More about: Psychosomatic and psychiatric disorders

PSYCHIATRY AND PREGNANCY (GUIDELINES FOR BIOLOGICAL TREATMENT OF PSYCHIATRIC DISORDERS IN PREGNANCY)

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Knowledge of the risks of exposure to psychoactive drugs during pregnancy remains limited, as retrospective cohort epidemiological studies on these drugs are often biased and flawed.[1]

The aim of these literature-based guidelines is to assist physicians with appropriate drug selection for women who contemplate pregnancy or are pregnant and suffer from psychiatric disorders that require drug treatment, and to provide information about the effects of these drugs on the fetus.[1]

Drug treatment decisions entail:

- a physician and informed patient (husband and family)
- risk-benefit analysis where the mother and child are the first priority
- a treatment goal to minimise infant exposure while maintaining maternal emotional health based on available family support
- a family medical history, especially of effective psychoactive drug treatment and the previous and current course of medical and psychiatric disorders
- other biological therapies (e.g. electroconvulsive therapy (ECT)) or
- non-invasive therapies (individual psychotherapy, cognitive behaviour therapy) as alternative treatment options for less serious disorders.

Discuss each treatment option with the patient and document it in the medical record. If serious controversy exists, a second opinion from an informed colleague may help. Close collaboration between the practitioner, psychiatrist and social worker is essential.

Possible potential adverse drug events can be linked to the pharmacokinetic variations in maternal, placental, fetal, or neonatal drug absorption, distribution, metabolism and elimination. Changes in plasma volume, as well as increases in hepatic metabolism and renal clearance, may significantly affect drug levels.[1]

Psychoactive drugs need to be tapered off 2 weeks before the estimated delivery date to minimise neonatal effects.[1]

Effect of psychiatric medication during pregnancy

Schizophrenia

Relative high-potency antipsychotics . Risperidone and olanzapine: data are too limited to provide a recommendation. Haloperidol, perphenazine, and trifluoperazine have a low risk for teratogenic action.

Lower potency antipsychotics , particularly chlorpromazine, have been cited as being teratogenic by some authors, with a small increase in nonspecific congenital malformations.[2]

Depressive disorders in pregnancy

Common medical disorders during pregnancy such as anaemia, gestational diabetes, and thyroid dysfunction may also cause depressive symptoms.[1]

Avoid tricyclic antidepressants because of sedation, maternal hypotension and gastrointestinal and cardiac side-effects.

To maintain serum tricyclic antidepressant concentrations within therapeutic levels, especially during the third trimester, the dose must be increased to 1.6 times that

administered during the first trimester. Maprotiline and monoamine oxidase inhibitors should be avoided because of lack of data. Data on fluvoxamine, citalopram, paroxetine and fluoxetine suggest they do not seem to pose a high risk to the developing fetus in a dose equivalent to that for non-pregnant women. Data on sertraline, bupropion, mirtazapine, nefazodone, trazodone, and venlafaxine are too limited to provide recommendations.[2]

Electroconvulsive therapy . Severely depressed and suicidal patients who refuse to eat or drink or with psychotic features require hospitalisation and ECT. This is relatively safe and effective in all trimesters of pregnancy, especially in high-risk situations, demanding rapid treatment.[1]

Anxiety disorder

Benzodiazepines . Neonatal side-effects include sedation and dependence with withdrawal signs. Administration of the benzodiazepines should be decreased or stopped during the last trimester.[1,2]

Reports of teratogenicity vary. For example, some reports indicate a 0.7% risk for cleft palate with first-trimester exposure; others show no increased risk for oral clefts. Data on other teratogenic effects (e.g. growth retardation, delayed motor development, and mental retardation) are controversial.[2]

Bipolar disorder

Mood stabilising drugs . Lithium is reported to cause congenital cardiovascular malformations (especially Ebstein's anomaly) when used in the first trimester. Its use during the second and third trimesters may cause fetal thyroid goitre.

Cardiac ultrasonography is recommended at 18 - 20 weeks of gestation.[1]

Carbamazepine, valproic acid, and phenytoin use during pregnancy causes facial dysmorphism, cleft lip and palate, cardiac defects, digital hypoplasia, and nail dysplasia.[1]

First-trimester carbamazepine or valproic acid exposure may result in a 0.05 - 0.1% or 3 - 6% risk of spina bifida, respectively.[2]

Try to avoid mood stabilisers during the first trimester of pregnancy.

Lamotrigine/gabapentin . As there are at present no clear data on these medications they should be avoided.[2]

Conclusion

The best strategy to prevent fetal drug exposure is the avoidance of drug therapy during pregnancy. However, it is not always possible in a pregnant woman who has a serious psychiatric illness.

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PSYCHIATRY AND THE MENOPAUSE

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Menopause is defined clinically as the permanent cessation of menstruation for at least 1 year and is accompanied by physiological, hormonal and clinical alterations that reflect changing ovarian function. As many women now live more than one-third of their lives after menopause, a thorough understanding of these changes is important.[1] The average age at menopause is 51 years. Perimenopause is the transition between full reproductive function and the final menstrual period and precedes menopause by 4 - 5 years. Educating women about the physical and clinical manifestations of this transition and the medical interventions that are available may help to allay fears, balance expectations, and prevent significant psychological distress in the long-term.[2]

Both perimenopause and menopause are characterised by biological changes that may alter mood, behaviour and cognition. As menopause approaches, the loss of ovarian follicular function is accompanied by fluctuation (and decline) of hormones of the hypothalamic-pituitary-ovarian axis, in particular oestrogen. In susceptible women, the central nervous system effects of oestrogen may also extend to problems such as insomnia, irritability, mood, cognitive dysfunction and reduced libido. Other wide-ranging symptoms that are common in both men and women in midlife and are probably not attributable to hormonal changes include fatigue, nervousness, headaches, irritability, joint and muscle pain, dizziness, and palpitations.

Vasomotor symptoms, namely hot flushes and night sweats, are common and secondary to the influence of oestrogen on hypothalamic neuroendocrine and thermoregulatory activity.[3] In some women, frequent and persistent hot flushes may disrupt night sleep and produce symptoms of fatigue, dysphoria and poor concentration that are difficult to differentiate from the somatic manifestations of depression.[4]

Menopause and depression

Although the menopause has long been associated with depression, depressive symptoms are neither specific nor universal to this time in a woman's life. Many symptoms and signs of mood disturbance have been attributed to oestrogen although it has been argued that these are not specific to the menopause. For example, the Massachusetts Health Study,[5] a longitudinal population-based survey, did not find menopause to be associated with an increased risk of depression, except among women who remained perimenopausal for longer than 27 months. For these women with a long perimenopause, depression was a transitory phenomenon. As they approached postmenopause, rates of depression declined. Studies of women attending gynaecology and general practice clinics suggest that depression is commonly endorsed during menopause, yet most community-based surveys of women contradict this assertion.Additionally, women who seek medical care for menopausal problems are more likely to report symptoms of emotional distress than women who do not seek care.[6] That said, there is no hard evidence that menopause puts women at an increased risk for depression.While many women do develop depressive symptoms in the perimenopausal period, women who have had previous episodes of depression (especially premenstrual or postpartum depression), surgical menopause, or severe social or life stresses are more likely to be at an increased risk, especially when oestrogen levels change perimenopausally.[1,7] Further, in these women depression may be multifactorial in origin, with biological (sex steroid), psychological, and social influences contributing to risk. Careful assessment of the role of these factors should be considered in any symptomatic woman who presents for care.

Management

As women are more likely to present to the physician with complaints of irregular bleeding, vasomotor symptoms, genitourinary problems and sexual dysfunction, a thorough physical examination is absolutely indicated to rule out genitourinary problems, cardiovascular disorder, thyroid disorder, osteoporosis, cancer, and sexual dysfunction. Education along with disease prevention, counselling, support and scheduled office visits that are not dependent on mood swings, are key to any successful treatment intervention.

When a woman develops major depression during the menopause, a question that arises is whether she should be treated by her family physician, gynaecologist or psychiatrist, or by all concurrently. In general, treatment by a psychiatrist is necessary in women with severe depressive episodes, treatment-resistant depression, suicidality, or psychosis.[8] Accompanying treatment by a psychiatrist may also be necessary where there is a past history of mental illness or where there are severe current life stresses.

The use of hormone therapy (oestrogen, progesterone, androgen) to improve mood in postmenopausal women has been a topic of much interest and debate. In clinical practice, oestrogens, progestogens, androgens and complementary and alternative therapies (e.g. phyto-oestrogens and vitamin B_6) are commonly used to enhance mood and libido in peri- and postmenopausal women. Hormones are also used as adjunctive treatments for other psychiatric manifestations, such as anxiety and psychosis.[4] With respect to oestrogen, conjugated equine oestrogen is probably the most commonly prescribed replacement. Although data are contradictory, there is substantial evidence that oestrogen replacement improves menopausal depressed mood.[9] However, only large pharmacological doses have been demonstrated to improve mood in clinically depressed patients.[7] A meta-analysis of 38 studies of hormone replacement therapy (HRT) found that oestrogen alone had a moderate to major effect on mood,[10] that is, women who used HRT had significantly less depression.When progesterone was added to oestrogen, the positive effects of oestrogen diminished. However, androgens used alone or in combination with oestrogens were the most effective treatment for depression.

In women who have an increased susceptibility to depression, oestrogen may be beneficial as prophylaxis. It may also be an effective adjunctive therapy as it has been shown to potentiate the effects of antidepressants, such as the selective serotonin reuptake inhibitors (SSRIs). In a multicentre geriatric depression trial, oestrogen replacement was shown to augment response to fluoxetine.[11] Further, it has been suggested that menopausal women with major depression may be responsive to lower doses of antidepressant when concurrent oestrogen therapy is used.[1]

Because of the high risk-benefit ratio associated with high-dose oestrogen, women with mild depression and prominent vasomotor symptoms at menopause should be given an initial trial of oestrogen.[4] If there is no improvement with oestrogen, psychotherapy (e.g. cognitive behaviour therapy) with or without an antidepressant medication is warranted. Moderate or severe depressive symptoms should generally be treated with an antidepressant (e.g. an SSRI). Oestrogen

may also be added to this treatment. In perimenopausal or postmenopausal women with treatment-resistant depression, augmentation therapy with oestrogen may be tried although retrospective analyses do not strongly suggest beneficial additive effects of oestrogen.

In summary, depression is not specifically associated with the menopause. However, many women do experience depressive symptoms. For the treatment of major depression, standard therapy remains antidepressants and/or behavioural or cognitive therapies. HRT may provide effective relief of depressive symptoms associated with the menopause in some women.

References available on request.

DELIBERATE SELF-HARM IN ADOLESCENTS

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Deliberate self-harm (DSH) is a quintessential psychosomatic problem that peaks in adolescence (for females) or young adult life (for males). It encompasses a spectrum of behaviours (generally self-poisoning or mutilation) which are performed with or without suicidal intent, which is difficult to gauge, across a range of causes and specific outcomes, viz. fatal (suicide) or non-fatal (parasuicide). DSH may be seen as the acting out and translation of intolerable psychic states into somatic form — internally (as in self-poisoning) or externally (as in self-mutilation).

Table I.

Comorbid diagnoses	**Proportion of serious self-harmers**
Behaviour/personality disorders esp. borderline, antisocial, conduct	43 - 67%
Depressive disorders (including adjustment)	30 - 40%
Eating disorders (bulimia, anorexia)	35 - 40%
Substance abuse disorders	common
Dissociative disorders	frequent
PTSD, panic, overanxious disorders	frequent
Organic mental disorders	above average

• 56% of serious self-harmers have ≥2 disorders

Why they do it

	Reason	**Self report**	**Staff report**
	Wish to die	33%	16%
	To relieve intolerable state of mind	40%	n/a
	To frighten, get back at or make someone sorry	10%	70%
	To release tension	n/a	n/a
	To reassure themselves they are alive and real	n/a	n/a
	Feel lonely/unwanted	>50%	n/a
	Angry with others	common	n/a
	Worries about future	n/a	n/a
	Feel guilt, shame, failure	n/a	n/a
From SA Studies	Unemployment, socio-economic deprivation	17%	17%
From SA Studies	Fear of AIDS	17%	17%
From SA Studies	Academic failure	n/a	n/a
From SA Studies	Teenage pregnancy	n/a	n/a
From SA Studies	Imitation/contagion	n/a	n/a

Precipitants	**Frequency**
Problems with parents	75%
School problems	50%
Boy-/girlfriend problem	50%
Social isolation/work problem	Common
Interpersonal conflict/loss	More in older adolescents
Family and peer conflict	More in younger adolescents

• stressful life exit or entrance events usually precipitate DSH

Epidemiology

Suicide and DSH rates vary with country, ethnicity, time periods, age, gender and definitions used. Most childhood suicides occur in older adolescents (15 - 19 years) as opposed to younger (10 - 14 years), reflecting the DSH rate as well.

Approximately 1% of females aged 15 - 19 attempt suicide each year (by DSH) and 0.3 - 0.5% of males — 25 - 33% of such suicide attempts are medically serious. By the age of 20, 1% of children younger than 10 years and 1.7 - 5.9% of adolescents will have attempted

Treatment/prevention methods

	Evidence-based	Non-evidence-based
Dialectical behaviour therapy	DBT (reduces DSH in borderlines)	Contracts (at best an adjunct for low-risk patients)
Cognitive behaviour therapy	CBT (reduces depression not suicide)	Family therapy (has some evidence base) ± psychological education
	Lithium (?other mood stabilisers) reduces suicide	Method restriction
PS Watch for early emergent suicide ideation, esp. if akathisia	SSRIs (even without depression) reduces suicide	Crisis hotline
	Systematically screen high-risk older adolescents	Media counselling PS Avoid: • coercion e.g. 'I'm delaying discharge till you say you're not suicidal' • benzodiazepines
	Therapeutic community Interpersonal (?other dynamic) psychotherapy	

suicide. Suicide is the fourth leading cause of death for 10 - 14-year-olds and third for 15 - 19-year-olds.

Community samples suggest the rate may be higher, as up to 39% of DSH is unreported. DSH is a common problem and accounts for high service usage across the board. Although prevalence rates for South Africa are unavailable, DSH is the commonest reason for liaison psychiatric assessments.

Types and levels

Between 90% and 95% of DSH occurs as self-poisoning, generally with medications; 5 - 10% occurs as self-mutilation of which there are 3 levels:

- **Major** — this is more severe but less frequent, e.g. limb amputation or enucleation and tends to occur in psychotic illnesses, e.g. schizophrenia, major depressive episode with psychosis.
- **Minor** (stereotypical), e.g. head banging, finger biting. It tends to be repetitive (often daily) and lacks symbolism, occurring with mental retardation, autism, Lesch-Nyhan, Prader-Willi or Tourette's syndromes and occasionally acute psychoses.
- **Moderate,** usually cutting or burning with minor external damage. This is commonest and may be compulsive, episodic or repetitive, usually as part of comorbid Axis I or II disorders.

Comorbidity

This increases with the seriousness of the attempt — up to 90% of serious attempters (patients) have Axis I and/or II diagnoses (as is the case for suicide). Comorbid diagnoses are listed in Table I.

Predisposing factors

- Early parental loss (especially SA black teenagers)
- Deculturation (especially SA Asian teenagers)
- Harsh authoritarian parenting
- Childhood sexual abuse (especially self-mutilation)
- Childhood trauma (79%)
- Disrupted parental care (89%)
- Neglect
- Childhood non-accidental injury (physical abuse)
- Marital violence
- Family history of impulsive DSH.

Risks for later suicide

- Multiple attempts
- Unusual method
- Medically serious attempts
- Avoidance of discovery/help
- Comorbid Axis I and/or II diagnoses
- Early onset (prepubertal) DSH
- Trait impulsivity
- Family history — suicide, DSH, violence
- Availability of lethal methods
- Low cerebrospinal fluid 5 hydroxy indoleacetic acid (CSF 5 HIAA) post DSH.

Management

Every DSH attempter should have a psychiatric assessment within a multidisciplinary service-delivery based system including in- and outpatient options for acute crisis work, stabilisation, extended treatment, follow-up care and monitoring. Treatment and prevention methods are listed in Table II.

Inpatient psychiatric admission should follow for high-risk/vulnerable adolescents who:

- have active suicidal ideation/intent
- cannot commit to not acting out
- are unpredictable, impulsive, agitated
- are psychotic
- have no home support/ supervision.

Treat comorbid Axis I conditions specifically.

Outpatient treatment/follow-up should occur for low-risk patients and high-risk ones post admission only after there has been an interview with carers and firearms/substances are removed/secured. Treatment should have clear goals, be short with closely spaced appointments with active phone reminders for non-attendance and follow-up with flexible crisis appointments as needed. Confidentiality should be assured but tell parents if suicidal acting out is imminent.

Outcome

- 75% improve (psychiatric and social symptoms).
- 1% per year for at least 10 years commit suicide after the index DSH. The risk is highest in the first year after DSH, and double for males.
- 13 - 35% repeat the DSH within 2 years.

Further Reading

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