CARBON MONOXIDE INTOXICATION IN CIGAR SMOKERS

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Introduction

CARBON monoxide intoxication, most frequently resulting from exposures in industry, poorly vented homes and suicidal attempts, is a relatively common clinical entity. Although cigarettes are known to produce significant amounts of carbon monoxide, the relationship between smoking and carbon monoxide intoxication has received little clinical emphasis. Cigar smoke contains comparable or greater amounts of carbon monoxide; however, since the smoke usually is not inhaled, less carbon monoxide is absorbed. Many reports appear in the literature concerning the clinical, experimental and biochemical effects of carbon monoxide, yet polycythemia in man secondary to arterial oxygen desaturation from smoking has to our knowledge not been reported.

The following cases relate chronic carbon monoxide intoxication from cigar smoking with polycythemia and decreased arterial oxygen saturation.

Case Report

Case 1: R. P., a 47 year old white male, was admitted to the Ohio State University Hospital, in September, 1964, for evaluation of polycythemia with decreased arterial oxygen saturation. He had been a heavy cigarette smoker from age 14 until three years prior to admission, thereafter smoking only commercial cigars, progressing to 40 to 50 per day and inhaling deeply. He had dyspnea on mild exertion, but had no other symptoms of congestive heart failure. For several years he had a morning cough productive of small amounts of non-purulent sputum. During his work as a welder he had found that breathing 100 per cent oxygen for a short period would improve many of his symptoms. Since December, 1963, he noted staggering when walking (without lateralization), fine tremor of his hands after exertion or on attempting precise movements, and bifrontal throbbing headaches.

In June, 1964, he was admitted to his local hospital with a history of seven months of nervousness, anorexia, shortness of breath and staggering gait. His evaluation revealed: a moderate plethora, a blood pressure of 120/90 mm. Hg. and a fine rhythmic constant tremor of the hands. The remainder of the examinations was within normal limits with no other signs of cardiac, pulmonary or neurological disease. Laboratory data included a hemoglobin of 19.2 gm.%, a hematocrit of 57 per cent and a normal WBC, differential, urinalysis, BSP, serology, FBS, BUN, thyroxin turbidity, PBI, and spinal fluid dynamics and chemistries. Blood volume determinations (RISA) revealed a plasma volume of 2.65 litres, a RBC mass of 2.45 liters and a total blood volume of 5.1 litres (normals : 3.1, 2.1 and 5.2 respectively). The arterial oxygen saturation was 70 per cent (capacity of 26.3 Vol. % calculated from the total hemoglobin, content of 18.4 Vol. % determined with the Natelson microgasometer). The arterial pH was 7.39, the pCO2, 35.9 mm. Hg., the HCO3, 20.9 mEq/liter, and the carbon dioxide, 22.0 mEq/liter. Repeat studies five days later, during which time his tobacco consumption decreased, revealed an arterial oxygen saturation of 82 per cent (capacity of 24.0 Vol. %, content of 19.6 Vol. %). The electrocardiogram showed a complete right bundle branch block. Pulmonary function studies included a normal vital capacity and maximum expiratory flow rate. The chest x-ray was normal, as were the pulmonary arteriograms which were of good quality. His discharge medications were chlordiazepoxide 10 mg. q.i.d., thiamine 25 mg. t.i.d. and multivitamin q.d., and he was advised to discontinue smoking. His subsequent tobacco consumption was reduced to 8 to 10 cigars per day. His symptoms improved progressively.

At the time of admission to Ohio State University Hospital, he had only moderate dyspnea on exertion with no tremor or ataxia. Physical examination revealed a well
developed, muscular white male in no distress with a blood pressure of 130/80 mm Hg. in both arms supine, a pulse of 80/minute and regular and respiration of 20/minute. The skin was warm with good turgor and slight facial plethora. Eyes, ears, nose and throat were unremarkable. The chest was symmetrical with equal expansion but slightly prolonged expiration. Scattered rhonchi were present bilaterally that cleared partially with coughing. The heart was not enlarged and the sounds were of good quality in sinus rhythm. No murmurs or adventitious sounds were present. The abdomen was soft with no hepatosplenomegalgy or masses. Equivocal softening of the nail beds was noted suggestive of early clubbing. The remainder of the examination including the neurologic was within normal limits. The laboratory data included a hemoglobin of 16.7 gm. per cent, a hematocrit of 50 per cent, normal WBC, sedimentation rate, platelet count, bilirubin, uric acid, electrolytes, creatinine and alkaline phosphatase. The electrocardiogram showed complete right bundle branch block unchanged from his previous tracing. The arterial oxygen saturation was 96 per cent (capacity of 22.7 vol. %, content of 21.8 vol. %, determined spectrophotometrically) with a PCO2 of 33.3 mm Hg. The hemoglobin was 13.9 gm. per cent, and the IVP x-rays were normal. Pulmonary function studies revealed a normal maximum breathing capacity, vital capacity, maximum expiratory flow rate. These were felt to be compatible with very minimal obstructive lung disease. At cardiac catheterization, right heart pressures, saturations and cardiac outputs were normal at rest and with exercise. No evidence of a shunt was found with repeated indocyanine green injections.

Following the above studies, on request he increased his cigar consumption to approximately 20 cigars per day. During this time his blood carboxyhemoglobin began to rise to 13% and 15% on two occasions. While at this level he developed bifrontal headaches and slight tremulousness. However, no neurologic deficits could be demonstrated.

Case II: F.W., a 46 year old white male was referred to the Lovelace Clinic, Albuquerque, New Mexico, for evaluation of polycythemia, headaches and dizziness of one year's duration. For many years he had smoked two to three packs of cigarettes daily until three months prior to referral, thereafter smoking only cigars (10 to 15 per day) and inhaling deeply as had been his custom with cigarettes. Three weeks prior to his initial evaluation he consulted his local physician with the above symptoms. His hematocrit at that time was reported to be 53 per cent (no other laboratory data are available) and a phlebotomy of 950 cc. was done. Two weeks later a second 500 cc. of blood was removed. Neither phlebotomy improved his symptoms. Physical examination at the time of his initial evaluation was unrevealing except for prolongation of the expiratory phase of inspiration. Laboratory data included: a hemoglobin of 13.9 gm., a hematocrit of 42 per cent, a normal WBC, differential, platelet count, blood volume and electrocardiogram. Chest fluoroscopy showed evidence of emphysema and mild fibrotic changes. Pulmonary function studies revealed moderate obstructive impairment with improvement after bronchodilator (isoproterenol aerosol) administration.

Using the gastrometric method, the arterial oxygen content was 15.4 Vol. % and oxygen capacity 17.1 vol. %; thus an oxygen saturation of 90 per cent. However, when the same content was related to the total capacity (18.9 vol. % calculated from the hemoglobin concentration x 1.36 = 18.9 vol. %), the saturation was only 81 per cent. Following the administration of 100 per cent oxygen, the arterial oxygen tension was 422 mm. Hg. At this tension the oxygen saturation was 92 per cent using the total hemoglobin concentration for capacity but 100 per cent plus .3 vol. % using the gastometric method. This discrepancy suggested that part of the hemoglobin was inactive as far as oxygen transport was concerned. Subsequent measurements of alveolar carbon monoxide concentration showed an average of 18 ppm, which corresponded to a carboxyhemoglobin concentration of 12-13 per cent.

Following one week of abstinence from cigars his subsequent symptoms had almost completely disappeared and his alveolar carbon monoxide was 10 ppm, which is within the normal range. Further arterial gas studies were not done.

Discussion

In these cases carbon monoxide intoxication from cigar smoking is suggested by the temporal relationship between the clinical and laboratory findings and excessive smoking. In Case I the reversal of all clinical and laboratory findings except the electrocardiographic abnormality after cessation of smoking and the ability to markedly elevate blood carboxyhemoglobin with increased cigar smoking tend to corroborate this relationship. In Case II, after cessation of cigar smoking, the alveolar carbon monoxide