Systemic Manifestations of Industrial Pollution

Tarachand K. Kamble * S.R.Tankhiwale **

The rapid proliferation of new industrial materials, new production methods and new commercial products in the 20th century - particularly since World War II – has gone forward with Rocket speed, however little attention is paid to assessment of their ill effects, resulting in ruining the environment and human health. The National Institute for Occupational Safety and Health (NIOSH) estimates that only 2% of employees in these businesses have access to industrial hygiene services and workplace monitoring programs[1]. The Institute of Medicine (I0M) estimates that the United States alone has need of 3100-5500 occupational physicians. However only 40 medical schools in United States offer residency training in occupational medicine [2].

The national research council of national academy of sciences concluded from the recent study that toxicologists have fairly complete information on health hazards for only 10% pesticides and only 18% of drugs in use today [1].

Moreover preventing work related and environmental illnesses in made difficult by –

1) The uninterrupted flow of new and untested chemicals into the workplace and environment.
2) The paucity of data about toxicological and environmental effects of these substances.
3) Diagnostic difficulties.
4) The problems of safe disposal of hazardous wastes.

The impact and effect of industrial pollution is enormous, virtually most of the parts and systems of body are involved.

Address for correspondence

***Professor in Medicine,**
J.N.M.C., Sawangi (Meghe), Wardha

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The nervous system is very vulnerable to a wide range of insults from environmental and occupational toxins. Despite of the presence of selective permeability barriers separating the systemic circulation from the brain and peripheral nerves, metals, gases, solvents and other chemicals penetrate sufficiently to cause delirious effect [1]. Many description of neurotoxicity exist through the history of civilization, like lead poisoning described by Greek physician before the birth of Christ, homicidal use of arsenic by Nero, Minamata Bay epidemic (Organic Mercury) and glue sniffer's neuropathy (Hexacarbons). Each part of nervous system, brain, spinal cord, peripheral nerve or muscle respond differently to the toxic injuries and manifest in a diverse manner. Depending on the location, type of neuronal dysfunction and selective vulnerability, toxicity may culminate in a wide spectrum of symptoms and signs in form of possible syndromes and combination of headache, pain, cognitive and psychiatrics disturbances, visual changes, seizures, ataxia, tremors, rigidity, weakness and sensory loss [1].

Despite of extraordinary advances in neuroimaging techniques over past two decades, CT scan, MRI have far less helpful in documenting neurotoxic injuries; PET and functional MRI are still in their infancy. Hence neurological evaluation of the patients largely depends on bedside history, physical examination, EEG, NCS, EMG and neuropsychological testing.[1]

Symptoms and deficits depend upon the involvement of neurons of brain and spinal cord. Acute syndrome like acute encephalopathy which can be diffused involvement of cerebral hemispheres, clinically characterized by

headache, irritability, disorientation, convulsions, amnesia, psychosis, lethargy, stupor and coma. It is due to acute exposure to many toxins for example - Acryl amide, arsenic, carbon disulfide, carbon monoxide, hexacarbons etc. [1,3,4,5]

Chronic encephalopathy having diffused involvement of cerebral hemispheres manifests with cognitive and psychiatric disturbances due to chronic exposure to many toxins. [1,3,4,5]

Parkinsonism where basal ganglions and extrapyramidal motor pathways are involved manifests with tremors, rigidity, bradykinesia and gait instability. This occurs due to exposure to manganese, carbon monoxide and methanol. [1,3,4,5]

Motor neuron disease where spinal cord motor neurons are involved, clinically presents with muscle atrophy and weakness is due to exposure to lead and manganese. [1,3,4,5]

Myeloneuropathy presents with parasthesias, sensory loss, hyperreflexia, Babinski's sign extensor, gait ataxia etc, is due to exposure to nitrous oxide and organophosphates n-hexane. [1,6,7]

Polyneuropathy where sensory, motor, autonomic nerve fibers are involved, manifest with anesthesia, numbness, weakness, loss of deep tendon reflexes and autonomic failure. It is due to exposure to acryl amide, arsenic, mercury, thallium, carbon disulfide, ethylene oxide, methyl bromide etc. Nerve conduction study and EMG are primary tools in laboratory to evaluate neuromuscular disorders, occasionally supplemented by nerve biopsy. [1,6,7]

Occupational hearing loss may be partial or total; unilateral or bilateral; and conductive, sensorineural or mixed. In work- place, hearing loss can be caused by blunt or penetrating injuries, explosions and thermal injuries such as where piece of welder's slag penetrates the eardrum. The sensory hearing loss results from deterioration of the cochlea, usually due to loss of hair cells from the organ of Corti. The common causes of hearing loss are due to continuous exposure to noise, blunt injury and exposure to ototoxic substances. Evaluation of hearing loss is done by test of spoken words, tuning fork tests, pure tone audiometer, Bekesy audiometer, speech audiometer etc. [1,8,9]

Immune hypersensitivity mechanism plays a important role in many disorders of occupational medicine. The most common immune hypersensitive occupational disorders include allergic rhinitis and asthma, hypersensitive pneumonia and contact dermatitis. The reactions are dependent on the host, duration, degree and type of sensitization and the antigens. [1,10,11]

Surveillance programs in United Kingdom and in British Columbia, Canada have indicated that occupational asthma accounts for 26 and 52% of all the occupational lung diseases respectively. In United States and Japan, 15% of newly diagnosed adult asthma is due to occupational exposure. [1,10,11]

The frequency of hypersensitive pneumonitis (HP) is unknown but varies with environmental exposure and the specific antigen involved. The prevalence of Farmer's lung among Wisconsin dairy farmers has been reported as 4.2 per 1000 and a Finnish cohort study demonstrated an annual incidence of 5 per 10,000 farmers. HP is due to inhalation of antigens present in moldy hay, such as thermophilia actinomyces. [12]

Due to industrial pollution, hematological toxicity has occurred in cyclic fashion, historically associated with the development of the chemical industries and the advent of each World War without adequate protection and education. [1,13,14.]

Exposure to hematotoxins may affect (a) blood cell survival (denaturation of hemoglobin and hemolysis), (b) metabolism (porphyria), (c) formation (aplasia), (d) morphology and function (Leukemia or prelenkemia), or (e) coagulation (thrombocytopenia). [1,13,14.]

RBC survival is shortened by oxidant chemicals like anilines, nitro benzenes and quinones, causing methemoglobinemia and hemolysis. Moreover hemolysis is caused by heavy metal like

arsenic, lead, mercury; copper etc. [1,13,14.]

Disorders of RBC associated with decrease oxygen saturation is caused by carbon monoxide. Blood cell formation and morphology (premalignant & malignant) is deranged by insecticides (Hexachlorocyclohexane, pentachlorophenol etc), chemicals, solvents, metals, minerals, petroleum products and ionizing radiation. Aplastic anemia, myelodysplasia and multiple myeloma are caused by benzene, ionizing radiation and trinitrotoluene, arsenic, ethylene glycol monomethyl ether etc. Toxic thrombocytopenia is developed due to days exposure of toluene diisocyanate, dieldrin, pyrethrin, ethane, turpentine, vinyl chloride etc [1,13,14].

The majority of cancers in adults are thought to be due to a combination of factors, including environmental exposure and lifestyle. About 2-8% of all human cancers are thought to be due to occupational exposure to carcinogens. The identification of occupational carcinogens is important because most of the occupational cancers are completely preventable with appropriate personnel practices and strict protective legislation. [1,15]

Lung cancers are caused by arsenic, beryllium, cadmium, chromium; mustard gases, radon, asbestiform fibers etc. Urinary bladder cancers are due to 4-aminobiphenyl, benzidine etc. Skin cancers are found due to exposure of coal tar, mineral oils, shale oils, solar radiation etc. Leukemias are seen in exposure to benzene, ethylene oxide ionizing radiation etc. [1,16]

Most occupational skin diseases result from contact with chemical substances of which there are more than 90,000 in the environment today. About 2000 or so are now recognized as contact allergens. [1,17].

Specific types of cutaneous irritants like hydrofluoric acid causes tissue destruction, wet cement can produce severe burns due high alkalinity of calcium oxide and hydroxide; contact with fibrous glass can manifest with itching, prickling, maculopapular rash, excoriation etc. [1,18].

Bacterial diseases like staphylococcal, streptococcal, cutaneous mycobacterium, atypical mycobacterium, tularemia, brucellosis, anthrax infections can occur in industrial exposures. Viral, fungal, parasitic diseases also are found in industrial environment. [1]

Chronic exposure to the varieties of toxic agents in industrial environment can be injurious to cardiovascular system. Cardiac arrhythmias, syncope and sudden death are caused by arsenic, chlorofluorocarbon propellants, hydrocarbon solvents, organophosphate and carbamate. Exposure to carbon disulfide appears to accelerate atherosclerosis and precipitate acute coronary ischemic events in workers of rubber and viscose rayon industries. Carbon monoxide and lead can cause and aggravate angina pectoris, intermittent claudication, cardiac arrhythmias, myocardial infarction, sudden death or congestive cardiomyopathy. Hypertension is induced by cadmium, carbon disulfide and lead. Myocardial injury can occur due to antimony, arsenic, arsine, cobalt and lead. Peripheral arterial occlusive disease leading to gangrene can be manifestation of exposure to arsenic or lead. In 1950, an epidemic of sudden death in young munitions workers was observed due to abrupt withdrawal from excessive exposure to organic nitrates. Coronary vasospasm with angina, myocardial infarction or Monday morning angina can occur in workers exposed to nitrates. [1,19,21]

The liver is the target organ of many industrial and environmental chemical agents and it plays a central role in their detoxification and elimination. Not only chemical agents but viral, bacterial and physical agents encountered in the workplace also affect the liver.[1] the liver can be injured and necrosed by carbon tetrachloride, chloroform, trinitrotoluene etc. The liver can be damaged by methylene dianiline or rapeseed oil by mechanism of cholestasis. The chronic liver involvement in form of cirrhosis is due to trinitrotoluene, polychlorinated biphenyls, tetrachloroethane; sclerosis and neoplasm because of arsenic and vinyl chloride; and

granuloma due to beryllium and copper. [1,20]

The diagnosis of liver dysfunction is done by liver function test which is sensitive enough to reflect the severity of underlying pathophysiological problem. However, unfortunately, no such test is available at workplace. [1,20]

The true incidence of renal failure secondary to occupational or environmental exposure in United States is not known. However, these exposures represent potentially preventable causes of renal failure.[1]

Following high dose exposure of certain organic solvents (Ethylene dichloride, chloroform, trichloroethylene, dioxane, toluence, phenol etc.), metals like arsenic chromium, cadmium, mercury and vanadium or pesticides, acute renal failure may develop within hours to days due to necrosis of tubules. Chronic renal failure or end stage renal disease (ESRD) may develop after exposure to cadmium, beryllium, in Chinese herb nephropathy due to aristolochic acid by way of chronic interstitial nephritis; and lead nephropathy. However glomerular lesion is seen after selected exposures to organic solvents or silicosis. [1,21]

Acute arsenic poisoning causes necrosis of intestinal mucosa and hemorrhagic gastroenteritis leading to fluid loss and hypotension. Cadmium and mercury also affect the system and lead to gastroenteritis. [21]

The occurrence of adverse reproductive outcomes is of great concern to the individuals and families. The concern has been surfaced by the incidents such as the contamination of fish with methyl mercury in Minamata Bay in Japan, resulting in an epidemic of mental retardation, cerebral palsy and developmental delay in their offspring. The use of polychlorinated biphenyl contaminated cooking oil in Taiwan resulted in intrauterine growth retardation and hyperpigmentation of the skin in the infants of exposed women. The agents that have been shown conclusively to be reproductive toxicants in human (other than medication) are few for e.g. ionizing radiation, mercury, lead and polychlorinated diphenyls.[1]

A number of adverse reproductive effects may results from male exposure to chemical and physical agents. The effects are infertility, sexual dysfunction (decreased libido or erectile dysfunction), azoospermia, oligospermia, teratospermia, asthenospermia, spontaneous abortion, still- birth, congenital defects, prematurity and low birth weight.[1]

Azoospermia and testicular atrophy is caused by alcohol, dibromochloropropane etc. Oligospermia manifests due to boron, carbon disulfide, chlordecone, 2-ethoxyethanol, ethylene glycol ethers, lead etc. Asthenospermia occurs due to ethylene dibromide chlordecone, carbon disulfide etc. Teratospermia is caused by 2,4-dichlorophenoxy acetic acid, 2-ethoxyethanol, ethylene dibromide etc. Decreased libido and impotence manifest due to exposure to manganese, mercury, vinyl chloride etc. [1,22]

The systemic manifestations due to industrial pollution are giant problems. However, they are preventable and can be solved by meticulous history, proper diagnosis, disability evaluation, workers' compensation, liability issues, insurance, legal responsibility and the last but not the least proper industrial hygiene at workplace and redesigned job procedure, equipments and working conditions, in a nutshell, "a workers' friendly environment".

BIBLIOGRAPHY:

1. Joseph LaDou (Editor): Occupational & environmental medicine, A Simon & Schuster co. 1997, Ed. 2
2. Castorina J, Rosenstock L: Physician shortage in occupational & environmental medicine. Ann Intern Medicine 1990; 113:983.
3. Carpenter D O: The public health significance of metal neurotoxicity. Cell Mol Neurobiol 1994; 14:591.
4. Davis L E et al: Methyl mercury poisoning. Ann Neurol 1994; 35:680.
5. Goldstein G W: Neurological concepts of lead

poisoning in children. Pediatric Annals 1992; 21:384.

6. Lotti M: Pathogenesis of organophosphate Polyneuropathy. Cri Rev. Toxicol 1991; 21:465.
7. Rosenstock L et al: Chronic central nervous system effects of acute organophosphates pesticide intoxication, LANCET 1991; 338:223.
8. Axelesson A et al (Editors): Scientific basis of noise induced hearing loss. Thieme Medical, 1996.
9. Berger E H et al (Editors): Noise and Hearing conservation manual, 5th Ed. American Industrial hygiene Association, 1996.
10. Chan-Young M, Malo JL: Occupational Asthma. N Engl J Med 1995; 333:107.
11. Cullen MR Cherniack M G, Rosenstock L: Occupational Medicine. N Engl J Med 1990; 594:322.
12. Dennis L Kasper et al (Editors): Harrison's Principles of Int. Medicine, McGraw Hill 2005 Ed. 16, vol II: 1516.
13. Rosenstock L, Cullen M R (Editors): Text book of Clinical occupational and Environmental Medicine, Saunders, 1994.
14. Sauter D, Goldfrank L: Hematologic aspect of toxicology. Haemetol onco clini North Am. 1987; 1:335.
15. Bang KM: Epidemiology of occupational cancer. Occup Med 1996;11 (3): 467.
16. Coultas DB, Samet JM: Occupational Lung cancer. Clinical Chest Med 1992; 13:341.
17. Adams R M (Editor): Occupational skin disease, 3rd ed W.B. Saunders, 1998.
18. Kirkpatrick J J et al: Hydrofluoric acid burns. A review. Burns 1995; 21:483.
19. Kaufman J D et al: Atrial fibrillation and sudden death related to occupational solvent exposure. Am J Ind Med 1994; 25:731.
20. Redlich C A et al: Clinical and pathological characteristics of hepatotoxicity associated with occupational exposure to dimethyl formamide. Gastroenterology 1990; 99:748.
21. Dennis L Kasper et al (Editors): Harrison's Principles of Int. Medicine; McGraw Hill 2005, Ed 16, Vol II: 2577.
22. Tas S et al: Occupational hozards for the male reproductive system. Critical Rev Toxicol 1996; 26(3):261.