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Fluid Shifts and Cerebral Pseudo atrophy Secondary to Leaky Vessels and Gravity: A Case Report

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Abstract

Introduction: The relevance of fluid shifts related to the impaired competence of the blood brain barrier in the absence of traumatic brain injury may be under-recognized and the clinical impact of shifts of fluids in the brain in the context of an acute systemic illness may pose a challenge to neurologists, intensivists and radiologists.

Case presentation: We report of a 52 year-old man who presented with severe cytomegalovirus pneumonitis resulting in respiratory and kidney failures, and became comatose. Imaging studies documented an unusual picture with bilateral frontal hygromas, frontal lobe pseudo atrophy and fluid accumulation in the posterior cerebral regions all related to the compromised blood brain barrier function under the effect of gravity in the supine position. As the underlying illness improved and the systemic fluid imbalance was gradually corrected, the cerebral volumes reversed to normal and the altered level of consciousness resolved.

Conclusions: The role of the blood brain barrier in the maintenance of intracranial fluids is well recognized in head traumas and in the context of posterior reversible encephalopathy syndrome, while the medical literature on the topic of cerebral volume changes in the settings of inflammatory illnesses is scanty. We highlight the impact of the systemic inflammatory state on the loss of blood brain barrier competence with consequent, reversible global intracranial fluid redistribution and we emphasize the importance of familiarizing with the unusual radiological appearance of such scenarios.

Introduction

The ability of the Blood Brain Barrier (BBB) to preserve the intracranial fluid homeostasis has been the subject of decades of investigations and its morphology has been elucidated at the molecular level [1,2]. In physiologic states, the equilibrium between the CSF production and its re-absorption as well as the interstitial cerebral water content are maintained through the balance of pressures in the different intracranial compartments with a modulating effect of the BBB that relies on tight junctions [3].

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Brain edema associated with traumatic brain injury is a common example of direct disruption of the BBB competence often leading to increased intracranial pressure.

Conversely, intracranial hypotension secondary to ventriculoperitoneal overshunting or situations of spontaneous CSF leaks can alter the intracranial pressure gradient and indirectly cause bilateral subdural hematomas, hygromas, brain tissue herniation and coma [4]. In addition, secondary to many etiologies, the posterior reversible encephalopathy syndrome is known to be caused by the impaired function of the BBB with variable severity of vasogenic cerebral edema [5]. With features that are common to all of the above mentioned conditions, the spontaneous formation of hygromas and the regional redistribution of cerebral fluids in the setting of a systemic illness point to the role of the BBB that has not been sufficiently emphasized in the medical literature. Through the description of a clinical case that manifested an extreme degree of intracranial fluid shifts, we highlight the importance of recognizing non-traumatic brain edema and spontaneous hygroma in critically ill patients.

Case presentation

A 52-year-old man diagnosed with T-cell lymphoblastic lymphoma, status post intrathecal Cytarabine three months prior to presentation, was admitted with superior vena cava syndrome and severe Cytomegalovirus (CMV) pneumonitis. Due to acute respiratory and kidney failures, he required ventilator assistance and hemodialysis. In the absence of any head trauma, he became comatose over the course of a few days. A CT of the head demonstrated changes of brain volume, consisting of regional pseudoatrophy of the frontal lobes with associated bilateral frontal hygromas and accumulation of fluids in the parieto-occipital regions (Figure 1 A-D).

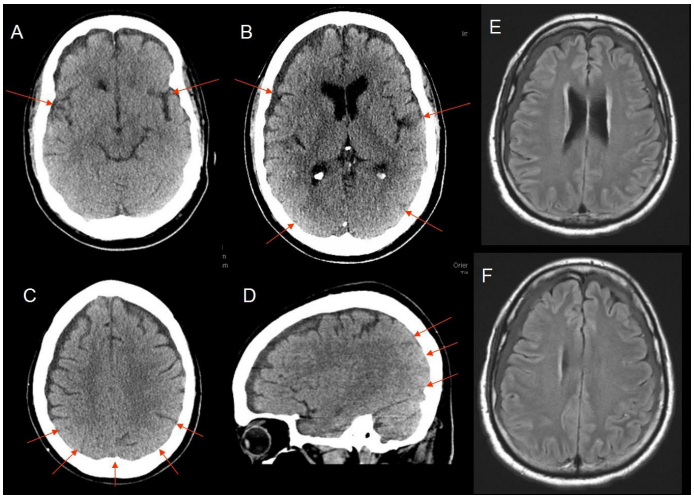

Figure 1: Non-contrast axial **(A,B,C)** and sagittal **(D)** computerized tomography of the head in the acute stage of the illness showing the presence of bifrontal hygromas and the sulcal effacements of the parieto-occipital lobes (arrows) as an effect of gravity in the critically ill patient lying supine. The axial Fluid Attenuated Inversion Recovery (FLAIR) MRI sequences **(E,F)** demonstrated the bifrontal fluid collection with signal characteristics consistent with blood containing hygromas. The frontal lobes show pseudoatrophy while in the parieto-occipital regions there is effacement of the cortical sulci.

The blood tests were remarkable for WBC 14.1 th/mm3, RBC 3.53 mil/mm3, Hb 10.6 g/dL, MCV 87.2 fl, hematocrit 30.7%, platelet 38 th/mm3, absolute neutrophils 13.84 th/mm3, absolute lymphocytes 0.1 th/mm3 and the serum quantitative PCR for CMV resulted at 2184980 copies/mL. The Cerebrospinal Fluid (CSF) was negative for CMV.

We hypothesized that in the setting of the systemic viral illness and the superior vena cava syndrome, an impairment of the blood brain barrier led to a gravity-dependent fluid redistribution in this critically ill patient lying supine. Consistent with the signal characteristics by MRI (Figure 1 E-F), the CSF contained large numbers of red cells (WBC 945/mm3, RBC 8989/mm3) and the bilateral frontal fluid collection was interpreted as blood leakage into the subdural space under the stretching forces of gravity applied to the veins bridging the meninges in the frontal regions in the setting of thrombocytopenia.

The patient received prolonged antiviral therapy with Acyclovir initially, followed by Ganciclovir, and the systemic fluid balance was cautiously corrected via dialysis. Over the course of ten days, the brain volume normalized proportionally to the correction of the systemic fluid adjustments (Figure 2) and the patient regained consciousness, ultimately returning to his baseline cerebral functions.

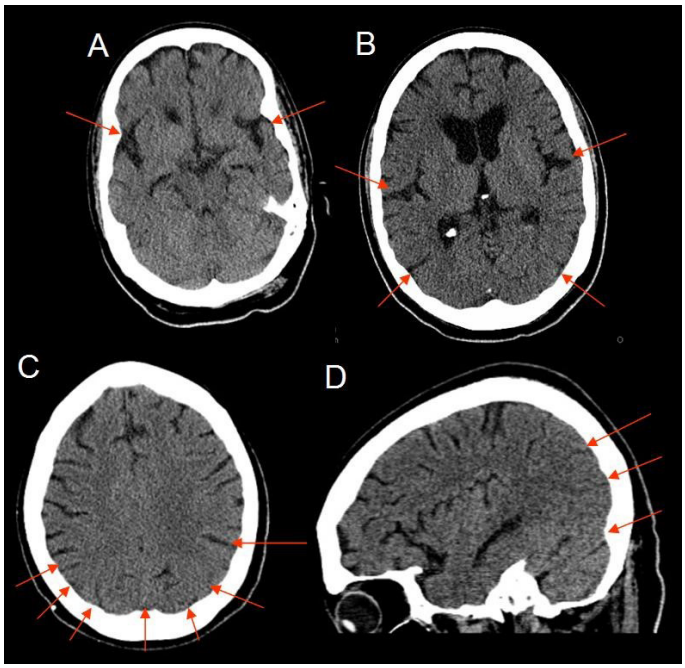

Figure 2: Non-contrast axial **(A,B,C)** and sagittal **(D)** computerized tomography ten days later, when the patient had regained normal alertness, demonstrating the resolution of the frontal hygromas and the cortical effacements as well as normalization of the subarachnoid spaces (arrows).

Discussion

The patient presented here had severe pneumonitis and superior vena cava syndrome that escalated to respiratory and renal failures requiring ventilator support and hemodialysis. Over the course of a few days, the patient became comatose, prompting the cerebral imaging studies. The development of subdural fluid collections with abundant blood contamination can be explained by virtue of the stretching forces applied to the bridging veins across the subdural space. The reversible effects of gravity on the cerebral fluid redistribution, secondary to the compromised competence of the BBB (discussed below), seem to be a major factor in this clinical scenario. The possible contribution of the superior vena cava syndrome to the alteration of the pressure gradient between dural sinuses and intracranial space remains speculative.

The brain parenchyma volume changes with pseudo atrophy of the frontal lobes and fluid accumulation in the posterior regions was the most likely cause of the altered level of consciousness. The temporal relationship (three months separation) made it unlikely that the prior exposure to the intrathecal Cytarabine was a factor.

The permeability of the BBB can be altered in the setting of systemic inflammation and metabolic derangements [6,7]. Numerous in vitro studies have demonstrated a complex interaction between cytokine production and BBB function [8]. In particular, the tight junctions of the endothelial cells of the cerebral blood vessels are specialized structures containing high concentrations of occludin and claudin-5 proteins whose expression is subject to dynamic variations under the influence of phosphorylation and recycling of junctional and cytoskeletal molecules [1]. It is conceivable that this patient's systemic viral infection generated a highly inflammatory milieu that impaired the BBB function creating the premise for vasogenic edema that led to the abnormal and unusual intracranial fluid distribution. As the CMV pneumonitis slowly improved and the whole body fluid balance was gradually corrected, the competence of the BBB was regained.

The distinction between the brain atrophy related to neuronal degeneration and pseudo atrophy secondary to reversible changes associated with resolution of acute inflammation has been subject matter in the medical literature relative to multiple sclerosis [9]. Yet, in the absence of inflammatory findings, pseudo atrophy could be less than easily recognized and possible fluid related volume changes of the cerebral white matter should be considered when interpreting a scan with a seemingly atrophic brain [10].

Other differential diagnoses to be considered include subdural empyema and subdural hematoma [11]. Subdural empyema [12] is usually associated with imaging findings characterized by hypo density on CT and abnormal signal on MRI with hyperintensity on both FLAIR and T2 sequences and hypo intensity on T1. The clinical symptoms can be confounded in a person with severe systemic inflammation and ruling empyema out by CSF analysis and examination is important. Subdural hematoma is characterized by serial changes on MRI as related to the different blood degradation products. Most cases of subdural hematoma are traumatic, which was not the case here.

The treatment for the vasogenic cerebral edema, as observed in this case, stems on addressing the underlying systemic inflammation and carefully adjusting the balance of electrolytes and fluids. It is worth noting how in parallel with the improvement of the CMV pneumonitis, the competence of the BBB was restored, the bifrontal subdural fluid collection completely reabsorbed and with the resolution of the cerebral edema, the normal neurological status was regained.

As long as the condition is recognized and properly treated, a favorable prognosis is to be expected in a case like the one presented here where there is no structural injury to the brain parenchyma.

Conclusions

With our report we highlight the impact of systemic inflammatory states on the loss of blood brain barrier competence with consequent, reversible global intracranial fluid redistribution and we emphasize the importance of familiarizing with the unusual radiological appearance of such case scenarios.

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