherapy or without trial of prostanoid therapy
Emerging Trends in Osteoporosis

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• All women need a DXA test by age 65, higher-risk postmenopausal women should be tested earlier
  o Risk factors that would prompt testing sooner rather than later include a family history of osteoporosis, personal history of prior fracture as an adult, cigarette smoking, low body weight, diseases (e.g., rheumatoid arthritis, etc.), and drugs (e.g., glucocorticoids, etc.)
• Adequate calcium (1200 mg/d, diet + supplement if needed) and vitamin D are important, but avoid excess
• Weight-bearing activities should be encouraged (e.g., walking 30-40 min per session, 3-4 sessions/wk)
• Pharmacologic treatment is recommended for women at high risk of fracture
  o A clinical dx (hip or spine fracture) or DXA diagnosis of osteoporosis (T-score -2.5 or below) or at high risk using FRAX (high risk is 3% or more 10-year risk of hip fracture or 20% or more 10-year risk of major osteoporosis-related fractures)
• Four drugs have evidence for “broad-spectrum” antifracture efficacy (alendronate, risedronate, zoledronic acid and denosumab)
  o Despite news reports of potential safety issues with long-term bisphosphonate treatment, risk of ONJ or atypical femur fracture is low; benefit/risk ratio is favorable for most patients at high risk of fractures
• Persistence with oral agents is poor
  o Patients need to understand why drugs are prescribed and have feedback on effectiveness (monitoring with DXA); non-oral therapies (e.g., zoledronic acid and denosumab) may help keep patients on therapy
ADHD in Adults: Making the Diagnosis and Optimizing Treatment

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- 4-5% of adults in the US have ADHD. The high prevalence of ADHD in adults but it is often undiagnosed.
- The impact of undiagnosed ADHD is serious. ADHD leads to: higher health risks and increase healthcare costs, higher divorce rates, lower levels of socioeconomic attainment and academic achievement, more motor vehicle accidents, and risk for co-morbid psychiatric disorders such as anxiety, depression, and substance use and abuse disorders.
- ADHD is a highly heritable disorder and it’s most likely genetic determinants are related to neurological factors such as production and regulation of neurotransmitters (i.e., dopamine, norepinephrine).
- Practitioners should ask about cardinal symptoms including problems with sustained attention, control of motor activity, and difficulty inhibiting behavior and emotions to an extent that impairs major life activities. You can use rating scales and interview data in your workup. The NACE Adult ADHD Toolkit includes samples of such rating scales and interviews (see [http://www.naceonline.com/AdultADHDtoolkit/QI1.php](http://www.naceonline.com/AdultADHDtoolkit/QI1.php) to download these scales) to help you evaluate ADHD symptoms and symptoms of other co-morbid psychiatric disorders.
- Stimulant medication has been used for over five decades to treat ADHD. FDA approved stimulants for adults with ADHD include: OROS methylphenidate (OROS-MPH), Dex-methylphenidate extended release (D-MPH XR), mixed amphetamine salts extended release (MAS XR), and lisdexamfetamine dimesylate (LDX). Atomoxetine is a non-stimulant approved for adults with ADHD.
- Monitor side effects closely, titrate upwards until symptom remission is achieved and/or side effects are causing concern. On follow-up visits check vital signs, symptom scale reports, side-effects, adherence, and plan to adjust medication dosages or switch or add agents as indicated. Be concerned about stimulant medication abuse, misuse (overuse or underuse), diversion, or problematic interactions.
with other agents.

- Adults with ADHD may have other psychiatric co-morbidities.
  
  o For ADHD adults with severe anxiety a non-stimulant such as atomoxetine may be considered before a stimulant to avoid exacerbation of anxiety.

  o For adults with ADHD who have substance abuse disorders, atomoxetine is generally preferred. When stimulants are used, abuse is less common with extended release stimulants than immediate release stimulants.

  o When active depression is present, the depression should be treated before the ADHD and when depression is improved then ADHD medication may be added.

  o Patients with bipolar disorder and ADHD the first target is mood stability. ADHD medications can be trialed if impairing symptoms persist despite mood stabilizing therapy.

  o Evaluate blood pressure/pulse prior to initiating ADHD treatment. Address hypertension before treating ADHD. Stimulants have a clinically insignificant effect on blood pressure in treated normotensive adults.

- Psychosocial interventions are very important in treating adults with ADHD. These include ADHD education, cognitive behavior therapy, educational accommodations, employment accommodations, treatments for relationship problems (couples therapy or family therapy), and financial management.

- Resources for ADHD can be found at www.chadd.org and by downloading the NACE Adult ADHD Toolkit.
Alpha-1 Antitrypsin Deficiency (AAT)
And Why it Should Matter to Primary Care Clinicians

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- 1-3% of all patients with COPD have Alpha-1 Antitrypsin Deficiency
- The AAT molecule is a serine protease inhibitor predominantly produced in the liver by hepatocytes, and is responsible for inhibiting the proteolytic enzyme neutrophil elastase. Neutrophil elastase destroys elastin which maintains bronchial and alveolar wall integrity. Thus AAT protects pulmonary tissue from aggressive proteolytic enzymes, and helps in regulating pulmonary immune processes.
- Alpha-1 antitrypsin deficiency predisposes patients to COPD, especially when they smoke.
- It is impossible to distinguish the majority of AATD patients from average COPD patients, therefore all COPD patients need to be screened once.
- Since more that 70% of COPD patients are cared for by primary care clinicians, screening for AATD needs to occur in both primary care and pulmonary settings
- Screening can be in form of AAT levels alone, or better yet with phenotyping or genotyping so that counseling can be given to carriers.
- Many resources including the Alpha-1 Foundation and Bio-pharma are making finger-stick blot tests available for patients at no cost. The test is quick (with explanation and processing 7 min) and can be performed in physician offices.
- Genetic Information Non-Discrimination Act of 2008 (GINA) protects the patient in terms of employment, health insurance and genetic discrimination.
- Treatment is available in terms of augmentation therapy to restore the deficient patient.
- Treatment is pooled human AAT, given once a week IV.
- In order to screen patients, it is best to make screening part of the routine workup of COPD patients either at the intake, via EMR, or via PFT/Spirometry reflexive testing.