Case Report

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Multi-infarct stroke secondary to reactive thrombocytosis in a patient with iron deficiency anaemia: A case report

Preethy Manoj*; Rosemarie Derwin; Pinar Avsar; Eamon Dolan; Zena Moore; Declan Patton; Tom O'Connor; Linda Nugent; Sherly George

*Corresponding Author: Preethy Manoj

School of Nursing and Midwifery, Royal College of Surgeons in Ireland, St Stephens Green, St Peters, Dublin 2, Ireland.

Tel: 0877829312; Email: preethymanoj@rcsi.ie

Abstract

A 47-year-old woman who presented to the hospital with left sided weakness, left sided paraesthesia, ataxic gait, headache, nausea and vomiting was found to have multiple infarct strokes. Her past medical history was significant for iron deficiency anaemia secondary to menorrhagia. Her haemoglobin level on arrival at the hospital was 7.3 gm/dl. Her symptoms improved after blood transfusions and iron infusion. She had low cardiovascular risk factors and her stroke work up was negative. So, it was assumed that her stroke was likely related to reactive thrombocytosis secondary to iron deficiency anaemia.

Keywords: Iron deficiency anaemia; Ischaemic stroke; Thrombocytosis; IDA.

Introduction

Iron deficiency anaemia (IDA) is an under recognised risk factor for acute ischaemic stroke [1]. In an observational study conducted in Pakistan, it was identified that 10.42% of the patients diagnosed with acute ischaemic stroke had IDA [2]. Scherbakov et al. (2022) identified that iron deficiency was associated with lower muscle strength in patients with acute ischaemic stroke [3]. Rathore et al. (2021) reported that every fifth patient with acute ischaemic stroke has a low haemoglobin level and is associated with worse neurological end results [4]. In this case, we describe a 47-year-old female with IDA who suffered multiple infarct strokes. This article is presented in accordance with the CARE reporting checklist [Appendix 1].

Case Presentation

A 47-year-old female presented to the emergency department (ED) with complaints of sudden onset of headache associated with nausea and vomiting, weakness to left upper and lower limbs, ataxic

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gait and left sided paraesthesia. The patient had a background of migraine, cholecystectomy (in 2000), uterine fibroid (noted on pelvic ultrasound in 2017) and longstanding history of menorrhagia over ten years leading to iron deficiency anaemia (IDA). The patient was dependent on iron infusions. The patient was a non-smoker, reported occasional use of alcohol. Her diet was mostly vegetarian and occasional meat intake once in eight to ten days. She lived with her husband and children, independent with activities of daily living at baseline. Her last menstrual period (LMP) was a week before the admission. Her admission weight measured 64.2-kilogram, height 1.62 metres and body mass index (BMI) of 24.5. Her Glasgow Coma Scale (GCS) was 15/15 on arrival to ED. She was haemodynamically stable on admission. An electrocardiogram (ECG) demonstrated normal sinus rhythm with first-degree atrioventricular (AV) block. No abnormality was identified on her chest X-ray. From a neurological standpoint, her cranial nerves were grossly intact. Tone and refluxes were normal in both upper and lower limbs. Power 5/5 in her right arm and right leg, 3/5 power in her left arm and 4/5 power in her left leg. Coordination was normal in her right arm and right leg , but impaired in in her left arm and left leg. The sensation was normal in her right arm and right leg, with decreased sensation in left arm and left leg. Planters were inconclusive in both right and left leg. Impaired proprioception was noted in her left upper limb. Shoulder adduction 5/5 on the right side, 4/5 on the left side, shoulder abduction 5/5 on right side, 4/5 on left side. Hip adduction 5/5 on right, 4/5 on left, hip abduction 5/5 on right, 4/5 on left, hip flexion 5/5 on right, 3-4/5 on left, hip extension 5/5 on right, 4/5 on left, knee flexion 5/5 on right, 4/5 on left. Neurological examination was repeated after one hour. Sensation improved on left side, close to baseline according to the patient. Power improved from range of motion (ROM) examination close to baseline as reported by the patient. Visual field examination was normal, coordination was normal in upper and lower limbs, and no ataxia noted on repeated neurological examination.

No cranial abnormality was reported in the computed tomography scan (CT scan) of the brain. Magnetic Resonance Imaging (MRI) of the brain identified multifocal infarcts involving the right frontal, parietal, occipital and temporal region. The patient received a loading dose of aspirin and Plavix.

Her blood film reported microcytic hypochromic red cells, anisocytosis ++, thrombocytosis and polychromasia. Her haemoglobin on arrival to ED was 7.3 g/dl. It dropped to 6.4 gm/dl the following day. Her ferritin level was 7 ng/ml, and her iron level was only 3.4 umol/L. Patient received two units of blood transfusion and one-gram intravenous iron infusion (ferrinject) during her stay in the hospital and was commenced on oral iron supplement (galfer) and folic acid. She was reviewed by the haematologist who suggested ongoing intravenous iron replacement due to previous poor tolerance to oral iron supplements. The patient's blood report during hospitalization is given in table 1.

Her echocardiogram, carotid doppler and bubble study were negative. Her glycosylated haemoglobin (HBA1C) was within normal parameters. An ultrasound scan of the abdomen to rule out splenomegaly showed a normal spleen. Patient received multi-disciplinary team input as a hospital inpatient. The patient reported she had noticed decreased memory one month prior to the admission. Montreal Cognitive Assessment (MoCA) scored 25/30 indicating mild cognitive impairment. The patient showed mild deficits in areas of attention and visuospatial ability/executive function. Memory 5/5. The patient felt reaction times in both function and cognition are reduced slightly compared to preadmission. The patient's upper limb

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function improved and had no concerns about completing functional tasks. She reported mild decreased sensation, but she felt that this is not impacting her activities of daily living. The patient was discharged from the hospital on day 8 with no significant residual deficits. Her haemoglobin on the day of discharge was 10.5 gm/dl. As no other reasons were identified for her multifocal infarct, we conclude that reactive thrombocytosis due to IDA secondary to menorrhagia was the cause of her stroke.

Table 1: Blood reports.							
	Day 0 of admission	Day 1 of admission	Day 2 of admission	Day 3 of admission	Day 6 of admission	Day 7 of admission	On discharge (Day 8)
Haemoglobin (Hb gm/dl)	7.3	6.4		9.3	10.7	10.6	10.5
Packed cell volume (PCV l/L)	0.242	0.214		0.291	0.344	0.330	0.330
Mean corpuscular Volume (MCV fl)	59.7	59.4		66.6	67.8	68.3	67.4
Mean Corpuscular Haemoglobin (MCH pg)	18.0	17.8		21.4	21.1	21.9	21.5
Platelet x 10 ⁹ /L	1170	1043		885	725	561	465
Ferritin (fer) ng/ml			7				
Iron (IR) umol/L			3.4				
Unsaturated iron binding capacity (UIBC) umol/L			79.7				
Folic acid ug/L			7.3				
Vitamin B12			777				
Glycosylated Haemoglobin (HBA1C)			35				

Discussion

As per the world health organisation (WHO) report, the most common nutritional deficiency in the world is IDA, affecting 30% of the population [5]. It is estimated that one third of the women in their reproductive age suffer from heavy menstrual bleeding leading to iron deficiency [6]. A woman with heavy menstrual bleeding loses five to six times more iron during each menstrual cycle than a woman with normal blood loss [6]. In the case presented here, menorrhagia was identified as the cause of IDA. At present, there are no proven justifications of why IDA causes stroke. However, a significant association between IDA and thrombocytosis was reported by Mhadgut et al. (2018) [7]. Even though there are many proven risk factors for stroke such as diabetes, hypertension, dyslipidaemia etc, the aetiology of stroke remains undetermined in 30% of cases, especially in young adults [8].

Recently, IDA has been outlined as a risk factor for stroke [9,10]. There are three physiological mechanisms that explain the potential link between IDA and stroke: thrombocytosis, hypercoagulable state, and hypoxia [11]. Low iron levels cause disinhibition of megakaryocyte activity thus resulting in a secondary thrombocytosis and a hypercoagulable state [12]. Microcytic red blood cells have altered deformability, which increases the blood viscosity and may increase the risk of venous thrombosis [13]. The decreased oxygen carrying capacity of the erythrocytes results in anaemic hypoxia [12]. To compensate for the lack of oxygen in the blood, an increased blood flow to the brain is needed in patients with anaemia. This increased blood flow can cause vascular endothelial damage, resulting in thrombus formation [12]. There are some case reports that indicate a potential link between IDA and ischaemic stroke [9,12,14,15,16].

An Asian study conducted by Hassan et al. (2022) in Bangladesh reported a significant prevalence of IDA in acute ischaemic stroke and a significant association between IDA and severity of ischaemic stroke [8]. A substantial correlation between IDA and ischaemic stroke was identified in the studies of Chang et al. (2013), Joshi & Agrawal (2016), and Dubyk et al. (2012) [11,12,17].

Conclusion

Taking into consideration this case, previous studies, and case reports on IDA and ischaemic stroke, we suppose IDA should be considered as a possible risk factor for ischaemic stroke. Early detection and timely management of IDA is very essential to reduce the incidence of ischaemic stroke. More studies with large sample sizes are needed to confirm a definite relationship between IDA and ischaemic stroke.

Declarations

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:

Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work.

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Authors Information: Preethy Manoj^{1,2}; Rosemarie Derwin¹; Pinar Avsar¹; Eamon Dolan^{1,2}; Zena Moore^{1,3,4,5,6,7}; Declan Patton^{1,9,10}; Tom O'Connor^{1,8,9}; Linda Nugent¹; Sherly George¹

¹School of Nursing and Midwifery ,Royal College of Surgeons in Ireland, St Stephens Green, St Peters, Dublin 2, Ireland. ²Connolly Hospital, Blanchardstown, Dublin 15, Ireland.

³Adjunct Professor, School of Nursing & Midwifery, Griffith University, Queensland, Australia.

⁴Visiting Professor, School of Health Sciences, Faculty of Life and Health Sciences Ulster University, Northern Ireland. ⁵Honorary Visiting Professor, Cardiff University, Cardiff, Wales.

⁶Adjunct Professor, Department of Nursing, Fakeeh College for Medical Sciences, Jeddah, KSA.

⁷Professor, Department of Public Health, Faculty of Medicine and Health Sciences, Ghent University, Belgium.

⁸Honorary Professor, Lida Institute, Shanghai, China.

⁹Adjunct Associate Professor, Fakeeh College of Health Sciences, Jeddah, Saudi Arabia.

¹⁰Honorary Senior Fellow, Faculty of Science, Medicine and Health, University of Wollongong, Australia.

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