

Review Article

Ageing of Bruise: Review of Histo-Chemical Changes with Time

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ABSTRACT

When a bruise develops, blood vessels are torn, blood escapes into the injured area, and there is an ensuing inflammatory reaction. Initially, the bruise appears red due to vasodilatation resulting from the inflammatory response to the trauma and extravasation of oxygenated blood. Later, interstitial deoxygenation of blood results in change of colour to blue or purple. The inflammatory response attracts macrophages that breakdown haemoglobin into biliverdin, which has a green appearance. During this process, some of the iron released combines with ferritin resulting in the formation of haemosiderin, which is brown in colour. Finally, a yellow colour is seen during the healing process which is attributed to the biliverdin being broken to bilirubin. Traditional forensic medicine textbooks in India document the formation of 'haematoidin' as one of the degradation products, but literature search does not prove this claim.

Keywords: Bruise, Dating, Colour change, Haematoidin, Haemosiderin, Biliverdin

INTRODUCTION

The word 'bruise' is derived from the old English word 'brysan', which means 'to crush'.¹ Bruises are common injuries that occur due to road accidents, physical assault, domestic violence, sexual assault, child abuse, and so on. They are caused by blunt instruments and are accompanied by pain, swelling, and inflammation.

Bruise occurs when the skin is stretched or crushed sufficiently without loss of integrity of the surface. This is followed by release of haemoglobin-containing erythrocytes from blood vessels into the tissue, if there is sufficient pressure of blood.

The appearance of the bruise may be influenced by the depth of the extravasated blood underneath the skin, the severity of the blunt force, the vascularity of the underlying tissues, diseases which affect the connective tissues or the ability of the blood to clot, and the age and gender of the injured person and colour of the skin. Over time the appearance depends on diffusion of haemoglobin through

the tissues and its removal by the inflammatory response.^{1,2}

Bruises change colour during the healing process and this colour change is used as a primary means to estimate the age of a bruise. It also helps to determine the approximate time of impact which can be corroborated with the history given by the patient. Forensic specialists document this colour change in medical records and this is often used as evidence by lawyers in criminal and/or civil courts. Therefore, accurate documentation of the age of the bruise requires a basic understanding of the biochemicals responsible for the colour change.³⁻⁷

The purpose of this article is to discuss the histo-chemical changes occurring during the healing of bruise and its correlation with the observed colour changes. Additionally, this article will discuss whether 'haematoidin' - a green-coloured pigment formed during the process of healing of a bruise - as mentioned in most of the forensic textbooks of Indian authors, is indeed a product of haemoglobin degradation.

A literature search of original articles, textbooks, and conference abstracts of Indian and foreign authors on haemoglobin metabolism and its degradation products with its associated colour change observed during the process of healing of a bruise was done using computerized databases done. Animals were not included in this because of significant difference in physiology between them and humans at a gross and histological level.

METABOLISM OF HAEMOGLOBIN

To understand the biochemicals formed during the healing process of a bruise, it is necessary to understand the degradation process of haemoglobin. Haemoglobin released from damaged erythrocytes is broken down to haeme and the globin is converted into amino acids. Haeme catabolism begins with oxidative cleavage of the porphyrin by haeme oxygenase to form biliverdin, a green linear tetrapyrrole which is then reduced by the enzyme biliverdin reductase to form yellow bilirubin (Figure 1). During this process, some of the haemoglobin is degraded by macrophages to haemosiderin, an intracellular storage form of iron consisting of an ill-defined complex of ferric hydroxides, polysaccharides, and proteins.⁸⁻¹³

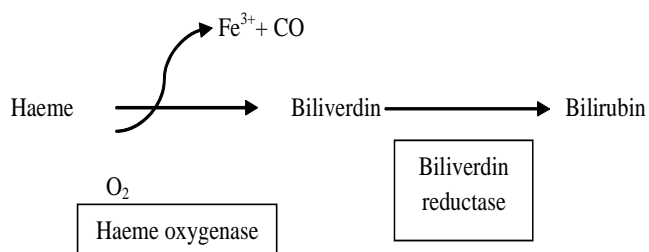


Figure 1: Metabolism of Haeme

DISCUSSION

In 1847, Virchow first described the histological pigment in old haemorrhages as a diffuse, granular and crystalline in structure, which he called 'haematoidin', and which was later identified to be bilirubin. Some texts describe 'haematoidin' as a haematogenous pigment apparently chemically identical with bilirubin. In 1869, erythrophagocytosis was detected in haemorrhages, and in 1888 the iron containing pigment in old haemorrhages was named haemosiderin and differentiated from haematoidin.¹⁴

The sequence of events in a bruise, as understood today, is that the red discoloration is caused by the release of haemoglobin and/or red blood cells from damaged vessels into the subcutaneous tissue. The extravasation of blood is followed by an inflammatory reaction that causes vasodilatation and attracts macrophages to the site of injury. Over time, these macrophages ingest the escaped erythrocytes and degrade the attached haemoglobin.^{5,14,15}

The erythema, secondary to vasodilatation and escape of blood, is quickly replaced by a blue or purple appearance due to the further extravasation of deoxygenated venous blood into the interstitial tissue. Haemoglobin is then broken down into biliverdin, which accounts for the green colour. Biliverdin is then quickly broken down into bilirubin, which accounts for the yellow colour of a healing bruise. As haemoglobin is broken down, some of the iron is released, which in turn combines with ferritin. This gives rise to haemosiderin, resulting in brownish appearance of a bruise.^{14,15,16}

Contemporary textbooks on forensic medicine written by Indian authors document the sequence of colour changes which a bruise undergoes with time. However, western authors have mentioned that haemoglobin is broken down into compounds that include haemosiderin, biliverdin and bilirubin which lead to a spectrum of colour change from purple to brown to greenish brown to green to yellow before complete fading, ultimately resulting in normal skin colour.^{2,15}

CONCLUSION

It is clear from the above discussion that haematoidin was synonymously used with bilirubin and is an outdated term. Moreover, it is not green in colour, as mentioned in most of the Indian forensic medicine textbooks. In most of the recent textbooks of biochemistry, there is no mention of haematoidin being formed during the degradation of haemoglobin. The word 'haematoidin' is used for historical purpose only and that too for bilirubin. Hence, the word 'haematoidin' should be replaced with the word 'biliverdin' which is greenish in colour and formed during degradation of haemoglobin.

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REFERENCES

1. Langlois NEI. The science behind the quest to determine the age of bruises-a review of the English language literature. *Forensic Sci Med Pathol* 2007; 3: 241-51.
2. Knight B. The Pathology of wounds. In: *Forensic Pathology*. 3rd ed. Arnold, 2004; p-136-73.
3. Maguire S, Mann MK, Sibert J, Kemp A. Can you age bruises accurately in children? A systematic review. *Arch Dis Child* 2005; 90: 187-89.
4. Bohnert M, Baumgartner, Pollak S. Spectrophotometric evaluation of the color of intra- and subcutaneous bruises. *Inter J Legal Med* 2000; 113: 343-48.
5. Bariciak ED, Plint AC, Gaboury I, Bennett S. Dating of Bruises in Children: An Assessment of Physician Accuracy. *Pediatrics* 2003; 112: 804-07.
6. Stephenson T, Bialas Y. Estimation of the age of bruising. *Arch Dis Child* 1996; 74: 53-55.
7. Carpenter RF. The prevalence and distribution of bruising in babies. *Arch Dis Child* 1999; 80: 363-66.
8. Murray RK. Porphyrins and bile pigments. In: *Harper's Illustrated Biochemistry*. Murray RK, Granner DK, Mayes PA, Rodwell VW. New Delhi: McGraw Hill 2003; p- 270-85.
9. Murray RK. Plasma proteins and immunoglobulins. In: *Harper's Illustrated Biochemistry*. Murray RK, Granner DK, Mayes PA, Rodwell VW. New Delhi: McGraw Hill 2003; p- 580-97.
10. Champe PC, Harvey RA, Ferrier DR. Conversion of amino acids to specialized products. In: *Lippincott's Illustrated Reviews: Biochemistry*. 4th ed. Lippincott Raven, 2007; p- 281-283.
11. Higgins T, Beutler E, Dumas BT. Hemoglobin, iron and bilirubin. *Tietz Fundamentals of Clinical Chemistry*, Burtis CA, Ashwood ER, Bruns DE (eds). 6th ed. Elsevier 2008; p- 509-26.
12. Nelson DL, Cox MM. Biosynthesis of amino acids, nucleotides and related molecules. *Lehninger Principles of biochemistry*, 4th ed. New York: W.H. Freeman and company 2005; p- 833-80.
13. Meisenberg G, Simmons WH. *Principles of Medical Biochemistry*. 1st ed. Mosby; 1998; p- 453-63.
14. Vanezis P. Interpreting bruises at necropsy. *J Clin Pathol* 2001; 54: 348-55.
15. Nash KR and Sheridan DJ. Can one accurately date a bruise? State of the science. *J Forensic Nurs* 2009; 5: 31-37.
16. Hughes VK, Ellis PS, Burt T, Langlois NEI. The practical application of reflectance spectrophotometry for the demonstration of hemoglobin and its degradation in bruises. *J Clin Pathol* 2004; 57: 355-59.
17. Dogra TD, Rudra A. *Forensic Traumatology*. In: *Lyon's Medical Jurisprudence & Toxicology*. 11th ed. Delhi: Delhi Law House, 2007; p- 444-56.
18. Parikh CK. Mechanical injuries-general aspects. Bruises (contusion). In: *Parikh Textbook of Medical Jurisprudence, Forensic Medicine and Toxicology*. 6th ed. New Delhi: CBS Publishers, 2004; p- 4.5-4.9.
19. Dikshit PC. Mechanical injuries. In: *Textbook of Forensic Medicine and Toxicology*. Delhi: Peepee publishers 2007; p- 155-71.
20. Reddy KSN. Mechanical injury. In: *The Essentials of Forensic Medicine and Toxicology*. 22nd ed. Hyderabad: K Suguna Devi 2003; p- 195-234.
21. Bardale R. Mechanical injury. In: *Principles of Forensic Medicine and Toxicology*. 1st ed. Delhi: Jaypee brothers 2011; p- 171-195.