case of evaluated mirtazapine levels in breastmilk at the time of expected maximum postdose plasma levels under the assumption that breastmilk levels peak at similar timepoints.

A 35-year-old, 60 kg, primiparous, breastfeeding woman who was prescribed mirtazapine (22.5 mg/day) approached our outpatient service for concerns about her 6-week-old child`s possible exposure to mirtazapine through her breastmilk. After description of the study procedure to the patient, written informed consent was obtained for herself and her child. We examined breastmilk levels following 14 days of mirtazapine therapy to ensure that steady state was reached. At the time of assessment, the mother was breastfeeding exclusively. Breastmilk was collected at 4 and 10 hours postdose, foremilk and hindmilk were collected separately. Because of nighttime medication intake, maternal and infant plasma could not be obtained until 12.5 hours postdose. Mirtazapine levels (mass transition, 266 >165) were determined by tandem mass spectrometry, as described previously (2), with an interday imprecision of 12%. Mass transition characterizes the mirtazapine molecule. In tandem mass spectrometry, the mother molecule is broken into fragments, and 165-dalton fragment is taken to characterize and quantify mirtazapine. Although considerably higher levels were found in the milk 4 hours postdose (130 ng/ml foremilk, 145 ng/ml hindmilk) compared with 10 hours postdose (61ng/ml foremilk, 90ng/ml hindmilk), the weight-adjusted maternal dose was still relatively low, ranging from 3.9%–4.4% at 4 hours to 1.8%–2.7% at 10 hours. Infant plasma levels were not detectable at 12.5 hours postdose. Weekly follow-ups showed no abnormalities of the infant, especially regarding sedation or weight gain. The infant's weight at 6 months was 6.3 kg, i.e. consistently below the 25 percentile even before mirtazapine therapy.

Mirtazapine is indeed excreted in breastmilk with slightly higher hindmilk than foremilk levels. Given the minimal infant exposure to the drug and lack of adverse events in our case report, substitution of feeding did not seem necessary at that time. However, because of the scarcity of reports on mirtazapine exposure to nursed infants, interindividual differences are not known. Further research in a larger cohort and a longitudinal design assessing changes in infants' behavior as well as behavioral problems in childhood are needed to confirm the compatibility of mirtazapine treatment during breastfeeding.

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False Positive Phencyclidine Results Caused by Venlafaxine

TO THE EDITOR: We report the case of an unexpected positive result on routine urine drug screening for phencyclidine in a patient who assured that she had never used such substance. Because similar results have recently been reported (1, 2) and in order to avoid inappropriate suspicion by medical caregivers, we performed a blood test that confirmed the absence of phencyclidine and the interpretation of the former urine test as a false positive result.

"Ms. A," a 48-year-old patient with a 31-year history of recurrent depressive disorder, was admitted to the psychiatric hospitalization unit for an acute exacerbation of her mental disorder. She was receiving treatment with venlafaxine 225 mg/day, lamotrigine 100 mg/day, and lormetazepam 2 mg/day. During the prior month she experienced worsening of the depressive symptoms, concurrent with mood-incongruent psychotic symptoms that were not of a clearly depressive nature (thought broadcasting, paranoid and mystic delusions, delusions of control and influence, and grossly disorganized behavior). A routine drug screening by the qualitative immunoassay INSTANT-VIEW Multi-Drug Screen Urine Test (Alpha Laboratories) upon admission was positive for phencyclidine and negative for other drugs. The package insert for the test (3) shows cross-reactivity to methylphenidate, pheniramine, and tenocyclidine, but not for venlafaxine. Given the importance of knowing the origin of the psychotic symptoms, because she would meet DSM-IV criteria for schizoaffective disorder if a toxic etiology was excluded, we performed another urine test 2 weeks later that was also positive for phencyclidine. We then extracted, on the same day, blood and urine samples to check this result by means of a gas chromatography-mass spectrometry analysis. This technique revealed the absence of phencyclidine in blood and in urine (and confirmed that the two prior positive urine tests for phencyclidine were false positives) and confirmed the presence of venlafaxine in blood and of venlafaxine and norvenlafaxine in urine. The urine analysis by gas chromatography-mass spectrometry revealed only prescribed drugs and caffeine. The patient was hospitalized and compliance of the other prescribed drugs was adequate. The diagnosis was changed to schizoaffective disorder, and treatment with amisulpride 100 mg/day yielded a significant improvement of the psychotic symptoms.

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