

Case report: Chlorpromazine and deep venous thrombosis

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Abstract

Purpose – Since the development of antipsychotic drugs in the 1950s, a variety of studies and case reports have been published that suggest an association between exposure to typical antipsychotics and venous thromboembolisms (VTE). Therefore, when starting treatment with antipsychotics, especially low-potency typical antipsychotics and clozapine, health-care providers must account for the patient's existing VTE risk factors.

Design/methodology/approach – In this case report, the authors describe the development of a pulmonary embolism associated with use of chlorpromazine in the treatment of an acute manic episode in a 51-year-old female patient with bipolar disorder type 1.

Findings – The patient was brought to the emergency room by the police on a legal hold for bizarre behaviors at a bus stop, which included incessantly yelling at bystanders. The patient was found to have disorganized thoughts, poor sleep, rapid speech, labile mood, distractibility, auditory hallucinations and grandiose delusions. During the course of her stay, the patient received extensive IM chlorpromazine for extreme agitation, in addition to chlorpromazine 200 mg IM Q8H, which was later decreased to chlorpromazine 100 mg chlorpromazine IM/PO Q8H. On day 4 of the treatment, the patient experienced difficulty breathing, hypoxia and tachycardia and was found to have bilateral expiratory wheezes. CT angiography showed sub-segmental pulmonary embolus and the patient was transferred to MICU service. The patient was then intubated and started on heparin by the medical team. Over the course of the next day, her respiratory distress resolved and the patient was extubated.

Originality/value – It is possible that chlorpromazine may indeed increase VTEs, and there are various physiological postulations regarding the mechanism of action. However, multiple confounding variables existed in the authors' report, including venous stasis and the use of restraints, tobacco and valproic acid. Each of these variables has been shown to increase VTE occurrence. Further controlled studies are necessary to identify the true relationship between antipsychotics and VTEs.

Keywords Clozapine, Risk factors, Antipsychotic drugs, Bipolar disorder, Chlorpromazine, Venous thromboembolisms

Paper type Case study

Introduction

Since the development of antipsychotic drugs in the 1950s, a variety of studies and case reports have been published that suggest an association between exposure to typical antipsychotics and venous thromboembolism (VTE). Such an association has also been identified with Clozapine (Walker *et al.*, 1997). VTE includes deep vein thrombosis (DVT) and pulmonary embolism (PE), which can be fatal. Therefore, when starting treatment with antipsychotics, especially low-potency typical antipsychotics and clozapine, health care providers must account for the patient's existing VTE risk factors. Clinicians should consider interrupting or changing the antipsychotic regimen in patients in whom this adverse effect is suspected, such as in patients with chest pain, dyspnea or oxygen desaturation. More studies are currently required to determine the incidence rate, the possible predisposing factors and the biological mechanisms involved.

In several studies, such as by Parkin *et al.* (2003) and Zornberg and Jick (2000), low-potency antipsychotics, such as the phenothiazines, were shown to be more strongly associated with increased risk for VTE than high-potency antipsychotics, such as haloperidol. Several hypotheses have been proposed for the biological mechanisms by which antipsychotics may portend increased VTE risk, including: elevation of anticardiolipin autoantibodies (Canoso *et al.*, 1990), enhanced platelet aggregation (Zornberg and Jick, 2000) venous stasis caused by sedation and hyperhomocysteinemia (Ray *et al.*, 2002). We cannot exclude the possibility that the underlying psychiatric disorders themselves are associated with thromboembolic phenomena. In fact, this link has been made by a number of studies, which discuss VTE risk factors present in psychiatric disorders, which are independent of

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