CASE REPORT

Melanoderma: uncommon presentation of a common condition

Priyadarshini Gunaseelan,¹ Sarveswari Narasingarajan,² Shriraam Mahadevan^{3,4}

¹Department of Family Medicine, Sundaram Medical Foundation Dr Rangarajan Memorial Hospital, Chennai, Tamil Nadu, India

²Sundaram Medical Foundation Dr Rangarajan Memorial Hospital, Chennai, Tamil Nadu, India

³Department of Endocrinology, Sundaram Medical Foundation Dr Rangarajan Memorial Hospital, Chennai, Tamil Nadu, India

⁴Department of Endocrinology, Endocrine & Specialty Clinic, Chennai, Tamil Nadu, India

Correspondence to

Dr Shriraam Mahadevan, mshriraam@gmail.com

Accepted 6 December 2016

SUMMARY

A 64-year-old man presented with blackish pigmentation of skin and tongue for more than 2 years that did not improve with local remedies. He had no history of comorbid medical or surgical illnesses. General examination showed severe hyperpigmentation of skin and mucosa and also showed anaemia. Systemic examination was unremarkable except for Romberg sign and mild hyperreflexia. Adrenal insufficiency was ruled out by normal cortisol levels and haemogram showed macrocytosis with hypersegmented neutrophils suggestive of severe vitamin B12 deficiency which confirmed by low serum B12 levels. After 3 months of parenteral vitamin B12 treatment, the pigmentation and anaemia improved significantly. The mechanism of hyperpigmentation due to B12 deficiency is postulated to be increased melanin deposition in the basal layer of the epidermis. Since vitamin B12 deficiency is not uncommonly encountered in general practice, atypical presentation of this easily diagnosable and treatable condition is emphasised.

BACKGROUND

Severe vitamin B12 deficiency is not infrequently encountered in clinical practice. Atypical manifestation of vitamin B12 deficiency is not rare as in our patient with severe hyperpigmentation. This case is discussed to remind physicians to think of this otherwise easily diagnosable and treatable problem whenever they encounter pigmentation and anaemia before going in for further investigations and complex treatments.

CASE PRESENTATION

A 64-year-old man on mixed diet presented with 2 years history of blackish discoloration of face, palms and tongue (figure 1A, B). Except for mild joint stiffness there was no history of any other significant comorbid medical or surgical illnesses. He had tried alternative methods of treatment (Ayurveda) for the same symptom for few months a year ago and stopped as there was no improvement. On examination he had conjunctival pallor and cutaneous examination showed blackish discoloration of palms, legs up to the level of ankle, tongue and diffuse hyperpigmentation of the face (figure 1A, B). Systemic examination was unremarkable except for Romberg sign and mild hyperreflexia.

INVESTIGATIONS

His fasting glucose level was 88 mg/dL with normal renal, liver functions and electrolytes. Haematological profile revealed anaemia (Hb 8.7 gm/dL) with mild

Figure 1 (A) Pretreatment picture of hands showing severe blackish pigmentation. (B) Pretreatment picture of the tongue showing blackish pigmentation.

thrombocytopenia and peripheral smear showed significant macrocytosis with hypersegmented neutrophils. Serum cortisol at 08:00 was 15.61 µg/dL and adrenocorticotrophic hormone (ACTH) level of 10.58 pg/mL ruling out severe cortisol deficiency. Free thyroxine (FT4) was 0.8 ng/dL (N=0.80–1.8) and thyroid stimulating hormone was 3.07 mIU/L (N=0.35–5.5) ruling out thyroid

CrossMark

To cite: Gunaseelan P, Narasingarajan S, Mahadevan S. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-217766

Figure 2 (A) Complete resolution of pigmentation of hands after treatment with vitamin B12 for 3 months. (B) Resolution of tongue pigmentation after treatment with vitamin B12 for 3 months.

dysfunction. Serum vitamin B12 level was <50 pg/mL (N=180–914 pg/mL) confirming severe vitamin B12 deficiency. Anti-tissue transglutaminase and anti-parietal cell antibody titres were normal.

DIFFERENTIAL DIAGNOSIS

Addison disease
Drug-induced hyperpigmentation
Thyrotoxicosis
Post inflammatory hyperpigmentation

TREATMENT

Parenteral vitamin B12 1000 mg once a week for 4 weeks followed by once a month regularly.

OUTCOME AND FOLLOW-UP

The skin colour of the patient nearly normalised to his baseline colour over the next 3 months on parenteral vitamin B12

treatment (figure 2A, B). Overall well-being, anaemia and the neurological symptoms, including ataxia, improved almost completely.

DISCUSSION

With the above clinical presentation, we initially considered primary adrenocortical insufficiency (Addison disease) either as part of polyglandular involvement or isolated which was ruled out by normal basal cortisol and ACTH levels. With Romberg sign being significantly positive, mildly exaggerated deep tendon reflexes and peripheral smear showing macrocytosis and hyper-segmented polymorphs we strongly considered vitamin B12 deficiency.¹ This was confirmed by very low serum vitamin B12 levels as well as by significant improvement in the hyperpigmentation on follow-up (figure 2A, B). Although vitamin B12 deficiency is usually reported to be common in vegans,^{1 2} our index patient was taking regular non-vegetarian diet. We considered coeliac disease (though not common in predominantly rice eating people of South India) and pernicious anaemia which was negated by normal titres of anti-tissue transglutaminase and parietal cell antibodies. We treated the patient with parenteral vitamin B12 (mecobalamin 1000 mg once a week for 4 weeks followed by once a month) in the lines of pernicious anaemia along with oral folic acid and other multivitamins. The patient showed dramatic improvement in pigmentation and anaemia as well as overall well-being over the next few weeks.

There are few reports hyperpigmentation with vitamin B12 deficiency in the literature.^{3 4} The likely explanations postulated for increased pigmentation in vitamin B12 deficiency include increased melanin synthesis and deposition in the basal layer of the epidermis rather than a defect in the melanin transport or melanocyte dysfunction.^{3–5}

Learning points

- ▶ Vitamin B12 deficiency may cause significant hyperpigmentation.
- ▶ This should be considered in any patient with pigmentation disorders.
- ▶ Treatment of vitamin B12 deficiency is simple and very rewarding.

Contributors PG was involved in the management of the patient, review of literature, manuscript preparation and review. SN was involved in diagnosis, management of the patient and manuscript review. SM was involved in diagnosis and management of the patient, manuscript preparation and literature review.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Stabler SP. Clinical practice. Vitamin B12 deficiency. *N Engl J Med* 2013;368:149–60.
- 2 Brescoll J, Daveluy S. A review of vitamin B12 in dermatology. *Am J Clin Dermatol* 2015;16:27–33.
- 3 Agrawala RK, Sahoo SK, Choudhury AK, et al. Pigmentation in vitamin B12 deficiency masquerading Addison's pigmentation: a rare presentation. *Indian J Endocrinol Metab* 2013;17:S254–6.
- 4 Kannan R, Ng MJ. Cutaneous lesions and vitamin B12 deficiency: an often-forgotten link. *Can Fam Physician* 2008;54:529–32.
- 5 Mori K, Ando I, Kukita A. Generalized hyperpigmentation of the skin due to vitamin B12 deficiency. *J Dermatol* 2001;28:282–5.

Copyright 2016 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow