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Letters to the Editor

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CHLORPROMAZINE AND FALSELY ELEVATED PARTIAL THROMBOPLASTIN TIME

Dear Editor:

The introduction of chlorpromazine in 1952 revolutionized the treatment of psychiatric disorders. By 1954 chlorpromazine had been linked to potential agranulocystosis (1). Over the last 37 years the risk to benefit ratio of the drug has been scrutinized. Chlorpromazine still remains the gold standard by which other antipsychotics must be judged.

We report the case of a 58 year old male with Bipolar Affective Disorder and Alcohol-induced Dementia who presented to the medical service with chest pain. Following evaluation of the patient's symptoms and risk factors it was elected to perform cardiac catherization. Prior to the catherization a protime and partial thromboplastin time were performed. The PT was normal at 11.2 seconds; however the PTT was markedly elevated at 51.5 seconds. Additional laboratory work including CBC and numerical platelet count was unremarkable.

Hematology Service was consulted to evaluate the elevated PTT. They recommended mixing studies which were compatible with a coagulation inhibitor. A Russel's Viper Venom Test was positive for a Lupus Antibody. The remainder of the hematology workup was negative and included RPR, ANA, ANA Antibody, and Rheumatoid Factor.

Psychiatric consultation was requested to determine if the patient's psychotropic medication was effecting the coagulation studies. The patient was on chlorpromazine, lithium carbonate and clonazepam. His chlorpromazine was discontinued and an equivalent dose of halperidol was begun. His PTT corrected within one week. Repeat PTT's obtained at 2, 4, and 6 months were normal and no further abnormality was noted.

The lupus antibody is an IgM, or IgG immunoglobulin that produces a prolonged PTT by binding to the phospholipid used in the in vitro PTT assay. This laboratory artifact does not cause a clinical bleeding disorder. The lupus anticoagulant is seen in 5–10 percent of patients with Systemic Lupus Erythmatosus, but can be seen in patients on phenothiazines as with our patient. (2)

Lupus like syndromes have been induced by phenytoin, hydralazine, procainamide and isoniazid. The phenothiazines, thioxanthenes, butyrophenones and lithium have also been reported to induce ANA in some patients with chronic use. (3,4)

Zarrabi, in 1979 demonstrated that most long term patients on chlorpromazine developed immunologic abnormalities of significant IgM and prolongation of PTT. Fifty percent of these patients also developed spleenomegaly. (5)

Our purpose in writing this letter is to caution other clinicians that chlorpromazine can often produce an artifactual elevation in the PTT which is secondary to a circulating Lupus Antibody.

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