Department of bioinformatics and biotechnology biology essay

Technology

Environmental ToxinsSubmitted To: Mr. Waseem SarwarSubmitted By: Muhammad Bilal (1041)BS BNB 3rd Afternoon 2011-2015

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Environmental Toxins

Definition:

(Environmental toxin) Pollution is the introduction of contaminants into a natural environment that causes instability, disorder, harm or discomfort to the ecosystem i. e. physical systems or living organisms.

Etiology:

Chemicals are ingested through food or water, inhaled or pass through the skin. Frequently toxicity is found in artists, researchers, or individuals who are exposed through their work in industry and agriculture. Exposure may be limited to small amounts over long periods or through massive amounts over a shorter time. Current research also implicates household products andCosmetics. Dental work can also be a source of toxicity.

Symptoms:

The most pronounced symptom is fatigue, frequently encountered even in young people. Joint pain and skin problems such as eczema and psoriasis are frequently found. Other serious heath conditions such as neoplastic processes and severe environmental sensitivity should be considered. We believe that further research will elucidate a more complete picture.

Common Environmental Toxins:

* Hydrocarbons* Inhaled toxins* Pesticides* Heavy Metals

Hydrocarbons:

Introduction:

One of most frequently reported poisonings. Presentation to ED classified into 4 types: 1.) Accidental ingestion,- Most common.- In children less 5 years. 2.) Intentional inhalation,- Abuse of volatile hydrocarbons.- Recreational. 3.) Accidental inhalation / exposure,- Household or workplace. 4.) Massive oral ingestion,- Suicide attempts.

Pharmacology:

Diverse group of organic compounds. Contain hydrogen and carbon. Most are petroleum distillates (e. g. gasoline)- derived from crude oil and coal- Turpentine derived from pine oil

Pathophysiology:

3 main target organs effected: 1) CNS2) Lungs3) HeartMost acute damage in the lungs.

LUNG DISEASE:

Mechanisms:

Penetrates lower airways ~ produces bronchospasm + inflammationDisplaces alveolar O2 (volatile hydrocarbon)Inhibits surfactantDamaging alveoli and capillaries

These effects cause:

Alveolar disfxVent / Perfusion mismatchHypoxiaResp. failure

CNS:

Narcotic – like effects:~ Euphoria~ Disinhibition~ ConfusionUsually substance abusers - recreational use. Single exposure with rapid onset of intoxication + recovery. Chronic use causes:~ Peripheral neuropathy~ Cerebellar degeneration~ Neuropsychiatric disorders~ Dementia~ Chronic encephalopathy

CARDIAC:

Sudden death. Sudden physical activity during / after intentional inhalation. Myocardial sensitization to endogenous + exogenous catecholamines. Precipitates vent. dysrythmias + myocardial dysfx.

Clinical presentation:

4 typical presentations:

1.) Accidental ingestion:

Usually toddlers. Reused beverage containers storing hydrocarbon. Mild Sx include ~ tachypnoea.~ Dyspnoea~ Bronchospasm~ fever within 6 hoursSevere poisonings ~ early resp. Sx~ Cyanosis~ grunting~ coughing~ Repeated vomiting~ These findings suggests aspirationChange in mental status ~ direct CNS effect OR~ caused by hypoxia

2.) Intentional inhalation:

Substance abuseMechanisms include: - " bagging"- hydrocarbon poured into bag/container+ deeply inhaled.- " Huffing" - inhaling through a saturated cloth.- " sniffing" Mostly volatile hydrocarbons – petrol.- Paint- Glue

3.) Accidental dermal exposure or inhaled resp. exposure:

In workplace / home. Not life threatening. Asymptomatic or transient non-specific symptoms. Sx resolve with fresh air / removal from offending environment.

4.) Intentional ingestion / intravenous injection:

Suicide attempts. Used in combination with other substances. Massive oral ingestion not associated with significant morbidity.

INHALED TOXINS:

* Smoke inhalation

* Cyanide

* Carbon monoxide

Smoke inhalation:

Inhalation injury common. Fires in enclosed spaces like homes / factories. Injury typically irritant in nature. Heated particulate matter + absorbed toxins injure normal mucosa. Carbon monoxide + Cyanide poisoning often associated with smoke inhalation.- These are systemic (not resp.) toxins.

Clinical presentation:

Morbidity + mortality related to resp. tract damage- Thermal / irritant in natureTime between smoke exposure + onset of Sx – highly variableMay

always be delayedDepend on degree + nature of exposureCough + stridor- Thermal + irritant induced laryngeal injuryCough stridor + bronchospasm- caused by soot + irritant toxins in the airwaysSubsequently – a cascade of:- Airway inflammation- Acute lung injury with pulm. Edema- Resp. failureBurned nasal hair + soot in the sputum suggest substantial exposureAlways consider CO + cyanide inhalation- in pt`s exposed to filtered / distant smoke (different room) OR- Relatively smokeless combustion

Cyanide:

One of the most rapidly acting poisons.

Causes:

1.) Smoke inhalation:

- Most common- Compounds containing carbon + nitrogen produce hydrogen CNGas when burned- Natural compounds (silk + wood) produces HCN as combustionproduct- burning of household furniture + plastics also causes HCN gas

2.) Intentional poisoning:

- Uncommon.- Cyanide salts in hospitals + labs.

3.) Industrial exposure:

- Occupations with easy access to cyanide* Chemists* Jewelers* Pest control* Mineral refining* Photography* electroplating* Dying + printing

Pathophysiology:

Cyanide inhibits mitochondrial cytochrome oxidase + blocks electron transport (binding with ferric iron Fe3+)Aerobic metabolism + O2 utilization

decreasesLactic acidosis occurs as a consequence of anaerobic metabolismO2 metabolism @ cellular level is grossly hamperedCyanide rapidly absorbed from:- Stomach- Lungs- Mucosal surfaces- Skin

Clinical presentation:

Sx appear seconds to minutes after exposureHCN gas can lead to cardioresp. arrest + death within minutesOnset of effects after ingestion / skin contamination:- Much slower (several hours)- Early signs: i) Dizzinessii) Bronchospasmiii) Dyspnoeaiv) Confusionv) Paresis- Later: i) Cardiovasc. Collapseii) Seizersiii) Coma

Pathophysiology:

Intense tissue hypoxia + cell injury caused by

2 mechanisms:

1.) Interrupts electron transport in the mitochondria (like cyanide), Leading to anaerobic metabolism2.) Reduces O2 delivery by:- competing with O2 for binding to Hb (CO has much higher affinity forHb, than O2!)- Shifting the HbO2 dissociation curve to the left

Clinical presentation:

Hypoxia without cyanosis. Myocardium + Brain mostly affected (high O2 consumption). Sx include:- Dizziness - convulsions- Headaches - coma- Confusion - cardio/resp. dysfx + death- Chest pain- Dyspnoea- Palpitations- Syncope

Pesticides:

1.) Organophosphates + Carbamates2.) Paraquat + Diquat Poisoning

Organophosphates + Carbamates:

Introduction:

Potent cholinesterase inhibitors. Accumulation of acetylcholine (Ach). Indirect stimulation of nicotinic + muscarinic receptorsAbsorbed through: - skin- Inhalation- IngestionCarbamate + OP poisoning clinically indistinguishableDifferences: - OP forms irreversible complex with cholinesterase- Carbamate complex reversible, with shorter duration ofaction (less than 24 h)- Carbamates penetrates blood-brain barrier poorly, therefore less CNS effects

Clinical presentation:

Minutes to 12 hours after exposure1.) Muscarinic effects: (post ganglionic)- Hyper secretion (sweating, salivation + bronchial secretions)- constricted pupils- Bradycardia + hypotension- vomiting + diarrhoea- Urinary incontinence- Bronchoconstriction- Also commonly referred to SLUDGE syndrome: 2.) Nicotinic effects: (preganglionic)- Muscle weakness- Fasciculations- Resp. muscle weakness

Paraquat + Diquat:

Most toxic herbicide known (weed-killers)Multiorgan toxicityDeath due to delayed pulm. fibrosis + resp. failure. Pathophysiology:- Cytotoxic O2 radicals generated- Selectively accumulates in the lungs- Lungs major target organs (except diquat)- Also liver, kidneys, heart + CNS- Absorption: * skin* GIT* Resp. tract

Clinical presentation:

1.) Chemical burns of oropharynx2.) Esophageal perforation + mediastinitis (extreme cases)3.) N + V4.) Skin irritation5.) Resp. injury:- High doses cause dyspnoea, ARDS + rapid multiorgan failure- Progressive pulm. Injury over 1 – 3 weeks with irreversiblepulm. fibrosis

Heavy Metal Toxicity:

Uncommon dxExceptions: 1.) acute iron toxicity (intentional / unintentional)2.) Lead exposureUnrecognized / inappropriately Rx result in significant morbidity + mortalityOther examples: arsenic, mercury, cadmium.

Toxicity depends on:

1.) Metal2.) Total dose absorbed3.) Acute/Chronic exposure4.) Age – children more susceptible to toxic effects + prone toaccidental exposures5.) Route of exposure - e. g. Elemental mercury, not dangerousif ingested / absorbed through skin

Sources:

Exposure through:* Diet supplements* Medications (herbal remedies)* Environment* Occupational / Industrial(Most acute presentation)* Ingestion of non food items e. g. Toys, paint chips, ballisticdevices, fishing sinkers, curtain, weights* Retained bullets (rarely causes lead toxicity)

Pathophysiology:

Remains relatively constant for all heavy metal toxidromesBinds to O2, Nitrogen + sulphydryl groups in proteinsResult in: ALTERATIONS OF

ENZYMATIC ACTIVITYNearly all organ systems involved:* CNS* PNS*

Haemapoietic* GIT* Cardiovasc.* Renal