THE ROLE OF DEXAMETHASONE FOR MILD TO MODERATE TRAUMATIC BRAIN INJURY; A CATCH-22 SITUATION

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INTRODUCTION

The use of steroids in an acute setting after a traumatic brain injury (TBI) has been controversial. However, recently, with a better understanding of the complex process of post-traumatic cerebral oedema – *biphasic; whereby it is initially cytotoxic followed by vasogenic*,[1] steroids could potentially be a good catch in the management of delayed post-traumatic cerebral oedema, hence avoiding surgical intervention. In this study, we report a case of delayed post-traumatic cerebral oedema and its response to steroid therapy.

METHOD

A case report whereby the patient was followed up prospectively until well.

CASE PRESENTATION

A 61-year-old male complained of moderate-to-severe debilitating headache 10 days after sustaining mild TBI (GCS 14 and left temporal contusion without mass effect). Clinically, his GCS was 13 without pupillary abnormalities and no focal neurological deficit. Repeated computed tomography (CT) brain showed resolving clot but worsening left temporal vasogenic cerebral oedema with mass effect. Magnetic resonance imaging brain also confirmed no mass or evidence of vasospasm. Parenteral dexamethasone 4mg thrice a day was administered for 2 days and then tapered over a duration of 7 days enterally. His condition and symptoms markedly improved within 48 hours and showed complete resolution by day 5. A repeat CT brain 1 week after dexamethasone administration showed drastic reduction of cerebral oedema with resolved mass effect. No adverse effect of steroid was encountered.

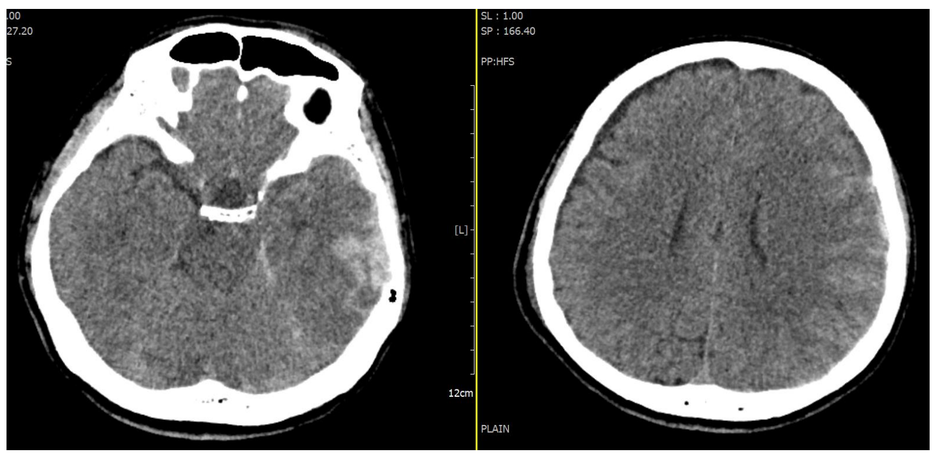

Figure 1: CT Brain on post trauma day 1 showed left temporal contusion bleed without any mass effect.

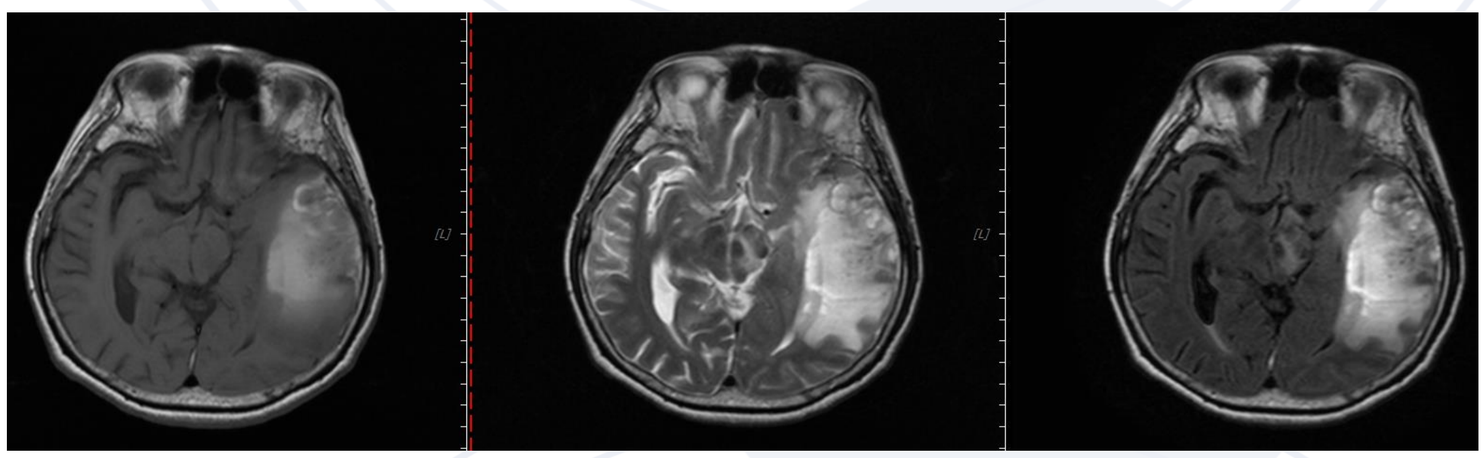

Figure 3: Magnetic resonance imaging of the brain confirmed blood products of varying ages and oedema.

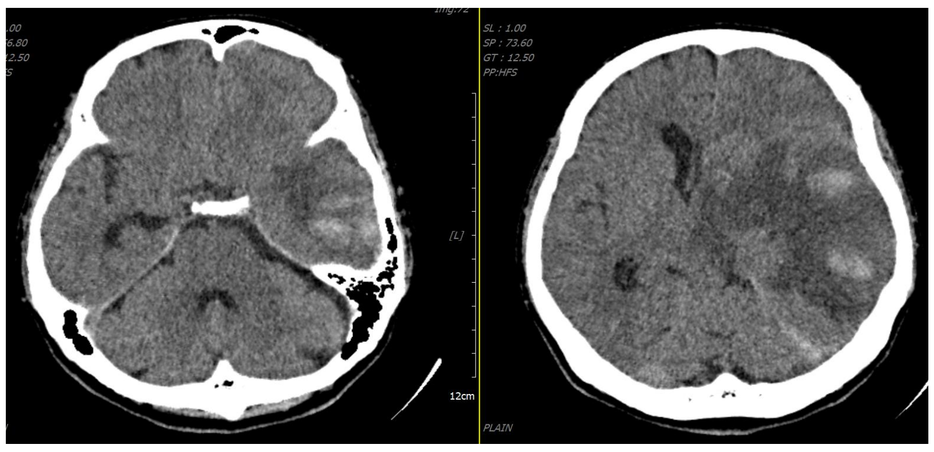

Figure 2: Resolving left temporal contusion bleed but with vasogenic oedema, exerting mass effect, at post trauma day 10.

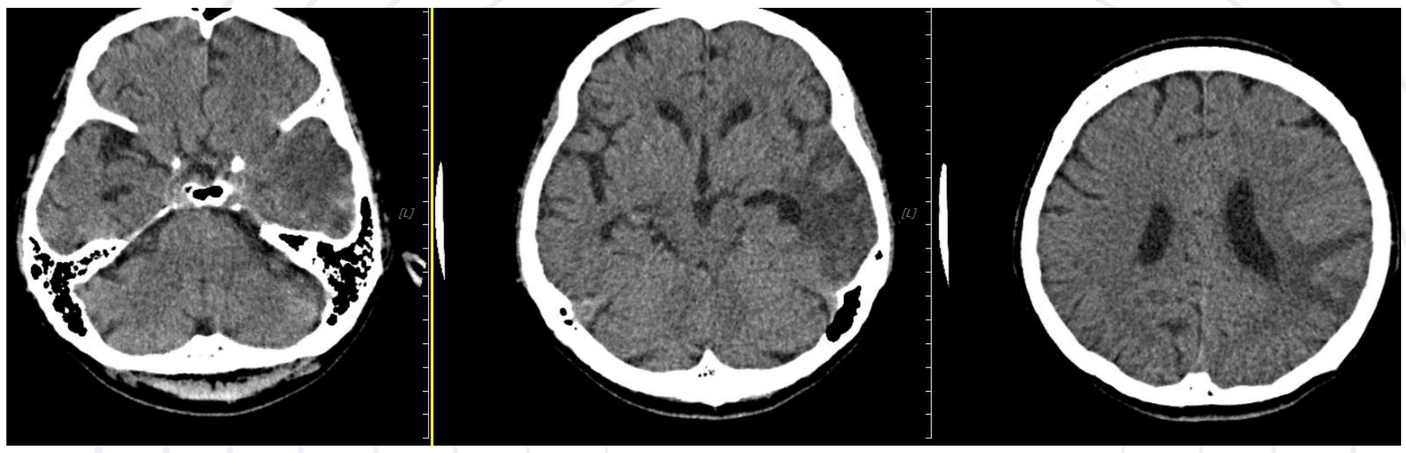

Figure 4: CT Brain one week after administration of dexamethasone showed improving oedema and resolved mass effect.

DISCUSSION

Cell death ensuing from primary TBI leads to blood-brain barrier disruption, thus inducing water influx across cerebral capillaries.[1] To date, most studies pertaining to the role of steroid in TBI involve high dose parenteral dexamethasone (50mg bolus) given in severe TBI during acute setting (mostly within 8 hours of trauma) and tapered intravenously over 5 days.[2] Only one study presented the efficacy of steroid in delayed post-traumatic cerebral oedema in mild to moderate TBI.[3]

CONCLUSION

Steroid could be beneficial in post-traumatic cerebral oedema in mild to moderate TBI. With such a renewed interest in steroids for delayed post-traumatic cerebral oedema, it is worth exploring further as it could bring about positive outcomes for patients. Future prospective trials are needed to confirm or refute these findings.

References:

1. Konar SK, Shukla D, Agrawal A. Posttraumatic brain edema: Pathophysiology, management, and current concept. Apollo Med 2019;16:2-7
2. Dearden NM, Gibson JS, McDowall DG et al. Effect of high-dose dexamethasone on outcome from severe head injury. J Neurosurg. 1986; 64(1):81-8
3. Prasad GL. Steroids for delayed cerebral edema after traumatic brain injury. Surg Neurol Int 2021;12:46.