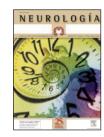


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ORIGINAL ARTICLE

Do cerebral venous thrombosis risk factors influence the development of an associated venous infarction?

I. Sanz Gallego, B. Fuentes, P. Martínez-Sánchez, E. Díez Tejedor*

Servicio de Neurología, Centro de Ictus, Instituto de Investigación IdiPAZ, Hospital Universitario La Paz, Universidad Autónoma de Madrid, Madrid, Spain

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KEYWORDS

Venous infarction; Cerebral venous thrombosis; Risk factors; Thrombophilia; Oral contraceptives

Abstract

Introduction: Cerebral venous thrombosis (CVT) is a multifactorial process with a wide clinical spectrum and many associated risk factors (RF) that could be complicated with venous infarction (VI). We study the influence of RF in the developing of venous infarction in patients with CVT.

Patients and methods: An observational study with consecutive inclusion of patients with CVT diagnosis admitted to the Stroke Unit of a Neurology Department between 1995 and 2007. RF were identified and their distribution according to the presence of VI was analysed.

Results: A total of 52 patients were included (37 female; 71.15%) with mean age of 46.73 years (range 18-78 years). The most frequent RF associated with CVT were thrombophilia (26.92%) and oral contraceptives (OC) (25% of all the patients and in 35.13% of females). The most frequent RF in patients with venous infarction was thrombophilia (40.9%), whilst in the CVT group without venous infarction the use of oral contraceptives predominated (26.7% of the total sample; 38% of females), with thrombophilic states only being detected in 16.5%. No cases of venous infarction were found in the group of patients with oral contraceptives but without an associated thrombophilic state. Conclusion: There appears to be a different profile of associated RF in patients with

venous infarction associated to CVT, with the presence of thrombophilia prevailing.
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E-mail: ediezt@meditex.es (E. Díez Tejedor).

^{*}Corresponding author.

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PALABRAS CLAVE

Infarto venoso; Trombosis venosa cerebral; Factores de riesgo; Trombofilia; Anticonceptivos orales ¿Influyen los factores de riesgo de trombosis venosa cerebral en el desarrollo de infarto venoso asociado?

Resumen

Introducción: La trombosis venosa cerebral (TVC) es un proceso multifactorial con amplio espectro clínico y de factores de riesgo (FR), que puede presentar o no infarto venoso. Estudiamos los FR que influyen en el desarrollo del infarto venoso en pacientes con diagnóstico de TVC.

Pacientes y métodos: Estudio observacional con inclusión de pacientes consecutivos con diagnóstico de TVC atendidos por la Unidad de Ictus del servicio de Neurología entre los años 1995 y 2007. Se identifican los FR y se analiza su distribución en función de la presencia del infarto venoso.

Resultados: Se incluyeron 52 pacientes (37 mujeres; 71,15%) con edad media de 46,73 años (18-78 años). Los factores de riesgo de TVC más frecuentes fueron los estados de hipercoagulabilidad hereditarios (26,92%) y el uso de anticonceptivos orales (ACO) (25% del total muestral y 35,13% de las mujeres). Entre los FR identificados en pacientes con infarto venoso predominan los trastornos de hipercoagulabilidad hereditarios (40,9%) mientras que en los casos sin infarto venoso, el factor más frecuente es el uso de ACO (26,7%; 38% de las mujeres), estando presentes los estados de hipercoagulabilidad sólo en el 16,5%. No observamos ningún caso de infarto venoso con tratamiento ACO y sin estado de hipercoagulabilidad asociado.

Conclusiones: En los pacientes con infarto venoso asociado a TVC parece existir un diferente perfil de factores de riesgo asociado, predominando la presencia de estados protrombóticos hereditarios.

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Introduction

Cerebral venous thrombosis (CVT) is an infrequent clinical entity (0.5% of the total number of strokes)¹ and comprises the occlusion of the flow in the cerebral venous system and its sinuses.²,³ From a pathogenic viewpoint, venous thrombosis is considered to be a continuous process in which the balance between the prothrombotic and thrombolytic processes is disturbed, entailing, over time, the formation of a venous thrombus. This formation is due to factors related to Virchow's triad: venous stasis, alterations in vessel walls and changes in blood composition.³,⁴

Many risk factors have been identified in association with CVT as having additive effects in such a way that CVT is, in the final analysis, a multifactorial process.^{5,6} In fact, the presence of more than one risk factor ha been shown in up to 44% of patients.⁷ Those most frequently associated with CVT are the use of oral contraceptives and hereditary hypercoagulability states.³ In addition, some studies have suggested that the combination of oral contraceptives and thrombophilias considerably increases the risk of CVT.^{6,8-10}

Bearing in mind the wide spectrum of risk factors, the clinical heterogeneity of CVT in terms of both their presenting pattern (acute, sub-acute or chronic) and the accompanying symptoms, with or without the presence of venous infarctions, it is possible that some risk factors may be particularly involved in the development of the latter. On the other hand, there are so far no studies specifically analyzing the presence of different mutations constraining

a prothrombotic state in patients with CVT when comparing patients with and without venous infarction.

The purpose of the present paper is to identify the risk factors influencing the development of venous infarctions in patients with CVT.

Patients and methods

Observational study with the inclusion of consecutive patients diagnosed as having CVT and seen by the Stroke Unit in the Neurology Department of our hospital between 1995 and 2007. The sources for identifying patients has been the Stroke Unit's database and the case histories of the patients admitted to the Neurology Department with a diagnosis of CVT.

For all patients, a note was taken of the following variables: age, gender, symptoms and the presence of the following risk factors: systemic infections or infections in nearby structures (head and neck), mechanical triggers (neurosurgery, cranioencephalic trauma and lumbar punctures performed in the month prior to the start of clinical symptoms), infections, neoplasias, haematological alterations: thrombocytosis (platelet figures in excess of 400,000/mcL), polyglobulia (haematocrit above 50%), anaemia (presence of fewer than 4.2 x 106 erythrocytes per mm³ in males or fewer than 3.6 x 106/mm³ in females), hereditary and acquired prothrombotic states, vasculitis, systemic inflammatory diseases, pregnancy, puerperium,

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