P02-455

VENLAFAXINE AND CYP2D6 IN CLINICAL PRACTICE: AN OBSERVATIONAL STUDY

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Introduction: Venlafaxine is a serotonin-norepinephrine inhibitor, mainly metabolized by CYP2D6 to its active metabolite ODV. Depending on CYP2D6 activity, patients may be identified as Poor, Intermediate, Extensive or Ultrarapid Metabolizers. There is some evidence that a PM phenotype is associated with poor tolerance more often than an EM; while a UM patient would only respond to a greater dose of Venlafaxine[1].

Objectives: To evaluate the impact of CYP2D6 phenotype on the efficacy of Venlafaxine XR in depressed patients.

Methods: This observational study evaluated 27 Caucasian adult patients (F=18, M=9), satisfying DSM-IV criteria for Major Depressive, Bipolar Disorder or Personality Disorder receiving treatment with Venlafaxine 75-300mg/die.

CYP2D6 alleles were evaluated with INFINITI CYP2D6 assay, which employs AutoGenomics proprietary film-based microarray technology.

Results: Most patients were identified as EMs, 4 as PMs, while only one was identified as UM. The only statistically significant difference between Extensive and Poor Metabolizers was, in contrast with current literature, the need of a greater mean dose of Venlafaxine in the second group (225 mg/die vs 159.38 mg/die, t student: p=0.01).

Likewise, in contrast with literature, the UM patient was responsive to average doses of Venlafaxine.

On the contrary, we found no statistically significant differences as far as efficacy, adverse events or duration of treatment are concerned.

Conclusions: In our sample, CYP2D6 metabolizer status does not seem to affect treatment response nor adverse events related to Venlafaxine.

https://doi.org/10.1016/S0924-9338(11)72756-0 Published online by Cambridge University Press