Case Report

INTRACRANIAL HAEMORRHAGE DUE TO MINOR TRAUMA IN A PATIENT WITH IDIOPATHIC THROMBOCYTOPENIC PURPURA - A CASE REPORT.

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Abstract

Introduction: Intracranial haemorrhage (ICH) is a rare but the most dangerous complication of idiopathic thrombocytopenic purpura (ITP) which is usually fatal. ITP is caused by autoantibodies to platelet which can be demonstrated in plasma.

Case-Report: A 32 years old male patient sustained minor trauma around his head, following which he developed features of raised intracranial pressure including headache, vomiting and loss of consciousness. On examination Glasgow Coma Scale was 7/15 (E2, M3, V2), left pupil was moderately dilated, there was bilateral papilloedema and haemorrhagic spots on the left sided retina. CT scan of the head showed significant intracranial haemorrhage. Laboratory investigation showed anaemia and thrombocytopenia with platelet count 40X10^9/L. All the relevant causes of thrombocytopenia were excluded and the diagnosis of ITP was established. Intracranial haemorrhage was managed conservatively without surgical intervention. ITP was managed with splenectomy on 12th day following a period of therapy with steroids, transfusion of packed cell and platelet concentrates.

Key-Words: Intracranial haemorrhage, Idiopathic thrombocytopenic purpura, Papilloedema.

Introduction

Although relatively uncommon, intracranial haemorrhage (ICH) is the most serious complication of Idiopathic thrombocytopenic purpura (ITP) and is the leading cause of death. Fortunately it occurs in less than 1% of all cases of ITP and account for 30%-100% of fatalities in reported series[1].

ITP is caused by autoantibodies to platelet which can be demonstrated in plasma. But the tests for platelet autoantibodies are not widely available. Therefore diagnosis is one of the exclusion and can be made only after other causes of thrombocytopenia have been ruled out[2].

We report the occurrence of intracranial haemorrhage due to minor trauma in a previously healthy adult patient who has been diagnosed as a case of ITP. Neurological condition was managed by conservative therapy, but splenectomy had to be done for ITP and a successful outcome was made.

Case Report

A 32 years old black male patient was admitted at UN Hospital, Sudan with 05 days history of headache, vomiting and loss of orientation following a minor trauma. He got involved in a domestic chaos 05 days back when received a few ordinary hand blows over his head and face. No past history of gum bleeding, tarry stool and easy bruising. No history of recent infection, fever, rash, arthralgia or blood transfusion.

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Examination revealed a remarkably drowsy patient, afebrile with bradycardia (pulse rate-48/minute) and slightly elevated blood pressure (BP-145/95 mm of Hg).

There was no skin rash, ecchymosis, diffuse purpura or subconjunctival haemorrhage. Neurological examination revealed that his Glasgow Coma Scale was low (E2, M3, V2), left pupil was moderately dilated. Fundoscopy revealed bilateral papilloedema and haemorrhagic spots on the left retina. There was no neck rigidity and planter response was extensor. Laboratory investigations revealed a haemoglobin of 9 gm/dl,white blood cell count of 10x109 with a normal differential count, platelet count 40X109/L and reticulocyte count was 2.8%. Prothrombin time, partial thromboplastin time, thrombin time, fibrin and fibrin degradation product (FDP) were all normal.

Electrocardiogram, chest radiograph and skull radiology were normal. CT scan of head showed significant intracranial haemorrhage involving both extradural and subdural space over the left cerebral convexity causing partial effacement of left lateral ventricles and midline shift.

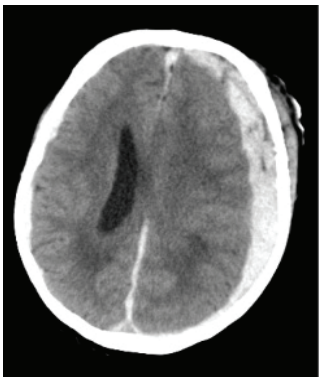

Fig-1: Photograph of CT scan (axial cut) head of the patient.

Bone marrow study revealed increased megakaryocytes, but no other abnormality. Antibody for Dengue was found negative by ICT method. Malaria was also found negative in blood film and by ICT method. HIV status was negative. Platelet antibody level was not performed as it was not available. By exclusion of other causes of thrombocytopenia, diagnosis of ITP was established.

Considering potential threat of unmanageable haemorrhage at operation, a conservative medical therapy was initiated. Treatment was started with intravenous dexamethasone 10 mg every 06 hours and 20% mannitol- 1 mg/kg to reduce cerebral oedema and intracranial pressure and phenytoin to prevent seizure. He was transfused 04 units of platelets and 02 units of packed red blood cells. Treatment was continued with dexamethasone 05 mg 06 hourly and phenytoin 300 mg daily along other symptomatic treatment. Neurological condition and platelet count improved gradually. Elective splenectomy was done on 12th day, when the platelet count was $100x10^9$/L. Surgery to evacuate the intracranial haematoma was not done as the neurological condition improved significantly with no deficit and follow up CT scan showing regression of the size of haematoma. Three weeks postoperatively his platelet count reached $180X10^9$/L and 06 weeks after discharge it was $350x10^9$/L. Follow up was continued for 09 months and platelet count and neurological examination have remained normal without any bleeding episodes.

Discussion

Intracranial haemorrhage due to ITP can occur spontaneously or with minor trauma. In our case, haemorrhage occurred due to a trauma which cannot be actuallly accounted as a severe one Idiopathic thrombocytopenic purpura may be acute or chronic. Acute ITP is mainly a disease of childhood occurring in equal frequency in both sexes and chronic ITP occurs most commonly in adult typically under the age of 40 with female to male ratio of 3:1 as observed worldwide[3]. Often there is a history of easy bruises, nose bleeding, bleeding from gum and haemorrhage with relatively minor trauma. The disease may manifest first with malaena, haematuria or menorrhagia and sometimes more terribly with intracranial haemorrhage[4]. Size of haemorrhage varies from a single petechae to large extravasations. ICH[5] are mostly found in subarachnoid areas but it can be in any of the intracranial spaces.

Posterior fossa haemorrhage is especially dangerous due to possibility of rapid cerebellar herniation and brain steam compression. Retinal haemorrhages often occur at the time of ICH[5]. In our case, there was haemorrhage at extradural and subdural area with retinal haemorrhage ipsilaterally.

A platelet count below 100000/ml is generally considered to constitute thrombocytopenia. However spontaneous bleeding does not become evident until platelet count falls to 20000/ml. Platelet count ranges of 20000 to 50000l/ml can aggravate post traumatic bleeding[2].

If a patient with ITP develops sign or symptoms of intracranial bleeding such as headache, vomiting, convulsion, meningism, personality changes, retinal haemorrhage or focal neurological signs, rapid and vigorous treatment must be initiated as this can be fatal.

Therapy for ICH associated with ITP consist of controlling the elevated ICP while also achieving a rapid rise in the platelet count to control the bleeding. Traditional method of head elevation, fluid restriction, avoidance of hypercapnia, intravenous dexamethasone and 20% manitol can be implemented to control cerebral oedema[6].If the level of consciousness deteriorates or neurological deficit aggravates and the haemorrhage is in an accessible location, neurosurgical intervention may be warranted. But the decision to operate has to be taken considering the risk and benefit of the individual case. Emergency splenectomy followed by platelet concentrates is employed in cases of ITP refractory to conservative therapy or if life threatening haemorrhage occur[7]. It has been stressed that splenectomy should be done prior to craniotomy to avoid further bleeding during surgery although both procedure may be done together under one anesthesia[8].

Conclusion

As intracranial haemorrhage is the most dreaded complication of ITP, it is essential to proceed on early diagnosis, prompt and aggressive management.

Again to adopt an invasive procedure in a platelet depleted patient with huge potential for haemorrhage depends on crucial clinical judgment. Neurosurgical intervention, if considered necessary, should be performed before the patient enters into an irreversible stage.

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