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Drug-Induced Bone Marrow Aplasia Revealed by Severe Hemorrhagic Stroke after Taking Amiodarone: A Case Report

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Abstract

Aplastic anemia is defined by the disappearance or rarefaction of the normal hematopoietic tissue of the marrow leading to quantitative bone marrow failure; the diagnosis of aplastic anemia is based on clinic biological criteria; multiple etiologies may be involved, in particular drug causes, in particular amiodarone. Mrs. SZ, 45 years old; follow-up for VT on ischemic heart disease put on amiodarone for 6 months. Symptoms dated back 2 days before admission with the onset of disturbance of consciousness and generalized tonic-clonic seizures. On admission neurologically: The patient had 9/15 GCS, pupils in tight miosis, no deficit, no convulsion; with a stable hemodynamic and respiratory state The remainder of the examination showed gingivorrhagia with bruising of the lower limbs. Complementary examinations had found: An aspect of a parafalcorial hemorrhagic stroke complicated by major tri ventricular hydrocephalus with ventricular flooding on the CT scan. With pancytopenia: Hb: 6.4 g / dl, GB 890e; PNN 30e; Lc: 780e; Pq: 4000e, absence of Blasts, a myelogram and a BOM showed a desert marrow with cytogenetic and molecular study, the rest of the work-up ruled out the other etiologies of AM and the drug etiology was retained. Treatment was mainly symptomatic, administration of tranexamic acid, transfusion of 2 GC of 5 CPQ; management of ACSOS; and discontinuation of the drug. The course was marked by the regression of pancytopenia. The occurrence of aplasia under amiodarone is a fairly rare occurrence. According to the recommendations, the severity criteria are essentially biological; the severity of the aplastic anemia in this patient is determined by the 2 criteria present: PNN less than 500th and the platelets less than 20,000th; the severity of this case is greater given the severe added stroke. Treatment in severe aplasia consists of the administration of immune suppressants (cyclosporine, high doses of corticosteroid, anti-lymphocytic serum) with 40% remission Or a hematopoietic stem cell transplant with 70% remission.

Keywords: Aplastic anemia; Amiodarone; Hemorrage; Stroke

Introduction

Amiodarone is a class III anti-arrhythmic drug that is highly effective in treating both atrial and ventricular arrhythmias [1]. However, it is associated with a variety of side effects that limit its clinical application including thyroid dysfunction, pulmonary fibrosis, optic neuritis, ataxia, and hepatitis [2-4], bone marrow granulomas, pancytopenia, hemolytic anemia, neutropenia, and thrombocytopenia [5-9]. Bone marrow aplasia is defined by the disappearance or rarefaction (hypoplasia) of the normal hematopoietic tissue of the marrow leading to quantitative bone marrow insufficiency (pan cytopenia); the diagnosis of bone marrow aplasia is based on clinical and biological criteria; multiple etiologies can be involved, in particular drug causes. The authors propose to report a new observation of bone marrow aplasia following an intake of amiodarone discovered by the occurrence of a hemorrhagic stroke.

Case Study

Mrs. SZ, 45 year old; having a history of repeated ventricular tachycardia on ischemic heart disease put on oral amiodarone and aspirine for 6 months with a normal blood counts before amiodarone therapy. The case presentation dated back to 2 days before her admission by the occurrence of disturbance of consciousness associated with generalized tonic-clonic convulsive seizures. On her admission neurological examination showed a patient at 9/15 of GCS, pupils in miosis tight with no deficit or convulsion; hemodynamically her BP: 150/70 mmHg, HR: 78 bpm, no paleness, no signs of peripheral hypo perfusion with normal cardiac auscultation; on the respiratory exam RR: 18 c/m, normal pleuropulmonary auscultation, no signs of struggle, no cyanosis; SpO2: 96%. Capillary blood sugar: 1 g/l T°: 37. The remainder of the examination showed gingivorrhagia with bruising of the lower limbs. The examination revealed the notion of taking 8 amiodarone tablets within 48 hours of admission. The additional examinations had found an aspect in favor of a parafalcorial hemorrhagic stroke complicated by major triventricular hydrocephalus with ventricular flooding on the brain scan. The biological assessment showed hemoglobin at 6.4 g/dl; MCV at 82fl; CCMH at 36.4 g/dl; white blood cells : 890 elements; neutrophiles : 30 elements; lymphocytes: 780 elements; platelets: 4000 elements; Absence of blasts, the hemostasis workup was normal, the ECG and chest x-ray were without abnormalities, a myelogram and a bone marrow biopsy with cytogenetic and molecular study were carried out and showed a deserted marrow, serum ferritin, serum iron, folic acid, and vitamin B12 were all detected at normal levels. The serum bilirubin was normal, and the urobilinogen and hemolytic tests were all negative. The rest of the work-up ruled out the other etiologies of aplastic anemia and the drug etiology was retained.

After initial conditioning, therapeutic management consisted of mainly symptomatic treatment (oxygen therapy, 30 degree position), administration of 1g tranexamic acid ; transfusions of 3 blood bags and 5 platelet culots; management of ACSOS; and discontinuation of the drug involved, including amiodarone. The patient was put on prednisolone with a beginning of regression of the pancytopenia but the patient's course was marked by worsening his neurological condition and death (Figure 1).

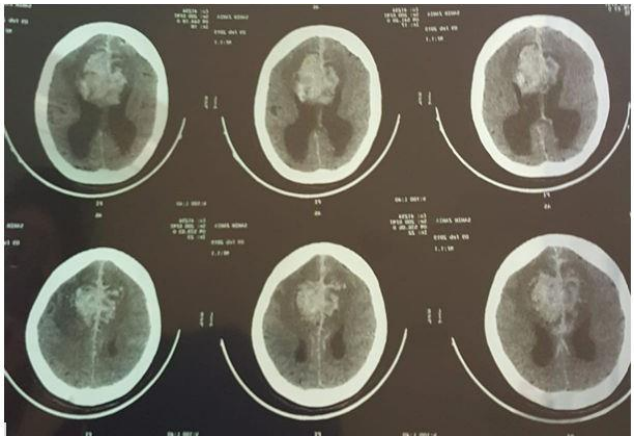

Figure 1: CT scan showing the massive hemorrhagic stroke.

Discussion

Amiodarone-related blood system damage is reported more often but reports of bone marrow aplasia are rare. Our patient had blood cells disorder before taking amiodarone whiles his hospitalization for the ventricular tachycardia. After the use of amiodaron anemia, neuropenia and thrombocytopenia were occurred, reticular red blood cells were reduced, and bone marrow examination concluded to a bone marrow aplasia, because of the very low platelets the patient had a severe hemorrhagic stroke causing his death. Major factors that lead to amiodarone-related side effects include long-term high-dose usage and low body weight [10]. This patient had an ischemic heart disease taking aspirin, low weight, and long-term usage of the drug due to irregular follow-up. Therefore, the use of amiodarone in low-weight adults, especially for long periods, should be carefully monitored.

According to the recommendations, the severity criteria of bone marrow aplasia are essentially biological. The severity of the aplastic anemia in this patient is determined by the 2 criteria present: PNN less than 500 elements and platelets less than 20,000 elements; the severity of this case is major given the added stroke.

The treatment for severe aplasia consists of the administration of immune-suppressants (cyclosporine, high-dose corticosteroid, anti-lymphocyte serum) with 40% remission or a haematopoietic stem cell transplant with 70% remission (Figure 2).

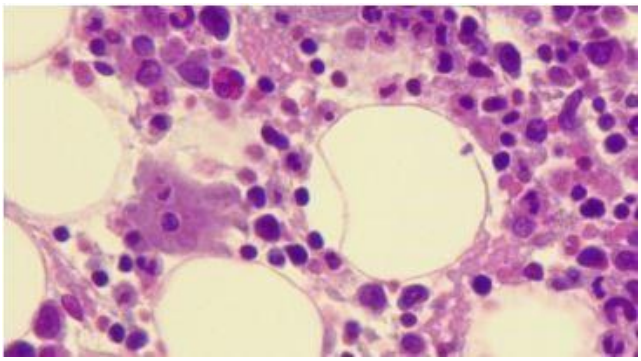

Figure 2: The deserted bone marrow aplasia caused by amiodarone.

Conclusion

Bone marrow aplasia (other than after chemo or radiotherapy) has multiple etiologies; mainly drug causes are found, hence the interest of careful examination. The diagnosis passes through the clinic represented by a medullar insufficiency syndrome without tumor syndrome, then by the biology represented by a pancytopenia, the myelogram is poor made mostly of lymphocytes and confirmation is by bone marrow biopsy which shows empty stalls with desert marrow. The drug in question must be stopped immediately and treatment is symptomatic as well as curative with the hope of a cure with the transplant of hematopoietic stem cells.

References

1. Vamos M, Hohnloser SH (2016) Amiodarone and dronedarone: An update. Trends Cardiovasc Med 26: 597-602.
2. Rickard JP, Negrelli J, Olson JL, Dick T (2018) Frequency of adverse event monitoring in ambulatory patients on amiodarone or dofetilide. J Pharm Pract 31: 457-461.
3. Rankin S, Elder DH, Ogston S, George J, Lang CC, et al. (2017) Population-level incidence and monitoring of adverse drug reactions with long-term amiodarone therapy. Cardiovasc Ther 35: e12258.
4. Rehman SU, Siddiqui N, Khan NS, Sobia R, Assaly R (2015) Multisystem side effects of amiodarone. Am J Med Sci 349: 454.
5. Erie AJ, McClure RF, Wolanskyj AP (2010) Amiodarone-induced bone marrow granulomas: An unusual cause of reversible pancytopenia. Hematol Rep 2: e6.
6. Arpin MP, Alt M, Kheiralla JC, Chabrier G, Welsch M, et al. (1991) Hyperthyroidism and immune hemolytic anemia following amiodarone therapy. Rev Med Interne 12: 309-311.
7. Groneberg DA, Barkhuizen A (2001) Neutropenia during treatment with amiodarone. Am J Med. 110: 671.
8. Sahud MA, Caulfield M, Clarke N, Koch R, Bougie D, et al. (2013) Acute thrombocytopenia in patients treated with amiodarone is caused by antibodies specific for platelet membrane glycoproteins. Br J Haematol. 163: 260-267.
9. Lossos IS, Matzner Y (1992) Aplastic anemia associated with amiodarone therapy. Acta Haematol 87: 213.
10. Yamada Y, Shiga T, Matsuda N, Hagiwara N, Kasanuki H (2007) Incidence and predictors of pulmonary toxicity in Japanese patients receiving low-dose amiodarone. Circ J 71: 1610-1616.