

A Review of Clinical Signs Related to Ecchymosis

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ABSTRACT

Ecchymosis is a large area of discoloration caused by extravasation of blood into the subcutaneous tissue. It is an objective physical finding that may provide valuable clues as to its possible etiology. Ecchymosis is associated with eponyms based on the physician who first described the physical findings, which can be divided into 4 anatomical categories: base of the skull, abdominal wall and retroperitoneum, groin and scrotum, and lower extremity. Classic external signs and eponyms associated with ecchymosis are reviewed. Knowledge of these signs on physical examination may prove to be a useful clue directing the examiner to consider potentially serious causes of disease.

INTRODUCTION

Ecchymosis is defined as a large area of discoloration of the skin due to extravasation of blood into the subcutaneous tissue. The term is often used interchangeably with purpura, which describes similar characteristic discoloration of the subcutaneous tissue but usually is reserved for a larger, more extensive area of involvement. Ecchymosis is an objective physical finding that may provide valuable clues as to its possible etiology. The causes of ecchymosis are many; however, there are certain regions where the discoloration aids in the search for the etiology. The color of the subcutaneous tissue reflects the physiologic sequences of hemoglobin catabolism and its conversion to bilirubin and hemosiderin. Thus, the tissue progressively transforms over time from purple or black and blue to a yellow and green color and finally a brownish discoloration. It is recognized that the ecchymotic

region will have different shades of color, reflecting differential rates of hemoglobin catabolism.

Ecchymosis caused by internal conditions can be divided into 4 anatomical areas (Table 1). These regions are assigned an eponym associated with the physician who first described the physical finding. In this report, we review the classical signs and eponyms associated with ecchymosis that may be markers of potentially serious internal bleeding. Furthermore, these signs

may be potentiated by anticoagulation therapy or qualitative and quantitative platelet abnormalities. Prompt laboratory and imaging studies are important to further elucidate the cause of the ecchymosis and guide appropriate intervention. It is important that, in addition to a careful physical examination, a thorough review of the patient's medications and past medical history be conducted.

DISCUSSION

It was not until 1761, when Leopold Auenbrugger first described the technique of percussion, that the physical examination came into vogue. However, it did not become popular until 1808 when Jean-Nicolas Corvisart (personal physician of Napoleon) endorsed the importance of medical signs.¹ Shortly thereafter, Laennec invented the first stethoscope, expanding the horizon of physicians' senses.² The latter half of the 19th century saw steady improvement in new inventions including the ophthalmoscope (Hermann von Helmholtz, 1850), medical thermometer (Carl August Wunderlich, 1871), and sphygmomanometer (Riva-Rocci, 1896). Thus, physical signs gained importance and popularity by the beginning of the 20th century.^{2,3} During this time period, there was a continued rise in new inventions and discoveries, as well as the emergence of novel diagnostic tests in medicine. Conducting a complete examination with cognizant focus on physical signs remained paramount for a physician to make a diagnosis. Several books and articles were published advocating the importance of the physical examination and providing

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Table 1. Classification of Ecchymotic Eponyms

Ecchymosis involving the anatomical regions	Eponyms
Skull	Battle sign Blepharohematoma or Raccoon eye sign
Abdominal wall and retroperitoneum	Grey Turner sign Cullen sign Stabler sign
Groin and scrotum	Fox sign
Lower extremity (thigh)	Meniscus sign

Table 2. Causes for Cullen Sign

Causes	References
Acute Pancreatitis	Bosmann et al ⁹
Bilateral acute salpingitis in the presence of intrauterine pregnancy	Orient and Sapira ¹⁰
Cirrhosis with portal hypertension	Orient and Sapira ¹⁰
Hemorrhaging ascites from hepatic tumor	Mabin and Gelfand ¹¹ , Dalal and Mace ¹²
Hepatocellular carcinoma	Orient and Sapira ¹⁰
Hypothyroid myopathy	Orient and Sapira ¹⁰
Ischemic and gangrenous bowel	Kelley ¹³
Ovarian cyst hemorrhage	Orient and Sapira ¹⁰
Percutaneous liver biopsy	Capron et al ¹⁴
Perforated duodenal ulcer	Evans ¹⁵
Retroperitoneal necrotizing fasciitis	Pryor et al ¹⁶
Ruptured abdominal aortic aneurysm	Armour et al ¹⁷
Renal sarcoma metastatic to the peritoneum	Orient and Sapira ¹⁰
Rectus sheath hematoma	Guthrie and Stanley ¹⁸
Splenic rupture	Chung et al ¹⁹
Strangulation of ileum with hemorrhage	Orient and Sapira ¹⁰
Strangulated umbilical hernia	Orient and Sapira ¹⁰

Table 3. Causes for Grey Turner Sign

Causes	References
Acute Pancreatitis	Bosmann et al ⁹
Bilateral acute salpingitis in the presence of intrauterine pregnancy	Orient and Sapira ¹⁰
Ischemic and gangrenous bowel	Kelley ¹³
Retroperitoneal necrotizing fasciitis	Pryor et al ¹⁶
Ruptured abdominal aortic aneurysm	Armour et al ¹⁷
Rectus sheath hematoma	Guthrie and Stanley ¹⁸
Sclerosing peritonitis	Pryor et al ¹⁶
Cardiac catheterization	Armour et al ¹⁷
Intra-aortic balloon pump insertion	Rob and Williams ²⁰

education for physicians.³ The latter half of the 20th century saw the advent of integrative medicine with continued technological advances in the field of medicine and the advent of sophisticated diagnostic aids. In the last decade or so, the major emphasis has been on diagnostic modalities with a declining focus on the physical examination. Educating physicians on history and physical examination skills required for optimal delivery of excellent clinical care is being reemphasized.³ This review focuses specifically on physical signs related to ecchymosis, which are not only of

historical interest but also are clinically important bedside observations for a possible etiology of the patient's condition.

Abdominal Wall and Retroperitoneum Ecchymosis

There have been a number of signs associated with ecchymosis of the abdomen or retroperitoneum named after the individual who first described the finding. These signs are markers for a potentially serious cause of internal bleeding and, according to the site of discoloration, are variously named as Grey Turner sign involving the flanks and Cullen sign involving the umbilicus.

Grey Turner Sign

In 1920, George Grey Turner (1877–1951) reported “dirty-green” discoloration appearing on the lateral abdominal wall in a patient with acute pancreatitis,⁴ henceforth bearing his name to describe the condition. This sign is caused by extraperitoneal diffusion of blood from the posterior pararenal space to the lateral edge of the quadratus lumborum muscle, gaining access to the abdominal wall musculature through a defect in the transversalis fascia and eventually to the subcutaneous tissue of the flank.⁵ The presence of this sign in patients with acute pancreatitis is associated with a mortality of nearly 40%.⁶

Cullen Sign

In 1918, Thomas Stephen Cullen (1868–1953), a Canadian-American gynecologist, first described a bluish discoloration of the periumbilical skin in a female patient with a ruptured extrauterine pregnancy.⁷ Tracking of blood from the retroperitoneum to the umbilicus along the gastrohepatic and falciform ligament explains the pathophysiology of this sign.

Both Cullen and Grey Turner signs convey the same message, which is that intraperitoneal or retroperitoneal hemorrhage dissects to the subcutaneous tissue overlying the flanks or to the anterior abdominal wall, causing skin discoloration. However, the topographic location of the ecchymosis does not point to the etiology. Various theories have been proposed to explain the chemical properties required to develop these signs, including a direct role of pancreatic enzymes on the soft tissues and abdominal wall. On average, it takes 3 days for the appearance of Grey Turner's or Cullen's sign after the onset of pancreatitis.⁶ These signs may be found in 1% to 3% of all cases of acute pancreatitis^{5,6,8} and can occur in a broad range of clinical conditions (Tables 2 and 3).

Seat Belt Sign

The seat belt syndrome described by Garrett and Braunstein in 1962 refers to a pattern of sustained injury, including those involving the lumbar spine and visceral and solid organ injury, caused by the use of lap restraints.^{21–24} Doersch and Dozier²⁵ first described the term “seat belt sign” in 1968 as linear ecchymosis of the abdominal or chest wall following a motor vehicle accident. The location of the ecchymosis (seat belt mark) on the subcutaneous tissue overlays the position of the lap or diagonal strap on

the seat belt at the time of the accident. The contusion sustained at the time of the impact is believed to be caused by mechanistic forces directed to the abdomen or chest at the time of deceleration or impact. Patients with this sign who sustain more serious intra-abdominal injuries are more likely to be in the passenger seat position when other independent confounding factors such as crash severity and impact have been accounted.²⁶

Chandler et al²⁷ reported the presence of an abdominal seat belt sign in 14 of 117 cases. Of these 14 cases, two-thirds had an abdominal injury. Wotherspoon et al²⁸ performed a 6-year retrospective chart analysis of patients with abdominal wall and intra-abdominal injuries and reported no difference in intra-abdominal injuries between those people with or without a seat belt restraint. Velmahos et al²⁴ prospectively reported a 4-fold and 8-fold increase in thoracic and abdominal trauma respectively in patients with the seat belt sign compared to those without this finding. Thus, the sign lacks sufficient sensitivity and specificity in itself, and the presence of other physical findings in the appropriate setting may warrant serial clinical assessments and additional diagnostic evaluation or surgical exploration.²⁹

This same principle applies to the presence of this sign in area of the neck.³⁰ In children, Paris et al³¹ identified that the presence of free intraperitoneal air, lumbar fracture, and pulse rate higher than 120 beats per minute in the presence of seat belt sign was predictive of intra-abdominal injury warranting abdominal exploration. Furthermore, the absence of abdominal pain or tenderness in the presence of seat belt sign may be associated with a lower rate of serious intra-abdominal injuries.³² Other factors that increase the likelihood of intra-abdominal injury include rebound tenderness, abdominal distention, guarding, and hypotension.³² The presence of a seat belt sign is associated with an increased likelihood of musculoskeletal (eg, rib fracture), solid (eg, splenic, hepatic, and pancreatic), and hollow viscera (eg, mesenteric and intestinal) injuries.^{33,34} Conversely, its absence does not exclude underlying visceral injury. There is currently no model that incorporates a variety of clinical factors to determine which patient does or does not require further diagnostic evaluation to exclude intra-abdominal injury.^{20,35} Thus, an approach that includes a search for and recognition of this sign is of particular importance in patients with altered mental status or who are unable to cooperate with the examination due to other injuries.

Groin and Scrotum

The Blue Scrotum Sign of Bryant

The blue scrotum sign of Bryant as described in 1903 refers specifically to ecchymosis caused by a ruptured abdominal aortic aneurysm (AAA) extending into the scrotum.³⁶ Identification of this area of ecchymosis due to ruptured AAA is rare and may involve a region from the anterior abdominal wall, perineum, scrotum, lumbar regions, and in some cases extending to the knee.³⁷ In some cases, the ecchymosis may resemble Cullen and Grey Turner

signs. The ecchymosis may be continuous or patchy, bilateral or unilateral, and may involve the lower extremities.³⁸ This sign is typically first seen 3 or 4 days after the initial symptoms of pain, but may present hours³⁹ or even weeks after rupture.⁴⁰

In Bryant sign, blood must transverse the inguinal canal and spermatic cord down to the subcutaneous scrotal tissue. For Bryant sign to occur, there needs to be a coexistence of certain peculiar and specific clinical circumstances including a closed (retroperitoneal hematoma) or sealed (surrounding retroperitoneal and aortic tissue) rupture. In addition to the mode of rupture, the rate of leakage and a prolonged interval prior to final rupture also bear importance. Ecchymosis typically appears within 3 to 6 days after rupture of AAA.^{38,39-41} The delay is accounted for by the time it takes blood to extravasate the facial planes to reach its final destination, which is presumably influenced by the volume of blood loss and patient's dependent position.

Stabler Sign

In some cases of retroperitoneal hemorrhage, the blood may extravasate and cause discoloration of the inguinal-pubic area.⁴² This sign originally was described in adult patients suffering from acute hemorrhagic pancreatitis or ruptured ectopic pregnancy. Subsequently, it has been reported in various other conditions including AAA rupture. Although rare, this sign is most commonly identified in neonates secondary to adrenal hemorrhage.^{43,44} Obstetric injury, perinatal hypoxia, and sepsis are common causes for neonatal adrenal hemorrhage. A nonsurgical approach is generally recommended when ecchymotic sign is present in a neonate;⁴³ however, rarely, it may be due to ruptured neuroblastoma, in which case prompt search for underlying adrenal malignancy should be undertaken.⁴⁵

Thigh

Fox Sign

JA Fox, in 1966, reported 2 cases where bruising was noted in the upper outer aspect of the thigh caused by acute suppurative pancreatitis in one, and a ruptured AAA in the other.⁴⁶ In both cases, this sign was noticed late in the course and is produced by tracking of the fluid extraperitoneally along the fascia of psoas and iliacus beneath the inguinal ligament until it becomes subcutaneous in the upper thigh.⁴⁶ This sign has been described in other settings including strangulated ileum, urethral instrumentation, reaction to subcutaneous injections, and pulmonary infarction.¹³

Skull

During the evaluation of an injured patient, it is important to search for and expeditiously diagnose a skull base fracture (SBF) due to its high morbidity and mortality. This can be done by computed tomography (CT) imaging of the head.^{47,48} Diagnosis may be delayed, since the patient's general condition may prevent prompt imaging. In this situation, the physician needs to rely on clinical signs and symptoms. Described are raccoon eye

sign (RES) and Battle sign, 2 important clinical signs associated with fractures of the base of the skull. Both of these signs are associated with a high positive predictive value for the presence of skull fractures and intracranial lesions.⁴⁹ In a postmortem study by Herbella et al,⁵⁰ both signs were present in 24 of 50 cadavers (48%).

Blepharohematoma or Raccoon Eye Sign

Trauma to the frontal region of the skull may cause a fracture to the anterior cranial fossa and rupture of venous structure at its base, leading to bleeding that extravasates to the regions of the eyelid and orbital adipose tissue. This sign has a high positive predictive value (PPV) for basilar skull fractures as well as for intracranial lesions.⁴⁹ Kral et al⁴⁸ identified RES in 14 of 67 patients (21%) with frontal fractures, while Goh et al⁴⁷ reported RES in 52% of patients with skull base fractures, of which 28% had concomitant clinical signs. Thus, RES is a useful clinical feature suggestive of basilar skull fracture.^{47,48,50,51}

Battle Sign

Mastoid ecchymosis or retroauricular ecchymosis, also known as Battle sign, is a clinical indicator of base of the skull fracture in the posterior cranial fossa. This sign is named after Dr. William Henry Battle (1855-1936), an English surgeon.⁵² The presence of this sign is associated with a high positive predictive value (>75%) for the presence of an associated basilar skull fracture,^{49,53} so its presence should raise high suspicion and further diagnostic imaging for the presence of a basilar skull fracture.⁵⁴ A recent report described a case of hepatic encephalopathy with blepharohematoma and mastoid ecchymosis in the absence of trauma.⁵⁵

Disruption of the emissary veins that travel from the sigmoid sinus to the postauricular soft tissue result in the retroauricular ecchymosis. This sign is caused by blunt trauma to the mastoid or temporal bone resulting in a longitudinal or mixed fracture within the temporal bone. Other associated findings that may be seen in longitudinal fractures include laceration of the external auditory canal, hemotympanum, facial nerve injury, and transient vertigo.⁵⁶ Battle⁵² recorded that mastoid ecchymosis was often first observed 1 to 2 days after the injury, not immediately after the injury. When present, however, the patient is more likely to have a slower than expected recovery from head injury.

Knee

Crescent Sign

Good and Pozderac⁵⁷ reported 4 patients with gross blood in the knee and an acute synovial rupture syndrome. Each demonstrated ecchymosis that eventually reached the ankle, forming a crescent above 1 or both malleoli. The crescent sign is the presence of ecchymosis above 1 or more malleoli caused by synovial rupture in the presence of knee hemarthrosis. Hemarthrosis can result from trauma to the knee or from anticoagulant therapy or a bleeding disorder and is usually accompanied by painful swelling

of the joint.⁵⁷ Spontaneous synovial rupture causes extravasation of blood through the fascial planes to the calf, extending to the ankle. This typically results in the disappearance and resolution of the knee effusion followed by pain and swelling in the calf. Therefore, in a patient who presents with pain and swelling of the calf, both synovial rupture and deep vein thrombosis should be considered in the differential diagnoses, with presence of a crescent sign suggesting the former.⁵⁷

CONCLUSION

In 1890, Battle⁵² described mastoid ecchymosis in 17 patients who had head injuries with fracture to the posterior aspect of the skull base. Bryant³⁵ described scrotal ecchymosis as a manifestation of ruptured AAA in 1903. In 1918, Cullen⁴⁷ first described a bluish discoloration of the periumbilical skin in a female patient with a ruptured extrauterine pregnancy, and in 1920, Turner⁴ reported “dirty-green” discoloration appearing on the lateral abdominal wall in a patient with acute pancreatitis. In 1934 Stabler⁴² described cutaneous discoloration of the inguino-pubic region in ruptured ectopic gestation, and Fox⁴⁶ described bruising in the upper thigh in 2 cases (acute pancreatitis and ruptured AAA) in 1966. These cases describing the ecchymotic signs occurred at a time when there were no sophisticated diagnostic aids, but it was the careful clinical acumen of the astute clinicians that helped unravel the pathophysiology related to these ecchymotic signs.

In addition to diagnoses associated with these classical signs or eponyms, consideration must be given to anticoagulation therapy as well as qualitative and quantitative abnormalities of platelets that may precipitate or exacerbate bleeding. It is vitally important that in addition to a careful physical examination, a careful history of the patient’s medications and past medical history be obtained to help determine the etiology of these clinical signs.

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