Acute nickel carbonyl poisoning: a report of 179 cases

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Since 1961, 179 cases of acute nickel carbonyl poisoning in China have been collected and the clinical findings are presented in this paper.

General data

SEX, AGE, AND CAUSES OF INTOXICATION

There were 122 (68.2%) male patients and 57 (31.8%) female, and their ages ranged from 18 to 53.

The causes of intoxication included (1) accidental spills or leaks of nickel carbonyl liquid or vapour from apparatus or tubes, (2) feed in raw material, (3) checking and repairing of the apparatus and tubes, and (4) fire fighting. Accidental spills or leaks were the commonest cause.

AIR CONCENTRATION

According to some samples, the air concentration of $\text{Ni}(\text{CO})_4$ exceeded 50 mg/m^3 and the air concentration of CO ranged from 118.3 mg/m^3 to 1462.6 mg/m^3 .

CONTACT TIME

Fifty nine cases (32.9%) had exposure lasting less than 30 minutes, 51 (28.5%) one hour, 48 (26.8%) one to two hours, and 21 (11.7%) more than two hours.

TIME OF ONSET

In 74 cases (41.3%) symptoms appeared within 30 minutes to one hour, some within five minutes, and in 67 cases (37.5%) symptoms appeared within one to four hours. In the other 38 cases (21.2%) onset was delayed for a week.

Clinical observations

The symptoms and signs noted in the patients are listed in tables 1 and 2. The most frequent symptoms were referred to the respiratory and nervous systems. Symptoms and signs typical of toxic pulmonary oedema or chemical pneumonia, or both, were present in six severe cases, and of toxic myocarditis in three cases.

LABORATORY STUDIES

Leukocytosis was found in 25 cases (14.0%), values ranging from $12\,000$ to $23\,000/\text{mm}^3$ with a shift to the left. Leukocytosis was found in parallel with the severity of the intoxication. No eosinophilia were found. SGPT levels were slightly increased in six cases (3.4%) but returned to normal within three weeks. The urine nickel concentration was determined in 27 cases (16.3%) and ranged from 0.003 mg/l to 0.66 mg/l . The maximum excretion of nickel in the urine occurred one to two days after the onset of intoxication and returned to normal after one week.

ECG FINDINGS

Sinus tachycardia was seen in seven cases (3.9%), sinus bradycardia in three (1.7%), sinus irregularity with or without heart block in five (2.8%), premature ventricular contraction in two (1.1%), and myocardial injury in eight (4.5%), including toxic myocarditis in three (changes in S-T and T wave, Q-T time was prolonged).

RADIOGRAPHIC FEATURES

Increasing irregular linear shadows were seen in 16 cases (8.9%), including four with expansion and increased density of the hilus. Diffuse irregular nodular mottling or patchy shadows were seen in the lung fields in four cases (2.2%), pulmonary oedema in two (1.1%), and pleural fluid effusion in two.

TREATMENT AND PROGNOSIS

Mild cases were treated with rest in bed after removal from exposure. Bronchodilators and symptomatic drugs were also given. Oxygen, glucose, vitamin C, and corticosteroids were also administered in moderate cases. In severe cases we used a series of drugs and methods, such as limitation of water intake, administration of oxygen, hibernation therapy, and parenteral administration of large doses of corticosteroid, Dithiocarb, antibiotics, and other drugs to help resolve pulmonary oedema, pneumonia, and toxic myocarditis.

All the patients recovered and had no severe sequelae, but about one third had a neurasthenic syndrome and weakness after discharge. In some cases these symptoms persisted for three to six months.

Table 1 Clinical symptoms of acute nickel carbonyl poisoning

	Mild cases (n = 148)		Moderate cases (n = 25)		Severe cases (n = 6)		Total (n = 179)	
	No	%	No	%	No	%	No	%
Respiratory system:								
Chest tightness and pain	90	60.8	25	100.0	6	100.0	121	67.6
Dryness in the throat	60	40.5	13	52.0	5	33.3	78	43.6
Cough	26	17.6	14	56.0	5	83.3	45	25.1
Sore throat	12	8.1	8	30.0	4	66.6	16	8.9
Difficulty with breathing	5	3.4	16	62.0	6	100.0	27	15.1
Haemoptysis	2	1.4	0	0	1	16.7	3	1.7
Expectoration	1	0.7	5	20.0	5	83.3	11	6.2
Frothy sputum	1	0.7	2	8.0	3	50.0	6	3.4
Dyspnoea	0	0	10	40.0	6	100.0	16	8.9
Nervous system:								
Dizziness	98	66.2	25	100.0	6	100.0	129	72.1
Headache	69	46.6	22	88.0	6	100.0	97	54.2
Sleeplessness	15	10.0	10	40.0	3	50.0	28	15.6
Dysphoria	8	5.4	3	12.0	5	83.3	16	8.9
Blurred vision	3	2.0	7	28.0	3	50.0	13	7.3
Somnolence	3	2.0	5	20.0	1	16.7	9	5.1
Numbness	2	1.3	4	16.0	1	16.7	7	3.9
Digestive system:								
Nausea	84	56.8	25	100.0	6	100.0	115	64.2
Loss of appetite	22	14.9	7	28.0	6	100.0	35	19.6
Vomiting	4	2.7	6	24.0	3	50.0	13	7.3
Abdominal pain	1	0.7	1	4.0	1	16.7	3	1.7
Others:								
Weakness	71	48.0	21	84.0	6	100.0	98	54.8
Palpitation	21	14.1	9	36.0	3	50.0	33	18.4
Cold sweat	12	8.1	7	28.0	3	50.0	22	12.4
Intolerance of cold	9	6.1	11	44.0	1	16.7	7	3.9
Fever	2	1.4	4	16.0	1	16.7	12	6.7

On average, the recovery time was seven days in mild cases, 18 days in moderate cases, and 40 days in severe cases.

Discussion

Nickel carbonyl ($\text{Ni}(\text{CO})_4$) is a liquid metal compound of considerable toxicity. By inhalation the LC_{50} to LC_{100} for laboratory animals ranges from 100 $\text{mg Ni}(\text{CO})_4/\text{m}^3$ for a 20 minute exposure in the rat to 190 mg/m^3 for a 30 minute exposure in the cat and to 2500 mg/m^3 in the dog.¹ The pathology of

lesions in the lungs of rats and dogs after acute inhalation shows pulmonary congestion and oedema, interstitial pneumonitis with focal atelectasis, and necrosis.²

Sunderman and Vuopala have given a detailed description of acute nickel carbonyl poisoning in man.^{3,4} The acute toxic effects occur in two stages, immediate and delayed. In the present study the immediate toxic effect was predominantly manifested by neurological symptoms and irritation of the upper respiratory tract. The cases who had only immediate symptoms were considered as mild.

Table 2 Clinical signs of acute nickel carbonyl poisoning

	Mild cases (n = 148)		Moderate cases (n = 25)		Severe cases (n = 6)		Total (n = 179)	
	No	%	No	%	No	%	No	%
Hyperaemia of the conjunctiva	58	39.2	21	84.0	3	50.0	82	45.8
Hyperaemia of the throat	19	12.8	6	24.0	6	100.0	31	17.3
Cyanosis	0	0	5	20.0	5	83.3	10	5.5
Raucous breath sounds	7	4.7	7	28.0	0	0	14	7.8
Wheezing	1	0.7	1	4.0	0	0	2	1.1
Dry rales	3	2.0	8	32.0	4	66.7	15	8.4
Moist rales	0	0	0	0	4	66.7	4	2.2
Tachycardia	11	7.4	8	32.0	2	33.3	21	11.7
Tachypnoea	7	4.7	3	12.0	6	100.0	16	8.9
Arrhythmia	1	0.7	1	4.0	1	16.7	3	1.7
High blood pressure	10	6.8	3	12.0	1	16.7	14	7.8
Hepatic signs	7	4.7	2	8.0	1	16.7	10	5.6

In moderate cases the delayed stage was characterised by the appearance of symptoms such as chest pain, cough, dyspnoea, vomiting, palpitations, intolerance to cold, fever with leukocytosis, and some x ray abnormalities.

All six severe cases had pulmonary oedema or pneumonitis or both and three had toxic myocarditis which does not appear to have been reported before.

The direct toxic effect of $\text{Ni}(\text{CO})_4$ and CO on the heart—an impairment in lung function, relative lack of oxygen, and an increased load on the heart—all probably play a part in the production of this toxic myocarditis.

Our experience also showed that prompt help (first aid) and comprehensive treatment are important for

the recovery of the patients and that severe sequelae are unlikely. In addition, we also confirmed that corticosteroids are useful and helpful drugs in these cases.

References

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Examples of common forms of references are:

- ¹ International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;1:532–5.
- ² Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;294:687–90.
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