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CONSERVATIVE MANAGEMENT IN EPIDURAL HEMORRHAGE IN KNOWN CASE OF THRMBOCYTOPENIC PURPURA: A CASE REPORT

Neurosurgery

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ABSTRACT

The severe and possibly lethal complication of immune thrombocytopenic purpura (ITP) is intracranial hemorrhage. Even though bleeding in the cranial cavity is rare in ITP, it is usually manifested as intracerebral or subarachnoid hemorrhage. Epidural hematoma is very uncommon in this kind of patients especially without trauma and its treatment is a therapeutic dilemma because of severe thrombocytopenia and the perceived urgency of immediate surgical evacuation. We describe the case of a 45-year-old woman with a known history of ITP since 2014 complained of the headache of 15 days. On examination, she was alert and oriented with the Glasgow Coma Scale of E4V5M6. Pupils were even and responsive bilaterally, focal neurological deficits were absent. Non-contrast computed tomography of the brain showed that there is a right parietal biconvex heterogeneous epidural collection of 3.3 cm in maximum thickness associated with a high mass effect with an 8.7 mm midline displacement to the left. Her platelet count was also critically low at 4,000/uL and hemoglobin 10 g/dL. The patient was treated conservatively in terms of high dosage of intravenous dexamethasone and close neurological and radiological observation despite the radiological severity. Serial imaging showed slow improvement in midline shift within the absence of hematoma enlargement. The case highlights that, under close monitoring and hematological care, and with a specific choice of patients with epidural hematoma and severe thrombocytopenia, the management of such patients can be effective without surgery.

KEYWORDS

Immune Thrombocytopenic Purpura; Epidural Hematoma; Intracranial Hemorrhage; Thrombocytopenia

INTRODUCTION

Immune Thrombocytopenic Purpura (ITP) is an acquired autoimmune disease, in which platelets are destroyed by immunopathy, leading to isolated thrombocytopenia. In adults, the incidence is estimated at 2-5 per 100,000 of the population annually.[1] Majority of patients present with symptoms of mucocutaneous bleeding, but the most severe and fatal complication is intracranial bleeding which occurs less than 1% but causes a significant number of deaths.[1,2] Subarachnoid and intracerebral bleedings are more frequently reported as intracranial bleeds, but the epidural hematoma (EDH) is extremely infrequent in patients with ITP.[2]

Acute EDH is normally linked with skull fractures and arterial bleeding and the modern neurosurgical practice suggests emergency surgery evacuation to patients who have large hematoma thickness, midline displacement, or dysfunctional decline.[3] This combination of critical thrombocytopenia also makes treatment choices difficult because in the case of operative treatment, there is a high risk of hemorrhage. As a result, the literature of success in conservative management in such situations is scarce.

Case Presentation

A 45-year-old female with a known history of ITP since 2014 with the complaints of the persistent headache over 15 days presented to our hospital. The headache was insidious in onset, progressive in the course, holocranial in the distribution, unrelated to vomiting, seizures, loss of consciousness, or history of recent trauma. She had been on intermittent oral corticosteroid and rituximab therapy for ITP in the last few years with intermittent follow-up. No hypertension history, diabetes mellitus or anticoagulant use was present.

At admission, the patient was oriented, alert, and conscious. She had a Glasgow Coma Scale (GCS) of E4V5M6. Vital signs were within the normal range, blood pressure was 126/78 mmHg, pulse rate was 84 per minute, and oxygen saturation was 98% in room air. Neurological exam showed bilaterally equal and reactive pupils, normal extraocular movements and no focal motor/sensory deficits. Papilledema was not observed under the fundoscopic examination, and there was no evidence of meningal irritation. Other systemic examination was not remarkable. On cutaneous examination, some petechial spots were present on the lower limbs.

Laboratory examinations showed that he was severely thrombocytopenic with a platelet count of 4,000/µL. Hemoglobin was 10 g/dl and total leucocyte count was 7,800/µL. The peripheral smear established a distinct case of marked thrombocytopenia and absence of clumping of platelets but presented normocytic normochromic red blood cells. There were normal prothrombin time, activated partial thromboplastin time and international normalized ratio. Liver and renal function tests were normal. Viral markers such as hepatitis B, hepatitis C and HIV were negative. Autoimmune workups, including antinuclear antibody positive, were not positive. The abdominal ultrasonography showed mild splenomegaly, which is in accordance with her known hematological condition.

A non contrast computed tomography (NCCT) of the brain revealed a right parietal biconvex heterogeneous extra-axial pool that is in line with an EDH (Figure 1). The lesion measured 3.3 cm at its maximum thickness and was predominantly hypodense with internal hyperdense areas suggestive of acute-on-chronic blood components. There was significant mass effect with compression of adjacent cortical structures and a midline shift of 8.7 mm toward the left side. No skull fracture was detected.

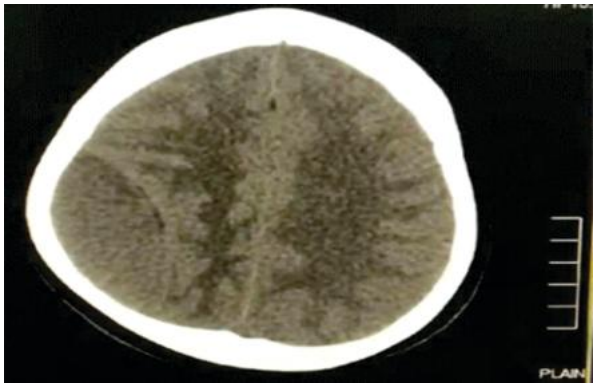

Figure 1: NCCT head of the patient demonstrating right parietal biconvex heterogeneous epidural collection of 3.3 cm in maximum thickness with a midline shift of 8.7 mm.

Given her stable neurological status despite significant radiological findings and severe thrombocytopenia, a multidisciplinary discussion involving neurosurgery and hematology teams was undertaken. Given the high risk of operations because of deep-rooted thrombocytopenia and the fact that there was no neurological degradation, a choice was made in favor of conservative management. High-dose intravenous dexamethasone 20 mg twice per day was initiated, platelet transfusion

and frequent neurological follow-up of the patient were carried out in the intensive care unit. Strict blood pressure regulation, head elevation and serial neurological examination were ensured. Repeat NCCT head of the patient on day 3 suggested stable lesion (Figure 2).

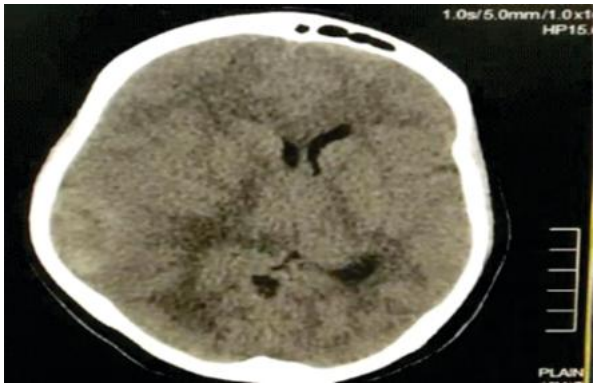

Figure 2: NCCT head of the patient on day 3 demonstrating right parietal biconvex heterogeneous epidural collection with a midline shift.

Her platelet counts kept on improving over the next few days to reach 28000/µL by day 4, and 65000/µL by day 7. Subsequent CT on day 14 revealed progressive decrease in the midline shift without enlargement in hematoma (Figure 3).

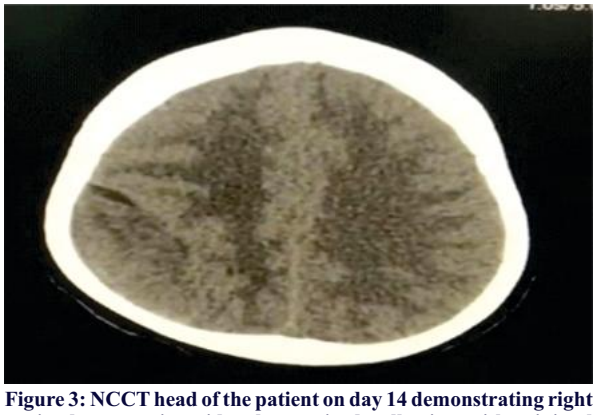

Figure 3: NCCT head of the patient on day 14 demonstrating right parietal crescentic epidural organized collection with minimal midline shift.

The patient was stable clinically, and exhibited neither focal impairments nor sensorium distortion. She was placed on tapering oral steroids and was discharged in a stable condition with recommendations of regular follow-ups on the hematology department. After 3 months follow-up, the patient had complete recovery and NCCT head revealed significant reduction in the EDH (Figure 4).

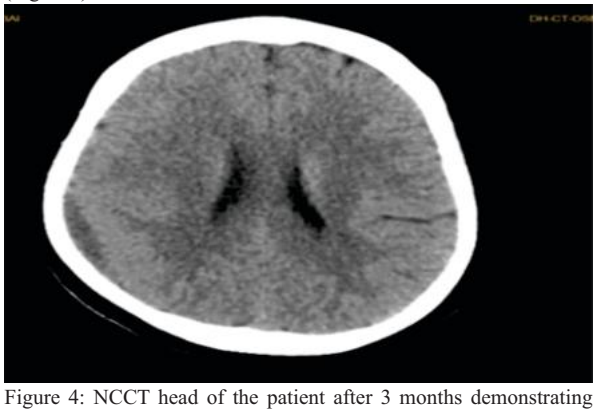

Figure 4: NCCT head of the patient after 3 months demonstrating minimal right sided parietal crescentic epidural collection with no midline shift.

DISCUSSION

ITP has been reported to cause almost all forms of bleeding complications in patients. Of all intracranial hemorrhages, the most frequent reported type of intracranial hemorrhage in ITP is intraparenchymal and subarachnoid hemorrhage.[4] Conversely, EDH is uncommon, especially without major traumas. Classically, EDH is caused by injury to the arteries, most of them being the middle meningeal artery, and is usually accompanied by skull fractures.[3] Spontaneous EDH in the conditions of thrombocytopenia is not well described, and its pathogenesis can be the rupture of the weak dural vessels, which is enhanced by the acme of platelet loss.

Standard neurosurgical practices suggest removing acute EDH surgically with a hematoma thickness that is greater than 15 mm or midline shift is higher than 5 mm irrespective of the GCS score.[3] Our patient exhibited a hematoma thickness of 3.3 cm and 8.7 mm midline shift which would normally cause immediate craniotomy. Nevertheless, clinical status is a determining factor. She had a GCS of 15 with no focal deficits, which implies that cerebral compensation remains intact despite radiological mass effect.

Major thrombocytopenia (<20,000/µL) was a major risk factor when it comes to increasing the risk of operative activity.[5] Severe ITP intervention surgery has a high risk of intraoperative bleeding that cannot be controlled and re-accumulation after the operation. Recent international consensus principles on ITP suggest an emergency treatment with high dose corticosteroids, intravenous immunoglobulin (IVIG) and platelet transfusion, where life threatening bleeding occurs.[5] Dexamethasone high dose has been shown to respond quickly to platelets during acute exacerbation and is viewed as a useful first-line treatment.[6] In this instance, intravenous dexamethasone was administered and platelet recovery and stabilization occurred with time.

One should note that not every EDH progresses in the same way. Conservative treatment of selected patients with small EDH and stable neurological examination can be used with intensive monitoring and serial imaging.[7] Although the majority of literature recommends the use of operative management in large EDH, case series have demonstrated neurological stability is a decisive outcome predictor.[7] Serial CT scans of our patient showed that the midline shift was reduced but hematoma was not increasing which supported the choice of non-operative intervention. Spontaneous intracranial hemorrhage has been reported previously in the case of ITP, however, the majority of the cases that have been reported necessitated surgical intervention after the neurological deterioration.[8] There is very limited literature that reports successful conservative management of EDH in severe thrombocytopenia. The case thus adds to the sparse literature that is expanding in regard to the use of individualized treatment strategies. Such complicated situations require multidisciplinary cooperation between neurosurgery and hematology. Intensive neurological follow-up, hematologic optimization, emergent surgical backup, and repeated neuroimaging are required in the case of conservative therapy.[9] The management should not just be determined by the radiological criteria without considering the clinical findings.

CONCLUSION

Spontaneous EDH in patients with ITP is highly uncommon and it presents a major management dilemma particularly where extreme thrombocytopenia is encountered. In spite of the fact that surgical evacuation is advised in most of the cases in the situation of large hematoma and midline shift, this case points to the fact that conservative treatment can also be in neurologically stable patients with close monitoring. The rapidity of platelet count correction, high monitoring, serial imaging of the brain and the multidisciplinary coordination are key factors in achieving good results. In such presentations which are complex and high-risk, individualized clinical assessment should be used as a basis of treatment as opposed to radiological parameters in isolation.

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