Histopathology 2

Lecture 1

Environment and life-style related pathology

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OVERVIEW

The list of toxins the human body can be exposed to is extensive. Since the list of toxins and their pathologic effects is so numerous, only some of the major toxins and their effects will be discussed in this chapter. Of these, by far the two most commonly encountered toxins are tobacco smoke and alcohol. In addition to a few major toxins, this chapter will also discuss very basic physical trauma.

SOME SELECTED TOXIC SUBSTANCES

CIGARETTE SMOKE

Constituents: Polycyclic aromatic hydrocarbons, arsenic, nickel, carbon monoxide, hydrogen cyanide, nicotine.

Effects of nicotine: Increased heart rate, increased blood pressure.

Important points

■ About 30% of all cancer deaths and 90% of all lung cancer deaths are related to smoking.

■ Pregnant females who smoke 10 or more cigarettes a day can induce hypoxia in their fetuses, which leads to complications that include decreased fetal weight and prematurity and to premature rupture of membranes and placental abruption at the time of delivery.

ETHANOL

Toxic levels

■ Naïve users: 0.3 to 0.4 mg/dL can result in coma or death.

■ Tolerant users: Can develop levels up to 0.7 mg/dL.

Complications of ethanol use by organ system

■ Liver: Fatty change; hepatitis due to direct toxic effect of ethanol on hepatocytes; and cirrhosis, which occurs in only 10–15% of chronic alcoholics.

■ Central nervous system (CNS): Thiamine deficiency, which may present as Wernicke encephalopathy (Figure 1-1) or Korsakoff syndrome (amnesia and confabulation). Morphologic features of thiamine deficiency include periventricular hemorrhage and petechiae of the mammillary bodies and cerebellar atrophy. Complications of ethanol use include alcohol withdrawal and associated delirium tremens.

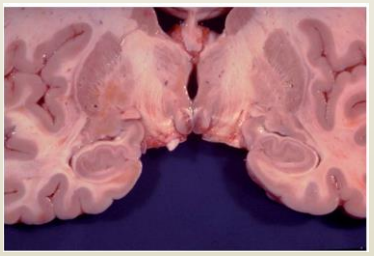

Figure 1-1:Wernicke encephalopathy. Note the punctate hemorrhages in the mammillary bodies. Wernicke encephalopathy is a complication of chronic alcohol abuse due to an accompanying thiamine deficiency

■ Cardiovascular: Dilated cardiomyopathy, hypertension.

■ Gastrointestinal: Gastritis, pancreatitis.

ALCOHOL WITHDRAWAL

Mechanism: Chronic alcohol use causes depression of α-receptors and β-receptors and enhances γ-aminobutyric acid (GABA), which serve as a stimulus to increase baseline neuronal activity. Therefore, when alcohol is withdrawn, patients have a sudden excited state of CNS activity because the depressive effect of the alcohol is removed.

Clinical presentation: Tachycardia, hypertension, tremulousness, and hyperreflexia.

Complications of alcohol withdrawal: Seizures (10%); hallucinations, usually visual (25%); and delirium tremens (5%). Severe alcohol withdrawal is life threatening, causing dehydration, hyperthermia, and electrolyte imbalances. A cardiac dysrhythmia can occur due to hypokalemia or hypomagnesemia.

■ **Delirium tremens**: Confusion, disorientation, tactile hallucinations (often of bugs on skin), and marked tremor.

- Treatment of alcohol withdrawal: Benzodiazepines; treatment of dehydration and electrolyte disturbances.

COCAINE

Mechanism

■ Blocks reuptake of dopamine, activating reward centers in the CNS.

■ Blocks reuptake of norepinephrine and epinephrine, leading to vascular constriction.

Complications: Sudden cardiac death, atherosclerosis, cardiac hypertrophy, intracerebral hemorrhage, placental abruption.

CARBON MONOXIDE

Mechanism

Hemoglobin has 200 times more affinity for carbon monoxide than for oxygen. Carboxyhemoglobin binds oxygen more avidly, resulting in impaired delivery of oxygen to tissues.

Presentation: Common in smoke inhalation, enclosed exposure to automobile exhaust, or in the wintertime with home furnaces. Patients present with cherry red skin (classic), flu-like symptoms, headache, and neurologic symptoms (Figure 1-2).

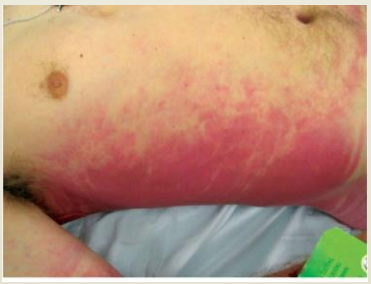

Figure 1-2. Cherry-red lividity due to carbon monoxide poisoning. This man committed suicide by remaining in a car with the windows closed and the engine running. Carbon monoxide produces a bright red discoloration of the blood. In this case, the lividity (postmortem change due to settling of the blood in dependent regions of the body) is cherry red in color instead of the usual red-purple, due to the carbon monoxide, which is irreversibly bound to the hemoglobin.

PHYSICAL TRAUMA

Basic descriptions (Figures 1-3 and 1-4)

■ **Abrasion**: Forceful removal of epidermis.

■ Contusion: Hemorrhage into soft tissue due to forceful rupturing of blood vessels.

■ Laceration: Splitting of skin due to blunt force. Unlike an incision, lacerations have nerves, vessels, and strands of soft tissue, which bridge the wound.

■ Incised wound: Sharp force injury; the wound is longer than it is deep and has no bridging.

■ Stab wound: Sharp force injury; wound is deeper than it is long.

THERMAL BURNS

Basic descriptions

■ Full-thickness burn: Involvement of epidermis, dermis, and dermal appendages.

■ Partial thickness burn: Involvement of epidermis and potentially the superficial dermis.

Important point: Involvement of > 50% of the total body surface area is serious (i.e., potentially lethal).

Complications of thermal burns

■ Edema, due to loss of protein.

■ Electrolyte imbalances.

■ Infections—particularly *Pseudomonas*.

■ Increased heat loss, which induces a hypermetabolic state that requires increased nutrition.

HYPERTHERMIA

Forms of environmental hyperthermia: Heat cramps, heat exhaustion, heat stroke.

Heat cramps

■ **Mechanism**: Loss of electrolytes through sweating.

■ **Manifestations**: Muscle cramps.

Heat exhaustion

■ **Mechanism**: Failure of the cardiovascular system to compensate for hypovolemia, which is secondary to water depletion.

■ **Manifestations**: Sudden prostration and collapse.

Heat stroke

■ Mechanism: Generalized peripheral vasodilation.

■ Complications: Necrosis of skeletal and cardiac muscle.

HYPOTHERMIA

Mechanism of injury: Crystallization of water; vasoconstriction can contribute to ischemia.

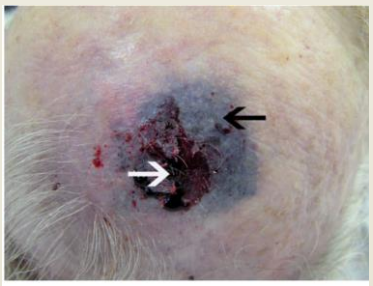

Figure 1-3. Contused abrasion of the scalp. This lesion on the scalp features components of both a contusion (black arrow) and an abrasion (white arrow).

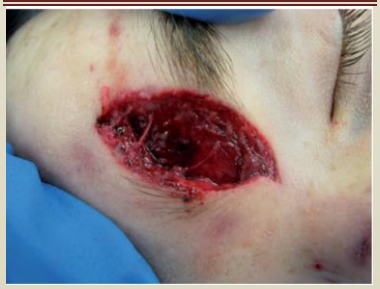

Figure 1-4. Laceration. Within the depths of this wound, note the vessels crossing perpendicular to the defect. This feature is bridging. The presence of bridging will distinguish a laceration from an incised wound. Incised wounds do not have tissue bridging.