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Talking to Your Patients About Alcohol, Marijuana, and Substance Use During Pregnancy

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Lessons Learned at the Interface of Medicine and Psychiatry

The Psychiatric Consultation Service at Massachusetts General Hospital sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. During their twice-weekly rounds, Dr Stern and other members of the Consultation Service discuss diagnosis and management of hospitalized patients with complex medical or surgical problems who also demonstrate psychiatric symptoms or conditions. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

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Have you been uncertain about what to tell your patients about using or discontinuing substances during pregnancy? Have you avoided asking questions of or making recommendations to your patients regarding substance use during pregnancy for fear that you would offend them? Have you wished that you had more information about the risks of smoking cigarettes and using drugs during pregnancy and the postpartum period? If you have, the following case vignette and discussion should prove useful.

CASE VIGNETTE

Ms A, a 33-year-old G3P1011 with a history of hyperthyroidism and generalized anxiety disorder, presented to our obstetric (OB) triage unit at 28 weeks and 2 days. On paper, her pregnancy was uncomplicated. Ms A established prenatal care in the first trimester, had serial thyroid hormone levels during the pregnancy showing well-controlled hyperthyroidism, and had a reassuring gestational diabetes screen the week prior to her presentation. Her genetic screening and a recent fetal anatomic survey reflected a normally developing XX fetus. For Ms A and her supportive partner, this pregnancy was going as smoothly as they could have hoped for, except for one thing. She was drinking a bottle and a half of wine daily.

Screening in the first trimester through the widely adopted "5P's" screening tool was negative. This screening tool for substance use and substance use disorders (SUDs) in pregnancy—which inquires about use during the current **p**regnancy, **p**ast use, **p**arental use, **p**artner use, and **p**eer use—is one of several used across the United States.[1,2] The screen was administered by the medical assistant at her initial prenatal visit. Ms A did not disclose any concerning exposures at that time, and her obstetrician's subjective gestalt deemed Ms A low risk.

However, when she arrived to OB triage that day, she explained that drinking wine had become a way to deal with the stresses of raising her 2-year-old toddler while working 70 hours each week as an investment banker. "Things will settle down once I am pregnant," she had reassured herself as she and her partner were trying to conceive. After her 13-week nuchal translucency ultrasound, it became clear that her plan to taper alcohol intake was not working. She noted strong cravings and withdrawal symptoms after brief periods of abstinence. Despite these symptoms that were indicative of physiologic dependence to alcohol, Ms A said she felt intensely ashamed about being unable to stop drinking. She feared that seeking help would lead to being viewed as an unfit mother and possible loss of custody of her infant. Meanwhile, reassurance about her pregnancy during prenatal visits up until that time that "everything was going well" contributed to her denial and towards isolation and secrecy. Those who knew about her pregnancy were unaware of her drinking. Those who knew about her excessive alcohol consumption were in the dark when it came to her pregnancy.

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Clinical Points

- Signs of neonatal abstinence syndrome can emerge shortly after birth, with their severity influenced by the type of drug used, the timing of the mother's last use during pregnancy, and whether the baby is full term or premature.
- While behavioral support is the first-line treatment for smoking cessation during pregnancy, some randomized controlled studies have shown that nicotine replacement therapy is superior to nontreatment or to behavioral support alone in supporting smoking cessation in pregnant patients.
- Under the federal Child Abuse Prevention and Treatment Act, state governments are required to have policies and procedures in place regarding mandated reporting to local authorities of pregnant patients who use substances.

Now at 28 weeks, secrecy was no longer a viable option. She told her partner and her obstetrician about her alcohol intake. This prompted her to present to our triage unit on a Saturday afternoon for medically supervised withdrawal or "detox." Ms A was admitted to our high-risk antepartum service and treated with a Clinical Institute Withdrawal Assessment–driven benzodiazepine protocol. Per institutional policy, a written consent was signed, which permitted collection of maternal samples for toxicology testing to guide clinical management. With her partner at her bedside, Ms A was visited by various specialists to ensure wrap-around care.

She was counseled jointly by addiction and maternal-fetal medicine specialists, and the pharmacotherapeutic options for alcohol use disorder (AUD) in pregnancy were reviewed (including use of naltrexone/Vivitrol, gabapentin, and acamprosate). Community resources were explored, and Ms A was introduced to a peer-recovery coach during her hospitalization. Ms A was torn between the promise of being on the path to recovery and the multitude of uncertainties that awaited her. The range of phenotypes seen in fetal alcohol spectrum disorders (FASDs), coupled with the inability to accurately test prenatally for this condition, was unsettling. Moreover, the requirements for involving the Department of Children and Families (DCF), the child welfare agency in our state, were not clear to her.

After reviewing the risks and benefits of pharmacologic interventions, Ms A was started on oral naltrexone and gabapentin for her AUD, along with a selective serotonin reuptake inhibitor for her worsening depression and anxiety. Her stated goal was abstinence from alcohol. She was referred for weekly visits with an addiction-trained psychiatrist and therapist. A plan for fetal testing was outlined, and referrals were provided to facilitate meeting with our neonatology and high-risk anesthesia teams.

DISCUSSION

How Often Do Women Use Alcohol, Tobacco, and Other Substances During Pregnancy and the Postpartum Period?

About half of the women who use tobacco, alcohol, or marijuana continue to do so (although often at a reduced frequency) after learning of their pregnancy,[3] which roughly mirrors the proportion of pregnancies that are unintended. National household surveys estimate rates of use during pregnancy to be 15% for alcohol, 5% for tobacco, 5% for marijuana, and 5% for opioids.[4–6] While substance use during pregnancy is a leading modifiable risk factor for poor maternal and child outcomes, it is under-recognized by clinicians due to stigma and is the subject of punitive policies that reflect this stigma.[4,7]

Which Congenital Abnormalities and Neurodevelopmental Complications Have Been Associated With Use of Alcohol, Marijuana, Cocaine, and Tobacco During Pregnancy?

Congenital abnormalities and neurodevelopmental complications associated with use of alcohol, marijuana, cocaine, and tobacco during pregnancy are shown in Table 1. FASDs describe a continuum of structural, cognitive, and behavioral deficits[8,9] as well as distinct craniofacial anomalies, including a thin upper lip, smooth philtrum, and small eyes, along with varying degrees of intellectual disabilities and behavioral dysregulation.[10] Beyond these hallmark features, prenatal alcohol exposure can disrupt fetal brain development, which contributes to cognitive deficits and emotional dysregulation. These neurodevelopmental complications have been associated with increasing rates of attention-deficit/hyperactivity disorder (ADHD), social isolation, and learning disabilities, which can persist into adulthood.[11] In addition to neurological and behavioral sequelae, prenatal alcohol exposure has been linked to congenital cardiac, renal, and skeletal system abnormalities, further contributing to long-term morbidity.[12] Importantly, manifestations of FASD can arise at any stage of childhood and persist throughout life, underscoring the need for early identification and intervention to mitigate adverse outcomes.

While prenatal exposure to cannabis has been viewed as being safe in pregnancy, it has been associated with low birth weight, preterm birth, and intrauterine growth restriction in offspring.[13,14] The main active compound in marijuana, δ-9-tetrahydrocannabinol (THC), readily crosses the placenta and directly affects fetal brain development. THC interacts with the endocannabinoid system, which plays a critical role in neurogenesis, neuronal differentiation, and synaptic plasticity. Disruptions in these neurobiological processes during critical phases of brain development may contribute to

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Table 1.

Congenital Abnormalities and Neurodevelopmental Complications Associated With Prenatal Exposure to Alcohol, Marijuana, Cocaine, and Tobacco

Substance	Congenital abnormalities	Neurodevelopmental complications
Alcohol (fetal alcohol spectrum disorders)	Dysmorphic craniofacial features (eg, smooth philtrum, microcephaly, short palpebral features, midface hypoplasia) Brain structural and functional abnormalities (corpus callosum agenesis, cerebellar hypoplasia) Congenital heart defects (eg, septal and conotruncal defects) Renal malformations (eg, renal hypoplasia, cross-fused ectopia) Limb and joint anomalies (eg, clinodactyly, hockey stick crease, short height)	Intellectual and cognitive deficits (eg, lower intelligence quotient [IQ], impaired working memory, problem-solving disability, and attention problems) Behavioral and psychiatric disorders (eg, attention-deficit/hyperactivity disorder [ADHD], conduct disorder, heightened aggression, emotional dysregulation, depression, anxiety) Social and emotional difficulties (eg, poor social skills) Language and motor deficits (eg, delayed speech, poor coordination, difficulty with balance)
Marijuana	Increased risk of gastroschisis Low birth weight Small head circumference	Cognitive impairments (in memory, attention, executive function) Increased risk of ADHD Delayed motor development Difficulties with emotional regulation Increased risk of anxiety and depression in adolescence
Cocaine	Skull malformations Cardiac defects (eg, ventricular septal defects) Gastrointestinal malformations (eg, intestinal atresia) Urogenital anomalies Limb reduction defects	Poor attention and impulse control Decreased cognitive performance Increased risk of behavioral disorders (externalizing behaviors) Poor emotional regulation Delayed development of language Increased risk of learning disabilities
Tobacco	Orofacial clefts (cleft lip and palate) Congenital heart defects (eg, septal defects, transposition of great arteries) Limb abnormalities clubfoot Intrauterine growth restriction Placental abnormalities (placental abruption, previa)	Lower IQ scores Increased risk of ADHD Behavioral problems (externalizing behaviors, aggression) Impaired auditory processing

long-term cognitive and behavioral impairments.[15] Furthermore, children who have been exposed to marijuana *in utero* are more likely to develop mental health disorders and externalizing behaviors (eg, hyperactivity and impulsivity).[16,17]

Prenatal exposure to cocaine has been linked with intrauterine growth restriction, preterm births, and an increased risk of a neonatal abstinence syndrome (NAS), which can result in withdrawal symptoms (eg, irritability, feeding difficulties, and respiratory distress in offspring).[18] The detrimental effects of prenatal cocaine exposure are largely attributed to its vasoconstrictive properties, which impair placental blood flow and reduce the delivery of oxygen and nutrients to the fetus. This placental insufficiency can lead to fetal hypoxia and restricted growth, which increases the risk of being born with a low birth weight and manifesting developmental delays. In addition, cocaine exposure disrupts fetal neurotransmitter systems, particularly dopamine and serotonin pathways, which are crucial for normal brain development.[19] Moreover, prospective epidemiologic studies suggest that cocaine consumption during pregnancy can lead to a heightened risk for cognitive and behavioral difficulties.[18,20] Maternal smoking during pregnancy is a well-established risk factor for low birth weight, preterm birth, and respiratory complications in infants.[21,22] Nicotine, the psychoactive compound inhaled from tobacco smoking, electronic nicotine delivery systems (ie, vaping), or via buccal mucosae, and various nicotine replacement therapies constrict uterine and placental vasculature, thereby reducing oxygen and the nutrient supply to the fetus. Prenatal tobacco exposure has also been associated with congenital anomalies (eg, cleft lip and palate), highlighting the potential teratogenic effects of the hundreds of compounds released from the combustion of tobacco.[21] In addition to physical defects, accumulating evidence suggests that *in utero* exposure to tobacco smoke may disrupt neural pathways that are critical for regulation of cognition and behavior. For example, children born to mothers who smoked tobacco during pregnancy have a higher incidence of ADHD, learning disabilities, and other neurobehavioral disorders,[23,24] although underlying mechanisms are not well elucidated.[25,26] The nature and severity of neurodevelopmental complications associated with intrauterine exposure to commonly used licit and illicit drugs in epidemiologic studies are shown in Table 2.

What Are the Manifestations of Drug Withdrawal in Newborns at Delivery?

NAS refers to signs and symptoms in the newborn attributed to the cessation of prenatal exposure (via placental transfer) to a wide variety of licit and illicit substances including prescribed medications.[27] Common neurological signs include tremor, irritability, and high-

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Table 2.
Clinical Signs and Symptoms of Neonatal Abstinence Syndrome (NAS) by Physiologic System

Physiologic system	Signs and symptoms of NAS
Neurological	Tremor, irritability, high-pitched cry, seizures, sleep disturbances, hyperactive deep tendon reflex, exaggerated Moro reflex
Gastrointestinal	Poor feeding, excessive suckling, vomiting, diarrhea, uncoordinated swallow patterns
Autonomic	Hyperthermia, sweating, sneezing, yawning
Respiratory	Tachypnea, nasal flaring, respiratory depression
Cardiovascular	Tachycardia, hypertension, arrhythmia
Metabolic and endocrine	Poor weight gain, electrolyte imbalance, hypoglycemia
Integumentary	Skin irritation, severe diaper rash

pitched crying.[28] Signs of NAS can emerge shortly after birth, with their severity influenced by the type of drug used, the timing of the mother's last use during pregnancy, and whether the baby is full term or premature. For instance, withdrawal from opioids and alcohol typically begins within 24 to 72 hours after delivery. Without appropriate management, withdrawal symptoms can interfere with the newborn's healthy development and often necessitates medical interventions (eg, pharmacologic treatment or supportive care).

A subset of NAS, neonatal opioid withdrawal syndrome (NOWS), describes the specific signs and symptoms attributed to the abrupt discontinuation of opioid agonists (including medications used to treat opioid use disorder) in the fetal circulation upon delivery. Despite known risks associated with NOWS, abrupt discontinuation of opioids before delivery in physiologically dependent pregnant patients is contraindicated. Increased oxygen demands associated with acute opioid withdrawal reduces available oxygenated blood to the placental and fetal circulation, which risks fetal distress or even demise.[29]

Why Might Nicotine Replacement Therapy Be Safer Than Cigarette Smoking or Vaping?

While behavioral support is the first-line treatment for smoking cessation during pregnancy, medications may also be necessary.[30] Although current data have not yet documented the effectiveness of nicotine replacement therapy (NRT) during pregnancy, some randomized controlled studies have shown that NRT is superior to nontreatment or to behavioral support alone in supporting smoking cessation in pregnant patients.[31] Success rates may be even higher when using a higher dose of NRT, because pregnancy increases the metabolism of nicotine.[32] Although NRT exposes the fetus to nicotine, NRT avoids fetal exposure to tobacco smoke and other toxins found in cigarettes that are known teratogens, thereby contributing to its safety when compared to cigarette smoking.[33] For the same reason, NRT is preferred to use of electronic cigarettes (vaping). Electronic cigarettes aerosolize additives that are created by heating propylene glycol and vegetable glycerol; unfortunately, the safety of fetal exposure to these additional components has not been well studied.[34]

In addition, NRT exposes the mother to continuous low doses of nicotine, rather than the acute nicotine spikes seen during cigarette smoking, which makes it theoretically safer to the fetus given that concentrations of nicotine are higher in the fetal serum than in the maternal serum.[35] Despite animal studies that have raised concern regarding the risks to a fetus with nicotine exposure, human studies have not identified any consistent association between NRT during pregnancy and adverse fetal or pregnancy outcomes. Therefore, experts typically counsel pregnant patients to use NRT only when they are committed to smoking cessation and after a thorough discussion of the risks and benefits.

When, Where, and To Whom Must Clinicians Report Maternal Use of Substances During Pregnancy and the Postpartum Period?

Under the federal Child Abuse Prevention and Treatment Act, state governments are required to have policies and procedures in place about mandated reporting to local authorities of pregnant patients who use substances. These policies, that vary by state, may be broadly divided into the 14 jurisdictions that explicitly define prenatal substance use as an act of child abuse or neglect that requires mandated reporting by health care providers to child protective authorities and states that leave the decision to report to the discretion of providers based on the extent to which parental substance use is expected to endanger the well-being of the infant after delivery.[36,37] Such mandates have been the subject of intense controversy.[38]

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The prevailing view of substance use during pregnancy as a form of child abuse and neglect not only diverges notably from the modern disease model of addiction that favors treatment over punitive action but dissuades expectant parents from seeking prenatal care,[39] treatment for addiction,[40] and participation in developmental research aimed at informing intervention and prevention strategies.[7] Furthermore, the criminalization of substance use by pregnant women has been applied disproportionately in ways that magnify systemic racial inequities.[41] Pregnancy is associated with a more robust protective effect on the risk of alcohol, tobacco, and illicit drug abuse than any known intervention.[42–44] In the absence of substance abuse treatment programs designed to meet the needs of new mothers of infants via the provision of childcare and parenting training and supervision, criminalization ironically removes the most salient motivation for recovery for many parents.[45]

How Can Clinicians Communicate Effectively With Their Patients (and their partners) About the Hazards of Drug Use and Abuse?

Unfortunately, the causal effects of prenatal exposure on offspring outcomes are not well-established. With the exception of alcohol, a known teratogen, the extent to which prenatal exposures directly influence fetal brain development, are markers for familial and social processes that covary with exposure, or are some combination is an intense area of ongoing inquiry.[26,33,46] Within-family and prospective cohort studies of children parentally exposed to tobacco and other drugs support the confounding of exposure-outcome associations by familial influences.[47] The myriad demographic, genetic, and phenotypic characteristics associated with substance use during pregnancy influence neurodevelopment directly and indirectly via interactions with environmental factors.[25,48–50] Gestational and fetal characteristics[51]; the timing, frequency, dosage, and patterns of use[52]; and conditions in the postnatal environment are further implicated in neurodevelopmental outcomes associated with prenatal exposures.[53]

Within this context, emphasis in primary care settings should be on screening, identification, brief intervention, and referral to treatment, as appropriate, for pregnant and nonpregnant patients. While some providers are hesitant to address potentially stigmatizing topics (eg, substance use) with patients, there is strong evidence for the efficacy and acceptability of such care in primary care settings.[54,55] Beyond minimizing blame, the emphasis on support for behavioral change and treatment, when indicated, not only removes unhelpful blame and shame but also addresses the most salient modifiable influences on familial well-being once exposure has occurred.

SUDs are highly comorbid with mood and anxiety disorders in both sexes,[56] and the persistent use of substances after pregnancy is strongly associated with psychiatric symptoms.[57] Therefore, screening for psychiatric conditions and referral for specialized care, when appropriate, are critical to the optimization of maternal and child health.

What Happened to Ms A?

Ms A returned to the labor and delivery suite for a scheduled induction at 39 weeks. The OB nurse involved in her care at 28 weeks was present for the uncomplicated vaginal delivery of Ms A's vigorous baby girl (who had Apgar scores of 9 and 10). Although her 11 weeks in recovery had been a winding road filled with moments of optimism, occasional lapses to alcohol use, and worries about what to expect in the postpartum period, Ms A continued to use her medications for AUD and had frequent follow-up visits scheduled for her high-risk postpartum period. Ultimately, with an informal plan of safe care in place, opening a case file with DCF was deemed unnecessary, as it was not in the family's best interest.

Ms A's courage to present for care at 28 weeks was her first step toward recovery. Ms A (a white, partnered woman with financial and social assets) sought help at 28 weeks, and her plea was answered. For many pregnant women, requests for help during pregnancy go unanswered and worse, lead to parental punishment.

CONCLUSION

Unfortunately, a myriad of demographic, genetic, and phenotypic characteristics associated with substance use during pregnancy influences neurodevelopment directly and indirectly via interactions with environmental factors. Moreover, while substance use during pregnancy is a leading modifiable risk factor for poor maternal and child outcomes, it is under-recognized by clinicians due to stigma and becomes the target of punitive policies that reflect this stigma.

Roughly half of infants who have been exposed to alcohol or other substances during their fetal development are delivered prematurely. While the exact mechanisms are unclear, ongoing use of many substances during pregnancy has been linked to adverse neurodevelopmental and cardiometabolic outcomes across the lifespan. The prevalence of intrauterine exposure to addictive substances is probably underestimated due to stigma, misperceptions (by patients and clinicians alike), and the criminalization of SUDs in pregnant women.

The prevailing view of substance use during pregnancy as a form of child abuse and neglect diverges from the modern disease model of addiction (that favors

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treatment over punishment) and dissuades expectant parents from seeking prenatal care, treatment for addiction, and participation in developmental research aimed at informing intervention and prevention strategies. Thus, consistent and compassionate conversations with patients facilitate the prevention of adverse sequelae.

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