I. Introduction
   a. Our patients often have multiple medical problems, some of which have a significant impact on eye health, others which have virtually no impact on the eyes but can profoundly affect daily activities and quality of life.
   b. This course will cover common and important mental health conditions, what interactions (if any) they have on the visual system and ocular health, their current management strategies, in an attempt to give the optometric practitioner a view of the patient ‘beyond the eyeballs.’

II. Anxiety
   a. Generalized anxiety disorder (GAD) is a common psychiatric condition, probably second only to depression in prevalence. Described as 6 months of symptom duration (prominent worrying and impairment) plus 3 or more of the following features occurring on most days: fatigue, restlessness, poor concentration, irritability, muscle tension, and unsatisfying sleep (but not anhedonia, which is seen in depression). GAD is characterized by unrealistic or excessive worry and is disproportionate to the severity of the ongoing life events. It is often described as uncontrollable and is diffused over many life events and activities (thus “generalized” as opposed to more focused worry with other anxiety disorders). Physical (somatic) symptoms include tachycardia, dyspnea, urinary frequency, tremor, or excessive sweating. In contrast to GAD, mild anxiety sharpens the senses and probably improves performance.
   b. Other anxiety disorders are panic disorder, obsessive compulsive disorder (OCD), social anxiety disorder, and PTSD. Generalized anxiety disorder is more common with the aging process (increasing exponentially with age) and more common in women (about twice as frequent as in men). GAD has frequent co-morbidities and major depression may co-exist in up to 2/3’s of patients.
   c. Symptoms are physical (tachycardia, chest pain), cognitive (confusion, poor concentration, difficulty making decisions), behavioral (restlessness, agitation), affective (crying, hostility), and uncontrollable worry (apprehension, dread). Symptoms are long-lasting, with a much longer duration than a panic attack.
   d. Benzodiazepines (BZD’s) are the corner stone of therapy for anxiety. They act quickly, are dependable, and have a long history of trusted performance. They act by binding to the GABA receptor, thus potentiating its affinity for GABA, which is the predominant
inhibitory neurotransmitter of the brain. Leading BZDs are alprazolam/Xanax, clonazepam/Klonopin, diazepam/Valium, and lorazepam/Avitan.

e. Physical dependence and withdrawal symptoms can occur with long-term BZD therapy but these drugs provide rapid relief. Other BZD side effects include sedation, incoordination, confusion, and anterograde amnesia.

f. Buspirone (BuSpar) is a non-BZD anxiety drug, taking about 2 weeks to have its effects felt, but has no sedating or muscle relaxant effects. It cannot be used on an as-needed basis.

g. Antidepressants (not all) are as effective as BZDs for anxiety and lack problems with dependence/abuse and adverse effects on memory and coordination.

h. SSRI antidepressants (specifically paroxetine/Paxil and escitalopram/Lexapro) are now considered to be as effective as BZD’s, but take longer to have their benefits noted; they are safer since there is no interaction with alcohol and no dependence. Venlafaxine/Effexor is a SNRI and is as effective as the SSRIs listed above. Tricyclics are as beneficial but anticholinergic effects are often intolerable.

i. References


III. Post-traumatic stress disorder (PTSD)

a. PTSD demographics: following sexual assault, witnessing mass casualties in war or in natural disasters, physical assault (mugging, etc.), motor vehicle accident, disease
epidemics. PTSD diagnostic criteria were established in 1980 in the Diagnostic and Statistical Manual of Mental Disorders (DSM III), resulting from increased awareness due to the Viet Nam conflict. PTSD following armed combat has been known by a variety of names, including “soldier’s heart” (US Civil War), “shell shock” (WW1), “battle fatigue” (WW2), and “Viet Nam stress syndrome.”

b. Exposed to a traumatic event
   i. Witnessed (not a direct participant) or involved with events or actual or threatened death or serious injury to self and/or others
   ii. Response is of intense fear, helplessness, horror

c. PTSD characterized by three “symptom clusters”
   i. Re-experiencing
   ii. Avoidance/numbing
   iii. Hyperarousal

d. Cluster 1 = re-experiencing, characterized by:
   i. Recurrent recollections (thoughts, images) of the event
   ii. Event is repeated (reliving it, flashbacks, “dissociative flashbacks” occurring upon awakening)
   iii. Recurrent distressing dreams (nightmares)
   iv. Psychological distress or “psychological reactivity” at events/entities that symbolize or resemble the trauma

e. Cluster 2 = avoidance/numbing, characterized by:
   i. Avoiding thoughts, feelings, activities, places, people relating to or connected with the traumatic event(s)
   ii. Diminished interest/participation, reduced range of affect, detached or estranged from family and society

f. Cluster 3 = hyperarousal, characterized by:
   i. Persistent symptoms of increased arousal
   ii. Difficulty falling asleep or staying asleep
   iii. Difficulty concentrating
   iv. Hypervigilance, exaggerated startle response (“watching the perimeter”)

g. Duration of above symptoms is 1 month or longer

h. Other aspects of possible PTSD
   i. Experienced or witnessed sexual or physical abuse
   ii. Experienced or witnessed emotional abuse or neglect
   iii. Experienced threat of violence in the home or community
   iv. Personally experienced sexual abuse associated with PTSD symptoms in 42-90% of kids; diagnosis of PTSD in 24-35% of kids having witnessed domestic violence

i. Symptoms cause marked distress and poor functioning in social and occupational situations. PTSD is a chronic disease, with frequent psychiatric and medical co-morbidities, marked functional impairment and economic costs. Family members of
PTSD patients can develop “secondary trauma” in which the maladaptive behavior (most typically hyperarousal, also anger, irritability) mimics that of the PTSD patient.

j. PTSD is not a simple diagnosis to make, since the great majority of patients with lifetime PTSD have co-morbidities (typically major depressive episodes, alcohol dependence, or drug dependence). Up to 88% of men and 79% of women with PTSD have co-morbidities.

k. The criteria for PTSD diagnosis are complex. (N Engl J Med 2002 reference below has the most detailed criteria listing). A short questionnaire is able to suggest PTSD. Questions relate to an individual’s reactions to a stressful event, those reactions occurring 2 or more times in the past week:
   i. Upsetting thoughts or memories about the event that enter your mind against your will
   ii. Upsetting dreams about the event
   iii. Acting or feeling if the event is happening again
   iv. Feeling upset by reminders of the event
   v. Bodily reactions (racing heart beat, upset stomach, sweating, dizziness) when reminded of the event
   vi. Difficulty falling or staying asleep
   vii. Irritability or outbursts of anger
   viii. Difficulty concentrating
   ix. Heightened awareness of danger to yourself or others
   x. Being jumpy or being startled at something unexpected

l. First line treatment = selective serotonin reuptake inhibitor ant-depressants (SSRI)
   i. Able to ameliorate the 3 core symptom complexes
   ii. Able to ameliorate depression, also other anxiety disorders that may accompany PTSD, such as panic, social phobia, or obsessive compulsive disorder
   iii. Primary drugs are sertraline/Zoloft, paroxetine/Paxil

m. Other antidepressants: venlafaxine/Effexor (serotonin norepinephrine reuptake inhibitor) is second line; third line drugs are tricyclics (amitriptyline, imipramine), and some MAO inhibitors (but little data on these groups and their side effect profiles are more severe than with the SSRIs).

n. Benzodiazepines: good for anxiety and sleep issues, but unable to ameliorate the 3 core symptom complexes; significant concerns for dependence/abuse with comorbid substance abuse, so their use is typically avoided.

o. Atypical antipsychotics – for comorbid psychoses (delusions, hallucinations) accompanying PTSD; not first- or second-line drugs but used PRN (Seroquel, Risperdal, etc.)

p. Anticonvulsants – help with re-experiencing symptoms, but uncertain benefits for numbing or hyperarousal symptoms.
q. Alpha-1 antagonist (prazosin) – benefit for relief of nightmares. Proposed increased CNS adrenergic activity at night, with overstimulation of alpha-1 receptors in the hippocampus and amygdale.

r. Beta-blockers – specifically propranolol. Proposed mechanism of consolidation of short-term, labile memories into long-term memories occurs in the amygdale; propranolol is proposed to interfere with neurotransmitters participating in memory consolidation.

s. PTSD is an extremely expensive disease, to the patient affected, to his/her family members, co-workers, and general societal costs: higher risk of suicide, depression, and substance abuse; higher risk of domestic violence, children/grandchildren witnessing this, costly treatment programs (individual/group therapy, cognitive behavioral therapy), disability compensation, court time, jail time, lost wages.

t. References


IV. Depression

a. Very common, second only to hypertension as the most commonly seen condition in primary care practice.

b. Lifetime prevalence of depression is 17%; 1 in 10 patients has a major depressive episode. Prevalence of depression is as high as 25% in nursing homes. Frequencies for men and women differ: 12% in men, 20% in women. The risk of suicide is 30-fold higher in clinically depressed individuals than in the community at large.

c. Major depressive episode (aka major depressive disorder, MDD) is depressed mood or anhedonia for 2+ weeks, plus 3+ of the following: insomnia, feeling worthless or guilty, fatigue, diminished concentration, changes in appetite, psychomotor retardation, or recurrent suicidal thoughts. The episode is characterized by a change in affect, behavior, and quality of life. The change is both recent and major, with a shift of perceptible difference in status and affect. Single depressive episodes raise risks of subsequent episodes: there is a 38% chance of a second episode in 1 yr, with a 62% chance of a second episode in 5 years.

d. Generally not felt to be related to a prior, predisposing stressful event (per DSM-4, but more “liberal”/looser criteria per DSM-5). Somatic symptoms (anorexia, weight loss, constipation, poor sleep, lost libido, vague aches, or poor concentration) may be part of depression but can obscure the classic “low mood.” This can be a missed diagnosis.

e. Association with chronic health issues: chronic medical illness, chronic pain, and the “3 C’s” which are cardiovascular disease, CNS disease (CVA, Parkinsons), and cancer.

f. Major depressive episodes need to be contrasted with dysthymia and bipolar disorder
   i. Dysthymia is depression for > 2 years; symptoms are less severe than in MDD
   ii. Bipolar disorder is diagnosed by a single manic or hypomanic episode. Bipolar type I is alternation between fully manic and depressive episodes; bipolar type II is alternation between “hypomanic” and depressive episodes.

g. 50-70% of patients respond to initial therapy (either meds and/or psychotherapy, typically cognitive behavior therapy or CBT). Therapy for MDD needs to be typically continued for months into the “maintenance period” as symptoms of the initial episode improve. Therapy of MDD very often consists of trial-and-error with a variety of drugs to find the right fit, buttressed by careful listening and analysis of symptoms and patient progress, with modifications in the medication regimen.

h. Length of therapy
   i. On therapy until there is a positive response (less anhedonia, etc.)
   ii. Therapy must continue while in remission, often up to 9 months after achieving remission (highest risk of relapse is in first 3-6 months after recovery)
   iii. For a patient with yearly depressive episodes or a high suicide risk, maintenance therapy lasts for 12-36 months.
   iv. For a patient with more frequent episodes or very severe depression, lifetime.

i. Serotonin- and/or nor-epinephrine reuptake inhibitors (SSRI, SNRI)
   i. Tertiary amine tricyclics (Elavil, Sinequan, Tofranil) – old drugs, “tricyclics”
ii. Secondary amine tricyclics (Norpramin, Pamelor) – again, old drugs

iii. Bicyclic = combined serotonin and norepinephrine reuptake inhibitors (venlafaxine/Effexor and duloxetine/Cymbalta) – newer drugs without as many adverse effects

iv. Older tricyclic drugs were called “dirty drugs” due to their profound side effects including sedation, orthostatic hypotension, and cardiac effects with ventricular arrhythmias and conduction defects; also known for anticholinergic problems of dry mouth, constipation, and urinary retention.

j. SSRIs are fluoxetine/Prozac, paroxetine/Paxil, sertraline/Zoloft, citalopram/Celexa, escitalopram/Lexapro, fluvoxamine/Luvox.
   i. Minimal/variable sedation and few anticholinergic effects, also minimal/no cardiac effects
   ii. However, notorious for causing sexual dysfunction (delayed ejaculation and anorgasmia, incidence up to 60% of users), and GI upset (nausea, vomiting, diarrhea); may cause anxiety and insomnia at the initiation of therapy.
   iii. Much preferred to older tricyclic antidepressants due to side effect profiles.

k. Mirtazapine/Remeron has a complex and unduplicated mechanism: effects are to enhance both norepinephrine and serotonin effects; no anticholinergic toxicities, less sexual dysfunction, but likely to cause somnolence and weight gain.

l. Norepinephrine- and dopamine-reuptake inhibitor is bupropion/Wellbutrin; considered to be the least likely to cause adverse drug events (less nausea, somnolence, or sexual dysfunction vs. the SSRIs).

m. Clinical trials have indicated that some drugs work better for certain types of depression if there are comorbid features or other characteristics. Although the SSRIs revolutionized the treatment of depression in the ‘90’s, the older SSRI drugs are more often used for anxiety, and the newer drugs are increasingly popular for depression.

n. There is significant controversy about treatment of depression, complicated by (a) proponents of drug therapy vs. “talk therapy;” (b) variable and contradictory results of drug trials; (c) appropriateness of drug therapy for mild depression (is it really depression or just ‘sadness’?); (d) lack of congruence between drug trials and “real life” management strategies. The STAR*D Study attempted to provide practical guidelines for treatment, noting 40% or less success with the initial drug choice (see references).

o. References


V. Schizophrenia

a. Affects about 1% of the population, but the greatest risk factor is a positive family history, being 6.5% in first-degree relatives of patients. Typically presents in late adolescence or early adulthood (70% of incident cases occur between 15-35 years of age). Men may have an earlier age of onset, often have a more serious form of the disease, with more negative symptoms (see below) and worse outcome. It is also more frequent in individuals born in cities, the larger the city and the long the duration of residence, the greater the risk. Overall, the heritable component accounts for about 70% of the risk for schizophrenia, and 30% of the risk results from perinatal ( prematurity) and childhood brain injury, plus psychosocial stress over life events. Life expectancy is 20% shorter than usual, with the most common cause of death being suicide (10% of patients), this risk of suicide being 20-fold higher than in the general
population. 60% of patients with a schizophrenia diagnosis are on disability within the first year.

b. Named as such by the observation of the “disconnection or splitting of psychic functions” but does not mean a “split personality.” A more apt description would be “shattered,” analogous to a waking nightmare – in which the often terrifying bizarre images and impossible occurrences of a typical nightmare (which would otherwise dissipate upon awakening) won’t go away.

c. Prodrome (build-up to the overt disease):
   i. Gradual social withdrawal
   ii. Loss of interest in school and work
   iii. Odd behavior or odd speech
   iv. Personality change with a flat affect
   v. Change in appearance and/or hygiene
   vi. Deterioration in social or work functioning and abilities

d. Positive symptoms (overt psychotic features): these are characterized by a lack of insight and failure to appreciate that these symptoms are not real or caused by the illness.
   i. Hallucinations – disturbed perceptions without stimuli; auditory hallucinations (“hearing voices”) are most common – speaking about or to the patient; hallucinations are also less specific auditory (machinery or repetitious noises), or can involve other senses (touch, taste, smell, vision).
   ii. Delusions – these are falsely held beliefs that are not shared by others in the community or family; persecution (victims of some threat), passivity (their thoughts or actions are controlled by an external force), and delusions along themes (grandiosity, sexual, or religious foci).
   iii. Thought disorders – distorted, disorganized, or illogical speech; “knight’s move” thinking characterized by a chess-like, sudden right-angle veering off the topic.

e. Negative symptoms, which include withdrawal, self-neglect, loss of motivation and initiative, emotional blunting (flat affect), social withdrawal, poor mental creativity, anhedonia, and paucity of speech. Schizophrenic patients have trouble with memory, paying attention, and executive function. Many individuals have a history of behavioral dysfunction (primarily social difficulties and learning difficulties). Negative features wreak havoc with family, friends, and co-workers.

f. Most common positive symptoms are lack of insight (97%), auditory hallucinations (74%), ideas of reference (70%), delusions of reference (67%), suspiciousness or flatness of affect (both 66%), delusional mood or delusions of persecution (both 64%), thought alienation (52%), and thoughts spoken aloud (50%).

g. Many schizophrenics are heavy smokers (nicotine helps their poor attention), and some abuse stimulants (for the same reasons.)

h. An acute psychotic episode results from a combination of multiple factors (increased dopamine, neuronal inhibitor transmitters, loss of neurons, etc.) so that the patient is
 hypersensitive to stimuli and unable to regular his/her responses through normal inhibitory mechanisms – the patient cannot sort through what is known and what is unknown, the world becoming overwhelming and threatening or controlling them.

i. Specific diagnostic criteria for schizophrenia (considered a diagnosis of exclusion)
   i. Deterioration of social and work function
   ii. Persistence of illness for 6 months (including the prodrome)
   iii. Active phase of the disease (at its worst) is 4 weeks’ duration, unless the patient is treated earlier
      1. 1 or both of either auditory hallucinations or bizarre delusions
      2. OR 2 of the following: hallucinations, nonbizarre delusions, disorganized speech and behavior, negative symptoms

j. Treatments started with the advent of “neuroleptic” drugs from the 1950’s, the first being chlorpromazine (Thorazine), found to have benefit in schizophrenic patients being tested for the drug’s use as a new antihistamine. The “classic antipsychotic” drugs (see below) decrease dopamine-mediated neurotransmission, which diminishes the distractibility and improves perceptual abilities of schizophrenic patients. Dopamine is not the sole factor: inhibitory interneurons are decreased in number, the enzymes that synthesize the inhibitory neurotransmitter GABA are less active, plus brain matter itself is less developed in several regions, including hippocampus and superior temporal cortex.

k. “Typical” antipsychotic drugs date from the 1950’s; “atypical” antipsychotic drugs date from 1991, beginning with clozapine. Atypical drugs have less extrapyramidal side effects (movement disorders), but cause more weight gain. Globally, antipsychotic drugs act as dopamine antagonists: “typical” drugs have D2 receptor antagonism, while “atypical” drugs have D2, plus D1, D4, and serotonin antagonism. In carefully structured, nose-to-nose trials, neither group is superior to the other for control of psychosis, but atypical drugs are better tolerated (due to less movement disorders, mainly tardive dyskinesia) and may better relieve “negative” symptoms. However, clozapine is the most effective last-ditch drug when others have failed.

l. Typical antipsychotic drugs are chlorpromazine (Thorazine, 1953), perphenazine (Trialphon, 1957), trifluoperazine (Stelazine, 1958), fluphenazine (Prolixin, 1959), thioridazine (Mellaril, 1961), haloperidol (Haldol, 1966), thiothixine (Navane, 1968), and loxapine (Loxitane, 1978).

m. Atypical antipsychotic drugs are clozapine (Clozaril, 1991), risperidone (Risperdal, 1993), olanzapine (Zyprexa, 1996), quetiapine (Seroquel, 1998), ziprasidone (Geodon, 2001), aripiprazole (Abilify, 2002), olanzapine/fluoxetine (Symbbyx, 2003), iloperidone (Fanapt, 2009), arsenapine (Saphris, 2009), paliperidone (Invega, 2009), and lurasidone (Latuda, 2010).

n. Typical antipsychotic drugs are famous for causing ocular side effects:
   i. Chlorpromazine/Thorazine (anterior subcapsular cataracts)
   ii. Thioridazine/Mellaril (pigmentary retinopathy)
iii. Fluphenazine/Prolixin (premature presbyopia)

iv. Of significant import is increased weight gain with higher risk of diabetes, worst with clozapine and olanzapine, least with aripiprazole and lurasidone, and middling with risperidone, quetiapine, iloperidone, paliperidone

vi. References


VI. Bipolar Disorder

a. Previously known as “manic-depressive disorder” which inaccurately implied a regular cycling between a manic episode and a depressive episode (additionally inaccurately implying equal durations of these episodes). Actually, depressive episodes last longer and may occur more repeatedly before there is a diagnostic manic episode. Presently, bipolar disorder is defined as “bipolar I” and “bipolar II” disorders. The prevalence is
approximately 1% each for bipolar I and bipolar II, with an additional 3% for the “not otherwise specified.”

b. Bipolar I has at least one episode of mania, while bipolar II has at least one episode of hypomania. The major difference between them is the greater severity of the elevated mood and the associated functional disability (which is characterized by psychosis, the need for urgent care or hospitalization, or marked impairment) seen with bipolar I. Both types have manic features and the symptoms are similar, but the dividing point is the degree of disability associated with the mania in type I.

c. Mania or hypomania require an elevated (euphoric) and/or irritable mood, plus at least 3 of the following symptoms (or 4 of the following if the mood is only irritable):
  i. Grandiosity or inflated self-esteem
  ii. Decreased need for sleep
  iii. Increased talking or pressured speech
  iv. Racing thoughts
  v. Distractibility
  vi. Overactivity (an increase in goal-directed activity)
  vii. Psychomotor agitation
  viii. Excessive involvement in pleasurable activities with a high potential for painful consequences

d. Bipolar I is characterized by a behavioral disturbance “sufficiently severe to cause marked impairment in social or occupational functioning or to require hospitalization, or is characterized by the presence of psychotic features.” Bipolar II has a hypomanic episode (in contrast to the manic episode) that is not severe enough to cause marked impairment in social or occupational functioning; there are no psychotic features.

e. The diagnosis of bipolar disorder is typically based on mania or hypomania, although the depressive phase confers the greatest burden and impact on quality of life. Similarly to schizophrenia, the age of onset is in adolescence and early adulthood. Many bipolar patients will experience multiple depressive episodes before the hallmark manic episode, leading to an incomplete diagnosis (and risk of inappropriate treatment).

f. Periods of depression vs. mania vs. normalcy
  i. Bipolar I: depressed 32%, manic 15%, asymptomatic 50%
  ii. Bipolar II: depressed 50%, hypomanic 4%, asymptomatic 45%

g. The cornerstone of pharmacologic management of bipolar disorders is mood stabilizers. The use of antidepressants is considerably less established at this time. There are a number of FDA-approved drugs for either acute mania or acute mania and maintenance therapy
  i. Mood stabilizers: lithium (oldest), then carbamazepine (Tegretol), then divalproex sodium (Depakote), and lastly lamotrigene (Lamictal).
  ii. Atypical antipsychotic drugs: Abilify (mania and maintenance); Saphris (for mania); Zyprexa (for mania and maintenance); Seroquel (mania or maintenance); Geodon (mania or maintenance).
iii. Antiepileptic drugs: Tegretol (mania); Depakote (mania); Lamictal (maintenance).

iv. Lithium (mania).

v. Medications for bipolar depression: Seroquel or Symbyax. Note the absence of SSRIs and virtually all other conventional antidepressants, which are not FDA approved for bipolar depression. SSRIs used alone in bipolar illness (unsupported by a mood stabilizer) have a higher risk of cycling back to mania or hypomania.

h. References


VII. Borderline Personality Disorder
   a. Epidemiology
      i. Diagnosed in early adulthood, but there may be features notable in childhood
      ii. Associated with a HIGH risk of suicide – 10% of patients will attempt suicide, which is a 50-fold higher prevalence than in the general population
      iii. Most characteristic features are high risk of suicide plus the overwhelming fear of rejection and abandonment
      iv. Like schizophrenia, both genetic factors plus childhood factors have been identified in the etiology. Childhood factors include history of emotional, physical, or sexual abuse (up to 70% of cases), plus insecure attachment and family marital problems.
      v. In clinic settings, about 75% of BPD diagnoses are in women.

b. Diagnostic features – DSM IV has identified a total of nine features, and the diagnosis of BPD requires five or more of them. These features can be in a laundry list but authors tend to clump them in sectors. The two most prominent factors suggesting BPD are extreme fear of abandonment plus suicidal gestures. Therapists present the following list to a possible BPD case and the patient frequently self-identifies with these features.
   i. “interpersonal hypersensitivity”
      1. Frantic efforts to avoid real or imagined abandonment (but do not include suicidal or self-mutilating behavior); profound fear of abandonment, desperate efforts to avoid being alone
      2. Pattern of unstable and intense interpersonal relationships – tumultuous, frequent arguments, repeated breakups
   ii. “affective dysregulation” or “affective criteria”
      1. Affective instability because of marked reactivity of mood (intense episodic dysphoria, irritability, anxiety usually lasting a few hours and only rarely more than a day or two)
      2. Inappropriate, intense anger or difficulty controlling anger (frequent displays of temper, constant anger, recurrent physical fights)
      3. Chronic feelings of emptiness
   iii. “impulsivity” (also called “behavioral criteria”)
      1. recurrent suicidal behavior, gestures, threats, or self-mutilating behavior
      2. Impulsive behavior in at least 2 areas that are potentially self-damaging (spending money, sex, substance abuse, reckless driving, binge eating, persistent gambling)
   iv. Other factors (also called “cognitive criteria”)


1. Identity disturbance with markedly and persistently unstable self-image or sense of self (depersonalization)
2. Transient, stress-related paranoid ideation or severe dissociative symptoms

c. Co-morbidities – there are lifetime prevalences of other mental health disorders for patients with borderline personality disorder, which include:
   i. Eating disorder – up to 53%
   ii. Panic disorder – up to 48%
   iii. Social phobia – up to 47%
   iv. Obsessive compulsive disorder – up to 25%
   v. PTSD – up to 56%
   vi. History of major depressive disorder (MDD) – up to 83%
   vii. Substance abuse – up to 66%
   viii. Dysthymia – up to 39%

d. Treatment strategies
   i. Good news, bad news
      1. High remission rates: 45% by 2 years after diagnosis, 85% by 10 years after diagnosis, plus a low remission rate (just 15%).
      2. Very high and frequent use of mental health services, poor function (only 25% are able to be employed full time), and 40% on disability after 10 years.
   ii. Psychopharmacology – previously hampered by too-small studies, poorly controlled, and still without large studies that are definitive. There are some trends with different groups of medications:
      1. Atypical antipsychotics may help with cognitive symptoms and anger, but frequently have challenges with weight gain (DM2), and some BPD patients have issues with binge eating
      2. Anticonvulsants – possibly most useful, helpful with disinhibition, negative affectivity (poor self-image, guilt, contempt), overall function, and “trait antagonism” (hostility, outbursts, aggression). Drugs most cited are topiramate, valproic acid, and lamotrigine.
      3. Omega-3 fatty acids – cited several times as helpful with antagonism and negative affectivity (mechanism is not determined).
      4. SSRIs – may help with reducing anger, but little benefit on impulsivity
   iii. Psychotherapy – the core treatment for BPD. There are multiple specialty types suggested, but the two following have the most data and most success.
      1. Dialectical behavior: combines cognitive behavioral therapy with Eastern philosophy; teaches skills in the domains of mindfulness, distress tolerance, regulation of emotions, and interpersonal effectiveness. This technique (typically once/week with one-on-one
therapist, plus weekly group therapy) has demonstrated an 80% reduction in suicide risk, anger issues, and improved social function.

2. Mentalization-based therapy: improves patient’s ability to “mentalize,” and to understand his own and others’ mental states. Again, this is both one-on-one (18 months), plus group therapy.

e. References


VIII. Ocular issues

a. Suicide attempts that did not succeed – this opens up a vast number of possibilities, including monocular status, TBI, ocular and facial damage, partially sighted or legally blind, field loss, etc.

b. Substance abuse: includes drugs (talc retinopathy, retinal neovascularization), alcohol consumption (possibility of optic nervehead pallor), and heavy tobacco use (with heightened risks for both ARMD and cataract).

c. Medications
i. Benzodiazepines – not much here (clonazepam causes blurred vision at 2% incidence)

ii. Anticonvulsants – variable degrees of problems with diplopia and blur (lamotrigine has reports of 28% incidence of diplopia)

iii. Typical antipsychotics – thioridazine (Mellaril) famous for retinal pigment dispersion; chlorpromazine (Thorazine) famous for anterior subcapsular pigmentary depositions; fluphenazine (Prolixin) able to cause premature presbyopia; variable degrees of anticholinergic effects with the phenothiazines (dry eye, accommodative dysfunction).

iv. Benztropine mesylate (Cogentin) – utilized to reduce drug-induced Parkinsonism from antipsychotic medications; may result in variable but significant mydriasis.

v. Atypical antipsychotics – variable degree of metabolic disorders with a higher risk of type 2 diabetes mellitus.

vi. Tricyclic antidepressants – anticholinergic effects, dry eye.

vii. SSRIs – not much here (fluoxetine causes “abnormal vision” at 2% incidence)

viii. Newer antidepressants – again, not much here (venlafaxine causes “abnormal vision” at 5% incidence)

ix. When more than one drug is in use (example: PTSD), the mix of drugs makes the process of teasing out causative agents of ocular side effects difficult. More vision problems are likely with more medications in tandem, but this is often more vague focus problems, and generalized but mild light sensitivity. (My personal experience: light sensitivity with quetiapine/Seroquel.)

x. Interesting note: there is on-going research using older psychedelic drugs in mental health conditions. Two examples: psilocybin for anxiety related to cancer and also for depression, and LSD for anxiety associated with life-threatening diseases. MDMA (3,4-methylenedioxymethamphetamine, Ecstasy) has been studied for PTSD. (See: Kupferschmidt K. High hopes, in Science 2014; 345:18-23, and Can ecstasy treat the agony of PTSD?, in Science 2014; 345:22-23.) Will there be ocular issues related to psychedelic drugs?

d. Solar retinopathy resulting from sun-gazing (this is not unique to schizophrenia, but has been observed as part of religious rituals, eclipse viewing, etc.)

i. Irradiation energy is initially absorbed by melanosomes in the RPE, then theoretically spread thermally to the outer segments of the photoreceptors. The injury combines thermal injury with photochemical damage. The RPE can regenerate, but the photoreceptors cannot.

ii. There is direct damage to photoreceptor lamellae and pyknosis (condensation) of photoreceptor nuclei.

iii. This is visible as a sharply demarcated, reddish, foveal spot, appearing about 2 weeks after the direct exposure. It is best imaged with SD-OCT, appearing as a very localized hyporeflective space in the outer retina with some thinning of neurosensory retina overlying the area
iv. References


IX. Conclusions – Some of these commonly encountered conditions have little impact on eye health or the visual system, and the drugs used for their treatment are claimed to have similarly little ocular impact. Ocular side effects are generally more common with older medications (particularly the typical antipsychotic medications), although light sensitivity has been attributed to a number of newer drugs. Additionally, we need to recall that many of the above conditions are rarely managed with monotherapy alone. Mental illness can be a subject which we avoid or stigmatize, to the detriment of those individuals living with it. Greater awareness and understanding of the challenging nature of these conditions is warranted, as is increased advocacy for its management.