Letters

RESEARCH LETTER

The Untapped Potential of Office-Based Buprenorphine Treatment

Opioid abuse and dependence are reaching epidemic proportions in the United States, resulting in a staggering number of overdose deaths\(^1\) and economic costs that exceed $56 billion annually.\(^2\) Medication-assisted therapies, such as buprenorphine (Suboxone) and methadone, represent the most efficacious treatments for opioid dependence. Office-based buprenorphine treatment is especially well positioned to provide a rapid response to the opioid crisis, particularly in rural areas where efforts to expand methadone clinics often face sizeable barriers and opioid-dependent patients are widely dispersed throughout a large geographic area.\(^3\) However, there has been increasing concern that these life-saving medications are severely underused.\(^4\)

Methods | We analyzed billed buprenorphine pharmacy claims data from Vermont Medicaid beneficiaries for each of 3 consecutive months (February-April 2014). An average was calculated for the number of buprenorphine patients treated by each physician buprenorphine provider during this 3-month period. The University of Vermont determined that this study did not require institutional review board approval and patient consent was not obtained because patient data were deidentified and only aggregate patient totals were used.

Results | During the evaluation period, 1964 Vermont residents received at least 1 buprenorphine prescription. Their prescribers consisted of 133 physicians (of the approximately 190 currently waivered physicians in Vermont), translating to an average of 14.8 patients per physician buprenorphine provider (range, 1-76). However, closer inspection of the data revealed that most physician buprenorphine providers were only treating a small handful of patients (Figure), with 29.3% of physicians having only a single buprenorphine patient and 48.1% treating 5 or fewer patients.

Discussion | Vermont has long been at the forefront of buprenorphine treatment. Our university was the site of National Institutes of Health-funded clinical trials demonstrating buprenorphine’s efficacy prior to its Food and Drug Administration approval. In recent years, Vermont has been identified as a model for other states to follow in their efforts to develop and expand office-based buprenorphine treatment, with a per capita rate of buprenorphine prescriptions that is more than 10 times the national average.\(^5\)

However, despite Vermont’s early and aggressive adoption of office-based buprenorphine for opioid dependence, pharmacy claims data suggest that we are far from realizing the potential of this important treatment modality. Indeed, our current use translates to approximately 10% of the maximum capacity possible with 190 waivered physician buprenorphine providers, which is significant underutilization in a rural state that desperately needs more treatment slots. These data also contrast sharply with an earlier national survey in which physician providers typically treated 25 to 40 buprenorphine patients.\(^6\)

Whether the Vermont experience is representative of other states is unclear. However, what is certain is that we cannot afford a backslide in our efforts to develop a robust office-based buprenorphine system. An improved understanding is needed of the factors limiting buprenorphine’s widespread use including physicians’ concerns about induction logistics, reimbursement challenges, and the potential for medication abuse or diversion. Efforts to address these barriers may include greater initial and ongoing support for physician buprenorphine providers, improved methods for screening and identifying the patients most appropriate for office-based buprenorphine, and improved methods for monitoring patient progress and medication adherence. Taken together, we must think hard about how to better support our physician buprenorphine providers, as this treatment modality plays a vital role in our ability to respond appropriately to the country’s opioid abuse epidemic.

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Figure. Patient Density Among Physician Buprenorphine Providers

The data points represent the number of physician buprenorphine providers (y-axis) who are treating the number of buprenorphine patients depicted along the x-axis. For example, 39 physician providers were treating a single buprenorphine patient whereas 7 physician providers had 2 buprenorphine patients.

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Amygdala-Hippocampal Volume and the Phenotypic Heterogeneity of Posttraumatic Stress Disorder: A Cross-Sectional Study

The amygdala and hippocampus have been implicated consistently in the pathophysiology of posttraumatic stress disorder (PTSD). While several studies have observed reduced hippocampal volume in PTSD, studies of amygdala volume and PTSD have been mixed. In addition to method differences, one reason for these mixed results is that most structural magnetic resonance imaging studies in PTSD have treated PTSD as a homogeneous entity instead of considering how amygdala volume may relate to its heterogeneous phenotypic expression.

Confirmatory factor analytic studies have revealed that PTSD is best represented by 5 symptom clusters: reexperiencing, avoidance, numbing, dysphoric arousal (eg, sleep difficulties), and anxious arousal (eg, hypervigilance). To our knowledge, no study has evaluated the relation between amygdala and hippocampal volume and this contemporary model of PTSD. Here, we evaluated these associations in combat veterans.

Methods | Forty-eight Iraq/Afghanistan combat veterans participated in this study. Recruitment was conducted to ensure a full dimensional range of DSM-IV PTSD symptoms (ie, including non/minimally symptomatic veterans and equal proportions of veterans with mild, moderate, and severe/extreme symptoms), with 23 veterans (47.9%) meeting diagnostic criteria for combat-related PTSD. Exclusion criteria included psychosis; bipolar disorder; drug abuse or dependence (current or lifetime); alcohol abuse in the past 30 days or alcohol dependence in the past 12 months; moderate and severe traumatic brain injury (ie, loss of consciousness >30 minutes); neurologic disorder (eg, stroke or seizure); learning disability or confirmed diagnosis of attention-deficit/hyperactivity disorder; use of antipsychotics, psychostimulants, or sedatives/hypnotics; antidepressant dose stable less than 30 days; and/or PTSD diagnosis prior to combat exposure. The VA Connecticut Healthcare System Human Subjects Subcommittee and Yale University Human Research Protection Program approved this study. All participants provided written informed consent.

Structural magnetic resonance imaging data were acquired on a Siemens Trio TIM 3T (MPRAGE; voxel size 1 x 1 x 1 mm; repetition time, 2.5 seconds; echo time, 2.77 milliseconds; flip angle, 7°). Blinded to the clinical status, image processing and segmentation were conducted using the fully automated Freesurfer recon-all pipeline (http://surfer.nmr.mgh.harvard.edu).

We computed partial correlations between independent variables and amygdala and hippocampal volumes adjusted for total intracranial volume and entered variables with associations at the P < .05 level into a multivariable linear regression analysis using total intracranial volume as a covariate. To evaluate subscales of the Clinician-Administered PTSD Scale associated with volumes, we conducted a post hoc multivariable linear regression analysis (α = .01). Finally, to evaluate interrelationships among variables related to regional volumes, exploratory path analyses were conducted using Mplus version 7.2 (http://www.statmodel.com).

Results | The Table shows sample characteristics and partial correlation results. After adjustment for intracranial volume, Combat Experiences Scale and total Clinician-Administered PTSD Scale scores were independently associated with right amygdala volume. Multivariable linear regression for right amygdala volume showed adjusted R² = 0.46 (Combat Experiences Scale: β = −0.27; t = 2.34; P = .02; Clinician-Administered PTSD Scale: β = −0.24; t = 2.10; P = .04). Post hoc analysis revealed that anxious arousal was independently negatively related to right amygdala volume (β = −0.38; t = 3.33; P = .002); no other symptom cluster was significant (β > −0.08; t < 0.53; and P > .59 for all). The best-fitting model in path analyses showed right amygdala volume mediating the relationship between combat exposure and anxious arousal (χ² = 0.03; P = .87; Bayesian Information Criterion = 921.38; Akaike Information Criterion = 906.41; root mean square error of approximation = 0.00 [0.00-0.20]; Comparative Fit Index = 1.00; Tucker-Lewis Index = 1.00; the other 2 models had χ² = 3.17 or higher, P = .07 or lower, and higher root mean square error of approximation and lower Comparative Fit Index and Tucker-Lewis Index values, which indicate worse fit). The Figure shows standardized coefficients of the best-fitting model.