

Exclusive Coverage of the

NEUROSCIENCE EDUCATION INSTITUTE (NEI) 2017 CONGRESS

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Clinical Considerations for Altering Treatment Patterns in Patients With Major Depressive Disorder

Of every 3 patients with major depressive disorder (MDD), only 1 patient will achieve remission on their first antidepressant, and 67% of patients require 4 antidepressant trials before symptoms remit, said Thomas L. Schwartz, MD, professor and vice chair, Department of Psychiatry, SUNY Upstate Medical University.

In a session at the 2017 NEI Congress in Colorado Springs, Colorado, Schwartz gave an informational session on strategies for switching, combining, or augmenting treatments for patients with MDD.

First, Schwartz explained what to look for when choosing the initial antidepressant. It is important to look at the comparable efficiency among, and within, classes of medications, and the initial selection of an antidepressant medication is based largely

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Genetic Determinants of Psychiatric Disorders and Utility of Genetic Testing

There is no known gene for any major psychiatric disorder, nor is one ever likely to be found, explained Stephen M. Stahl, MD, PhD, adjunct professor of psychiatry, University of California, San Diego, during an overview of the age of personalized medicine and the role of pharmacogenetics.

"The classical theory is that genes cause mental illness, but that theory does not work for psychiatry," he said. Genes do not code for psychiatric disorders or psychiatric symptoms, but what they do code for are proteins and epigenetic regulators, many of which regulate the efficiency of information processing in brain circuits, which can be visualized with neuroimaging techniques, explained Stahl, who is also chairman of the NEI. Currently, psychiatry research is attempting to link circuits upstream to treatment response and downstream to regulatory genes.

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Clinical Considerations (Continued from page 1)

on: anticipated adverse effects, safety/tolerability for individual patients, patient preference, medication response in prior episodes, and cost.

Schwartz outlined factors to consider when choosing between switching and augmenting treatment. A physician should consider switching to a different antidepressant when:

- It's the first antidepressant trial.
- There are poorly tolerated adverse effects to the initial antidepressant.
- There is no response (less than 35% improvement) to the initial antidepressant.
- More time is available to wait for a response (less severe, less functional impairment).
- The patient prefers to switch to another antidepressant.

Schwartz shared several techniques for switching treatments:

- Direct switch: Stop the first antidepressant abruptly and start the new antidepressant the next day.
- Taper and switch immediately: Gradually taper the first antidepressant and start the new antidepressant immediately after discontinuation.
- Taper and switch after a washout: Gradually withdraw the first antidepressant and start with the new antidepressant after a washout period.
- Cross-taper: Taper the first antidepressant (usually after 1-2 weeks or longer) and build up the dose of the new antidepressant simultaneously.

There is no evidence to support preference for one agent or class over another, and switching within the same class and switching to another class are both options, said Schwartz. The most commonly used treatments when switching are other selective serotonin reuptake inhibitors (SSRIs)/serotonin and norepinephrine

“ The initial selection of an antidepressant medication is largely based on anticipated adverse effects, safety/tolerability for individual patients, patient preference, medication response in prior episodes, and cost. ”

reuptake inhibitors (SNRIs), bupropion, and mirtazapine. Underused treatments include tricyclic antidepressants and monoamine oxidase inhibitors.

Withdrawing SSRIs and SNRIs typically cause flu-like symptoms such as nausea, lethargy, and irritability, said Schwartz. “Withdrawal symptoms can generally begin within hours to days of dose reduction,” he said. Venlafaxine is associated with the most severe withdrawal effects; meanwhile, fluoxetine rarely causes withdrawal symptoms because of the long half-life of the parent drug and its active metabolite. Withdrawal effects can be more common or more severe with paroxetine than with some other SSRIs.

Closing the session, Schwartz explained that a physician should consider an adjunctive medication when:

- There have been 2 or more antidepressant trials.
- The initial antidepressant is well tolerated.
- There is partial response (greater than 25% improvement) to the initial antidepressant.
- There are specific residual symptoms or adverse effects to the initial antidepressant that can be targeted.
- There is less time to wait for a response.
- The patient prefers to add another medication. ●

Genetic Determinants (Continued from page 1)

The way an individual inherits a psychiatric disorder or responsiveness to one treatment over another is determined by a genotype having a subtle molecular abnormality, which causes abnormal information processing, leading to behavior with complex functional interactions and emergent phenomena, said Stahl. Psychiatric disorders, although descriptive and reliable, are not predictive of treatment response or linked to neurology and therefore are not diseases.

“Each psychiatric disorder is likely to represent many diseases, perhaps hundreds,” said Stahl. The current diagnostic strategy is to attempt to link symptom domains that cut across psychiatric disorders to inefficient information processing in specific brain circuits.

There are 4 genotypes that affect drug metabolism:

- Poor metabolizers or inhibitors of P450 may have increased drug serum levels and adverse events.
- Intermediate metabolizers or inhibitors of P450 may have increased drug serum and adverse events.
- Extensive metabolizers metabolize substrates normally.
- Ultra-rapid metabolizers or inducers of P450 may have reduced drug serum levels and poor efficacy.

Stahl finished up the session by discussing where genetic testing fits in modern psychiatric practice. According to Stahl, pharmacogenomics can add to the modern psychiatric practice because the associated experts are:

- Genetically informed.
- Neurobiologically empowered.

- Data oriented.
- Equipoise oriented, weighing all the evidence, including genetic test results.
- Aware of red herrings in any of the data collected, including genomics.
- Aware of a strategy for situations in which there is no evidence from large randomized controlled trials or all these approaches have failed.

For treatment-resistant patients, Stahl recommended that providers:

- Exhaust evidence-based solutions.
- Think.
- Take another history, including asking a new informant. For example, consult the patient’s spouse about the patient’s history.
- Reconsider the diagnosis. The treatment-resistant patient may be bipolar, have dementia, etc.
- Collect new data, including genomics.
- Use this new information to rebalance the evidence and come up with a genetically informed, neurobiologically empowered novel and rational treatment or combination.

“Genetic testing as a clinical tool is still in its infancy,” concluded Stahl. “Genotyping may be especially useful for patients who do not respond to or tolerate a drug as expected. Caution is essential when bringing genetic testing into the selection of treatment in clinical practice.” ●

Symptoms of MDD May Indicate Another Psychiatric Disorder

A session at the 2017 NEI Congress presented by Stephen M. Stahl, MD, PhD, adjunct professor of psychiatry, University of California, San Diego, emphasized the importance of making an accurate diagnosis in patients presenting symptoms of depression.

Several psychiatric disorders are commonly misdiagnosed as major depressive disorder (MDD) but may be bipolar disorder (BD), pseudobulbar affect disorder (PBA), and post traumatic depression (PTD), said Stahl, the NEI chairman.

Stahl began the session by explaining that individuals with unipolar depression and “a bit of mania,” also known as depression with mixed features (DMX), are more likely to have an eventual diagnosis conversion to BD. Indications of this can be seen from many clues across the spectrum including family history of BD, suicidal ideation, severe depression, antidepressant remission, and impulse control, said Stahl.

“Several psychiatric disorders are commonly misdiagnosed as MDD but may be bipolar disorder, pseudobulbar affect disorder, and post traumatic depression.”

“The prognosis for depression with co-occurring (hypo)mania is much worse than for pure unipolar depression or bipolar depression without mixed features,” said Stahl.

Symptoms most commonly seen in DMX include irritability, distractibility, psychomotor agitation, racing/crowded thoughts, increased talkativeness, emotional lability, and rumination.

Next, Stahl discussed PBA disorder, which occurs in the context of brain injury, including traumatic brain injury (TBI), stroke,

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How Patient Suicide Affects Clinicians

According to data from the CDC, there were 41,149 suicides in the United States in 2013. Suicide is the 10th leading cause of death in the United States, and for every associated death, there are approximately 25 suicide attempts. In practice, 5000 to 10,000 clinicians lose a patient to suicide every year, and 40% to 60% of psychiatric residents will experience a patient suicide during their first year of training.

A panel of doctors at the 2017 Neuroscience Education Institute Congress in Colorado Springs, Colorado, discussed the impact of losing a patient to suicide and how they deal with it.

The moderator, David W. Goodman, MD, FAPA, assistant professor, Department of Psychiatry and Behavioral Sciences, Johns Hopkins School of Medicine, and director and founder, Adult Attention Deficit Disorder Center of Maryland, kicked off the discussion by asking the panelists about the impact patient suicide has on them.

Roger S. McIntyre, MD, FRCPC, professor, Departments of Psychiatry and Pharmacology, University of Toronto, and head, Mood Disorders, Psychopharmacology Unit, University Health Network, told the audience an anecdote from 3 months ago when one of his residents lost a patient to suicide. The resident was severely affected by this, said McIntyre.

“Most of it was about an automatic assumption process,” said McIntyre. “The automatic assumption process was that because this person killed themselves, de facto that must mean that something was done wrong, and I think that’s an automatic assumption many people have.”

McIntyre noted that there still remains a lot of confusion

about what is a risk factor for suicide and predicting suicide. They are not the same thing, said McIntyre. This was another point of education for the resident, because the guilt is “Could I have predicted suicide?” Psychiatrists can’t predict this type of behavior, said McIntyre.

Following the suicide of one of his long-time patients, Thomas L. Schwartz, MD, professor and vice chair, Department of Psychiatry, SUNY Upstate Medical University, felt self-doubt. Feeling alone and wondering whether he would get sued and whether he was a bad doctor, he found that what helped was speaking to other colleagues who had dealt with patient suicide.

In most other fields of medicine, physicians accept the fact that there’s such a thing as terminal illness; there’s no feeling that you messed up or did something wrong, said William M. Suavé, MD, Greenbrook TMS NeuroHealth. However, that’s not the case for psychiatrists.

Goodman agreed with McIntyre that one of the biggest impacts is the feeling of guilt when a patient commits suicide.

“It becomes a thread in the fabric of who you are,” said Goodman. “And for me, it was ‘Was I at fault?’ The first thing I went to was it must have been my fault; I missed something. There was a red flag; I didn’t get it.”

Goodman then moved into how he was able to power through the guilt. What helped him was an editorial in the local newspaper written by the mother of the patient, saying that her son got the best care available and died from his illness. Goodman now uses this as his perspective: It was not his fault that his patient died. The patient died because of his illness. ●

Symptoms of MDD (Continued from page 3)

Alzheimer disease, amyotrophic lateral sclerosis, and multiple sclerosis. PBA is characterized by uncontrollable, inappropriate laughing or crying. For psychiatrists, the most inclusive term for the disorder is “involuntary emotional expression disorder.”

Symptoms of the disorder include pathological crying and laughing, irritability, aggression, and unpredictable and rapidly changing emotions. Treatment for the disorder includes selective serotonin reuptake inhibitors and tricyclic antidepressants. Both therapies can help reduce the frequency and severity of PBA episodes and are typically prescribed at lower doses than for depression.

“PBA is often under recognized, misdiagnosed, and under-treated,” said Stahl. “Only 40% of patients who discuss PBA symptoms with their clinician [receive a diagnosis].” The reason PBA is so frequently misdiagnosed is that it is often mistaken for depression, explained Stahl. However, there are several

distinctions—the duration of a PBA episode is shorter, crying is not congruent with subjective mood in PBA, and other symptoms of depression are not associated with PBA.

For patients with TBI, depression is the most common psychiatric complication, said Stahl. More than 50% of patients with moderate to severe TBI will experience a depressive episode in the first-year post injury, and they have a risk of suicide that is 5 times that of the general population.

PTD is an adjustment-based depression that may occur rather than biologically based depression after TBI. The difference between MDD and PTD is that PTD is characterized by more irritability, anger, and aggression versus sadness or fearfulness. “Symptoms that are seemingly indicative of MDD may actually be manifestations of a different psychiatric illness,” concluded Stahl. “Making an accurate differential diagnosis in patients presenting with symptoms of depression is critical to the implementation of optimal patient care.” ●

Pharmacogenomic Testing Tool Can Reduce Utilization of Benzodiazepines

An economic analysis presented at the 2017 NEI Congress showed that pharmacogenomic testing in patients with specific psychiatric disorders can reduce the utilization of benzodiazepines.

The prospective study was conducted in individuals with either generalized anxiety disorder (GAD, n = 318) or major depressive disorder (MDD, n = 459). Eligible patients included those who had augmented or changed their antidepressant or antipsychotic medications within the previous 90 days. Combinatorial pharmacogenomic testing was offered following a medication switch or augmentation; the authors classified the medications into 1 of 3 categories following the test results:

- Use as directed
- Moderate gene–drug interactions
- Significant gene–drug interactions

Pharmacy claims data were assessed for a year post testing, and cost savings were calculated per member per year (PMPY), based on whether the test was used in decision making. For 660 patients who had been prescribed at least 1 benzodiazepine, the cost of utilization was analyzed for 6 months pretest and 6 months post test.

The analysis found that in both the GAD and MDD groups, PMPY cost savings were significant if the healthcare provider's decisions were congruent with the results of the pharmacogenomic testing. The authors found that congruent prescribing yielded \$6747 in PMPY medication savings in the GAD group compared with incongruent prescribing; PMPY savings in the MDD cohort were \$3738 with congruent prescribing.

When the authors drilled further into the savings observed in the claims data in the congruent group, they found the highest savings were with medications used to treat neurological and psychiatric disorders (GAD, \$2700; MDD, \$1332), followed by antineoplastic drugs (GAD, \$2696; MDD, \$1038). The lowest savings were observed with dermatology medications for the GAD group (\$1) and vitamins for the MDD group (\$5).

Physician prescribing of benzodiazepine changed significantly following pharmacogenomic testing, both with respect to drug counts and refills, the study found.

The authors credited the pharmacogenomic test, GeneSight, as a useful treatment decision support tool in GAD and MDD. Using the tool resulted in cost savings when providers made congruent decisions with the pharmacogenomic results. Authors added that reduction in benzodiazepine prescriptions in the congruent cohort could reduce the need for benzodiazepines. ●

Emotional Dysregulation and Executive Dysfunction in Patients With ADHD and Bipolar Disorder

A session on distinguishing between bipolar disorder (BD) and attention-deficit/hyperactivity disorder (ADHD) was presented by David W. Goodman, MD, FAPA, assistant professor, Department of Psychiatry and Behavioral Sciences, Johns Hopkins School of Medicine, and director and founder, Adult Attention Deficit Disorder Center of Maryland, at the 2017 Neuroscience Education Institute Congress in Colorado Springs, Colorado.

First, Goodman gave statistics on the prevalence of ADHD and BD in both children and patients. In the United States, the prevalence rate of ADHD in children is 8% and the prevalence in adults is 4.4%. The rate of BD in US children is 0.5% and in US adults, 2.5%. The comorbidity of ADHD and mood disorders in children from the Multimodal Treatment Study of Children With ADHD study is about 4%, but when looking at adults, the comorbidity is about 40%, and 19% have comorbid BD, said Goodman.

Symptoms of BD include hypomanic or manic symptoms of increased talkativeness, racing thoughts, distractibility, psychomotor agitation, and increased risky behavior. For ADHD, the symptoms include talking too much in social situations, having difficulty with maintaining attention, being fidgety and restless, and being impulsive.

“Executive function is a categorical, cognitive, higher-cortical thinking ability, and it encompasses several factors,” said Goodman.

The factors of executive function are:

- Response inhibition: impulse control
- Working memory: holding information while making decisions
- Set shifting: moving from task to task
- Interference control: resisting distractions

There is a symptomatic overlap of emotional dysregulation and executive dysfunction in ADHD and BD, said Goodman.

“Where emotional dysregulation used to be part of the bipolar mood disorder arena, we now understand it to be part of ADHD as well,” said Goodman. “And the cognitive problems and the executive function, which were subsumed under ADHD, are now understood to be part of BD, major depression, and a variety of other psychiatric disorders.”

Regarding emotional dysfunction, compared with controls, patients with BD had a poorer performance on immediate verbal memory tasks, said Goodman. Both clinical groups exhibited significantly lower scores than controls on the recognition phase of verbal and nonverbal memory tasks and tasks of executive function with high working memory demand.

Emotional dysregulation, when associated with ADHD, involves 2 primary deficits:

- An inhibitory deficit: socially inappropriate behavioral responses to strong emotion
- A self-regulatory deficit: an inability to self-soothe psychologic arousal that strong emotion induces, refocus

attention, or organize the self for coordinated action toward an external goal

Emotional dysregulation in BD has been extensively studied and is marked by:

- Emotional hyperresponsiveness
- Poor recognition and acceptance of emotions
- Difficulties in adapting behaviors to experienced emotions

During manic or mixed episodes, patients with BD show a high degree of emotional hyperresponsiveness. During depressive phases, 2 types of patients are identified: emotional hyporesponsiveness patients and emotional hyperresponsiveness patients, who are more at risk of experiencing mixed episodes.

Emotional regulation causes several social impairments for patients, including a low threshold for emotional excitability/impairment, behavioral dyscontrol in the face of strong emotions, and an inflexibility/slow return to baseline. ●

The Effects of Misdiagnosing Depression With Mixed Features as Unipolar Depression

During a session at the 2017 Neuroscience Education Institute Congress in Colorado Springs, Colorado, Roger S. McIntyre, MD, FRCP, professor, Departments of Psychiatry and Pharmacology, University of Toronto, and head, Mood Disorders Psychopharmacology Unit, University Health Network, discussed the contributing factors and effects of misdiagnosing or inappropriately treating patients with antidepressants.

“We know that major depressive disorder and bipolar disorders are common conditions,” said McIntyre. “And we know still, in 2017, that the majority of people who have major depressive disorder or bipolar disorder either are not diagnosed accurately and/or are not diagnosed in a timely fashion.”

McIntyre first explained that many patients with unipolar depression who show a “little bit of mania” are more likely to have an eventual diagnostic conversion to bipolar disorder.

He then discussed how the *Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5)*, the standard classification of mental disorders used by mental health professionals in the United States, is a contributing factor to the difficulty of diagnosing depression with mixed features (DMX).

“Although irritability, distractibility, and psychomotor agitation are among the most common symptoms of DMX, they are excluded from *DSM-5* mixed features criteria due to the overlap of these symptoms with other disorders and between mania and depression,” said McIntyre.

When utilizing research-based diagnostic criteria, 4 times as many cases were identified. Using *DSM-5* criteria, all patients

identified as DMX will have DMX; however, only 5.1% of individuals who have DMX will be identified and about 95% are at risk of receiving inappropriate treatment. Following criteria by psychiatrist Franco Benazzi, MD, PhD, approximately 10% of patients who are identified as having DMX will not actually have DMX, but less than 50% are at risk of receiving inappropriate treatment.

There is a question of which will be more detrimental, McIntyre said: misdiagnosing someone who is “pure unipolar” as DMX or treating unidentified DMX with antidepressants?

The consequences of misdiagnosis/inappropriate treatment include years (often a decade or more) of unnecessary suffering, treatment resistance, reduced likelihood of responding to eventual appropriate mood stabilizer treatment, treatment-emergent activation syndrome, and suicidal ideation.

There are several tools that can be used to assess DMX, said McIntyre.

- Bipolar Depression Rating Scale: a clinician-administered assessment of current symptoms
- Mini International Neuropsychiatric Interview: patient self-report assessing current symptoms
- Clinically Useful Depression Outcome Scale With *DSM-5* Mixed: patient self-report assessing current (hypo) manic symptoms.
- Hypomania Checklist (HCL-32): patient self-report that screens for lifetime (hypo)manic symptoms

“The inappropriate overprescribing of antidepressants may contribute to drug-induced (hypo)manic episodes, treatment resistance, suicidality, and overall poor quality of life for many patients suffering from depression,” concluded McIntyre. “You

will not know if a depressed person has (hypo)manic symptoms or a positive family history of bipolar disorder unless you ask every patient, every time.” ●

Combining Psychotherapy and Medication to Improve Outcomes in Patients With Axis I Disorders

According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)*, the Axis I classification represents acute symptoms that need treatment. These disorders include major depressive disorder (MDD), schizophrenia, and anxiety disorders. During a session, Ira D. Glick, MD, professor emeritus, Department of Psychiatry and Behavioral Sciences, Stanford School of Medicine, emphasized the importance of combining psychological and psychopharmacologic strategies for patients with Axis I disorders and provided guidelines for administering the combination.

Glick first outlined the rationale for combining medication and therapy:

- Patients value psychotherapy.
- Patients may not be on medication.
- Etiology: Although biological, “stress” may precipitate episodes.
- Pathogenesis: Illness has effects on the family.
- Treatment: To help improve adherence.
- It may work better than 1 modality (medication or therapy).

Psychotherapy is important because some conditions have no effective pharmacotherapy treatment, medication can be contraindicated, and the patient may not want to take medication. Most important, most patients have social and interpersonal problems accompanying Axis I disorders as either the source or the consequence of the illness, said Glick.

However, there are several reasons why combination therapy is not delivered, said Glick. Many providers deliver one or the other, most insurers only pay for nonintegrated treatment, many professionals are trained in one or the other, and provider bias.

Psychotherapy has been shown to improve outcomes over medication alone in bipolar disorder, childhood attention-deficit/hyperactivity disorder, MDD, schizophrenia, posttraumatic stress disorder, sleep disorders, and bulimia nervosa, said Glick.

He then provided the audience with guidelines for administering combined therapy for their patients:

1. Diagnosis:

- Make a DSM-5 diagnosis.
- Make a family systems diagnosis.
- Make an individual dynamic diagnosis.
- Formulate a case.

2. Goals

- Select appropriate modalities and their combination.
- Develop specific goals for each modality.
- Decide about sequencing.
- Be aware of, and enquire about, adverse effects of each modality as well as their interactive effects.

3. Sequencing of combined treatments

- Establish an alliance.
- For psychotic disorders, start medication early.
- Add individual intervention as the patient is able to participate.
- Add family intervention early; start with psychoeducation and referral to the appropriate consumer group depending on DSM-5 diagnosis.
- Add family dynamic and systematic interventions as the patient stabilizes.
- Rehabilitate in maintenance phase.
- Do not add another modality if the first intervention is adequate for efficacy.

Glick ended the session by describing the advantages and disadvantages of combined therapy. Medication disadvantages include an increased risk for adverse effects and early termination of all therapies and there is a perceived need for medication decreases with psychotherapy. For patients who are biologically oriented, psychotherapy promotes a sense of increased collaboration and targets intrapsychic and interpersonal problems. For patients who are psychologically oriented, medication response relieves hopelessness associated with the lack of improvement in psychotherapy and targets the primary symptoms of the illness. It provides a faster response than either modality alone. Lastly, family and individual therapy can increase medication compliance and medication can increase psychotherapy compliance. ●