CASE STUDY OF THE BHOPAL INCIDENT

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Summary

The explosion at the Bhopal Union Carbide factory in 1984 is still the world's most lethal industrial disaster. It is well established that in its immediate aftermath many thousands of people died as a result of the inhalation of toxic fumes.

A large proportion of these deaths were probably due to acute respiratory damage. The existence, nature and extent of chronic respiratory disease in the survivors is far less well established. The results of the case study described above strongly suggest that such disease exists independently of the background of disease in the city's population.

The findings are also compatible with the belief that much chronic disease, attributable to gas exposure, is characterized by airflow limitation, probably including small airways.

Since this is likely to be relatively unamenable to direct treatment, the finding has important implications for the provision of appropriate health care to survivors in the city. The study also demonstrates the feasibility of epidemiological studies in this population, and reinforces the need and practicability of further work in this unfortunate population.

1. Introduction

Although the immediate health effects of the Bhopal gas disaster are undisputed (the deaths of many thousands of citizens) the long term *sequelae* are poorly understood. This case study describes an epidemiological survey which aimed to establish the existence (or otherwise), nature and extent of chronic respiratory disease attributable to gas exposure in 1984. In addition, the relevant literature in this, and related, areas has been examined and is summarized.

2. Background

2.1. Union Carbide in Bhopal

Bhopal, the state capital of Madya Pradesh, is geographically at the centre of India. About a third of its one million inhabitants live in tightly packed, shanty ('kucha') housing in its northern and central districts (see Figure 1).

In 1969, Union Carbide (India), a subsidiary of the large American corporation, set up a pesticide formulation plant (Figure 2) on the north edge of the city, originally to import, mix and package pesticides manufactured in the United States. Ten years later, a 5000 ton methyl isocyanate (MIC) production unit was installed, primarily to manufacture an effective and inexpensive carbaryl pesticide marketed as 'Sevin'.

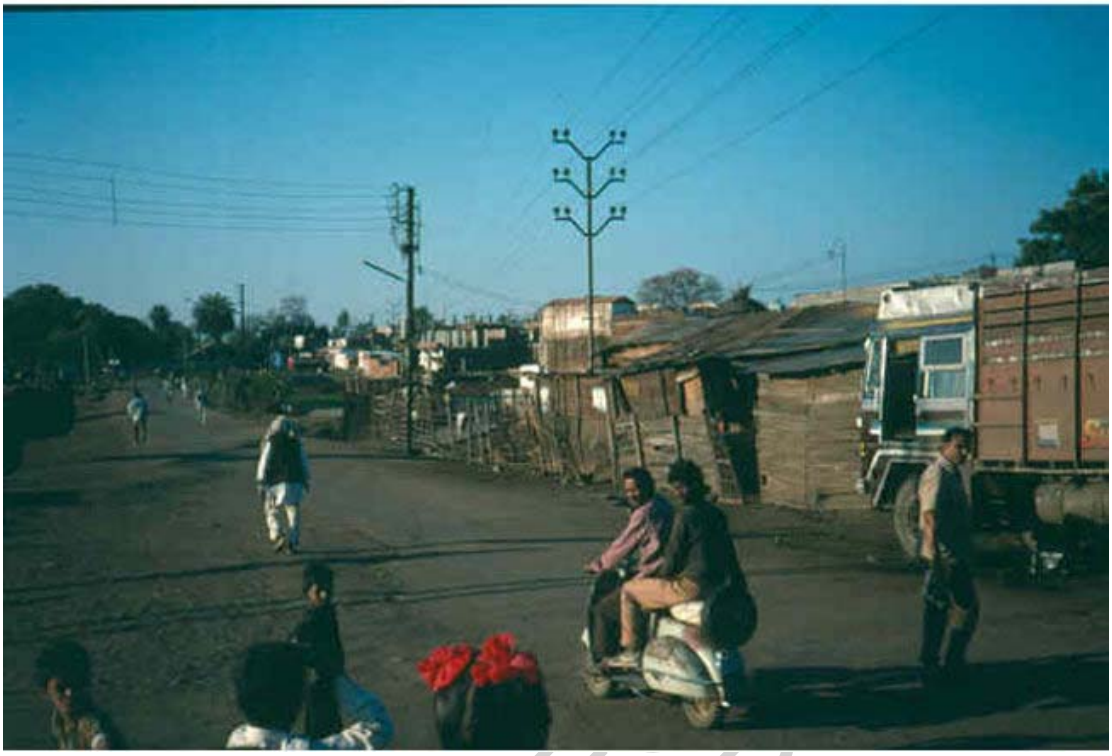

Figure1. Kucha housing adjacent to the site of the Union Carbide factory.

Figure 2. The (defunct) Union Carbide factory, Bhopal 1994.

MIC is produced by the reaction of (mono)methylamine with phosgene, both of which were manufactured elsewhere in India and transported in bulk to Bhopal. There it was mixed with 1-naphthol to produce Sevin for sale throughout the country. MIC is

colorless, with a low boiling point (39 ^{O}C) and high vapor pressure; because of its chemical instability it is stored under refrigeration in dry, stainless steel vessels. At the Bhopal plant, there were several such storage tanks, one (#610) having an unusually large capacity of 60 tons.

2.2. The Gas Disaster

For reasons that remain unclear, the cooling system of tank 610 was not functioning in the last months of 1984. Late in the evening of December 2nd, it is hypothesized that water (either through mechanical malfunction or operator error) entered the tank, mixing with the stored MIC.

The result was a violent, exothermic reaction, possibly catalyzed by ferrous corrosion of the tanks lining. By 01.00 a.m. the next morning, the tank ruptured and over the next few hours approximately 27 tons of vapor was discharged. Although most of this was probably pure MIC, products of hydrolysis (monomethylamine, carbon dioxide and various ureas) and pyrolysis (carbon monoxide, nitrous oxides and hydrogen cyanide) may also have been released in smaller quantities; the exact constitution of the discharged gases remains a matter for conjecture.

There is very little available information on meteorological conditions that night, but data from the city's airport suggest an air temperature of about 10 ^{o}C and a slow, northerly wind. At this temperature, the discharged MIC would have rapidly condensed and fallen groundwards, the plume passing over the northern edge of the city and towards its centre. An estimated 350 000 people were exposed. Immediate effects, and those over the following month, included the deaths of approximately 5000 people, most attributable to the direct respiratory effects of inhalation.

Over the next three years, health studies of survivors confirmed residual, obstructive airways disease, though its nature was poorly characterized. Ophthalmic *sequelae*, prominent in the weeks after the disaster, were believed to be more transient; the presence of disease in other organ systems was not convincingly established. Since 1986, only small case studies of persistent (respiratory) disease have been published and the question of causation has been poorly addressed. No further epidemiological studies have been completed.

2.3. The International Medical Commission

The Permanent Peoples' Tribunal is an international organization of health care, legal and environmental professionals which aims to provide an objective examination of situations with important health, environmental or human-rights implications. In 1992, the Tribunal met in Bhopal to examine the question of the disaster and its aftermath; and subsequently recommended an assessment of any persistent effects of gas exposure in combination with an examination of current health provision for survivors resident in the city. Applications were sought from interested physicians in 60 European, Asian and American universities. Of those responding, thirteen were selected and formed an International Medical Commission on Bhopal (Appendix). One, from Pakistan, was subsequently refused a visa. The Commission visited Bhopal, and carried out most of its

fieldwork, in December 1994.

3. Literature review

3.1. Technique

Searches of two computed bibliographies (*Medline* and *EMBASE)* were made; specifically, these sought literature both on the Bhopal disaster and on the epidemiological modeling of exposure-response relationships following toxic exposures. These required supplementation with methodological textbooks and further references listed in primary sources. A large quantity of grey literature, provided by the Indian Council of Medical Research (ICMR), survivors organizations in Bhopal and by The Other Media in Delhi, was also examined.

3.2. Findings

3.2.1. Immediate Effects of the Disaster

The timing of the explosion was such that most of those exposed (an estimated 350 000) were asleep. Survivors report being awakened by an acrid ("chili-like") stinging of the eyes and throat. The low boiling point of MIC caused it to be re-vaporized at body temperature and inhaled deeply into the lungs; as a result, damage to the bronchial tree was extensive and post-mortem findings in those who died immediately after the leak reported widespread airway necrosis with pulmonary edema and hemorrhage. It has not been possible to enumerate such deaths exactly, since public health resources in the city were rapidly overwhelmed, a large part of the population fled the city and most bodies were cremated before they and the cause of death could be officially documented; nor, given the paucity of district population figures within the city, has it been possible to calculate area-specific mortality rates. As a result, the only available figures have been crude numbers of deaths. Within 24 hours, it is estimated that 1700 people died, and a similar number within the next three weeks. By 1989, a final toll of 3598 deaths attributable directly to the gas leak—almost certainly an underestimate—was set by the State Government of Madya Pradesh. Given the pathological findings described above, most early deaths were believed to be due to acute pulmonary toxicity.

3.2.2. Toxicology of MIC: Experimental Studies

Most authorities agree that MIC, in vapor and gaseous form, constituted all or most of the original discharge; additional compounds may have been formed by the heat of the initial reaction (including hydroxyureas and oxides of nitrogen) and by the exothermic reaction with water in the mucous membranes and airways of those exposed (methylamine and dimethylurea). No simulation studies have been conducted, but examination of a core of residue from the ruptured storage tank, by a Union Carbide team of engineers in December 1984, produced results consistent with these speculations.

Prior to the disaster, toxicological data on MIC in both animals and humans were very limited, though its intensely irritant properties were recognized, as was its ability to

induce pulmonary edema after inhalation. An incompletely reported, dose-ranging study in human volunteers documented that there were no effects at levels of 0.4 ppm, but that exposures to 21 ppm were 'unbearable'. Soon after the Bhopal disaster, several groups undertook experiments in which experimental animals were exposed, by inhalation, to varying concentrations of pure MIC and the effects, over varying periods, were examined functionally and histologically. Its pulmonary irritant properties were confirmed and epithelial inflammation and loss, followed by repair with some peribronchial fibrosis were documented. The remarkable repair capability of the respiratory epithelium was commented upon, though excess and obliterating fibrous tissue were found in the small airways of both mice and rats who had survived a single, high-dose (30 ppm) exposure to MIC. Similar findings were reported, three months after exposure, in mice exposed for two hours to the same concentration of MIC (11). Longer term outcomes have not been reported.

3.2.3. Bhopal: Epidemiological Studies

The literature on the human effects of the gas leak is surprisingly scanty and largely comprises case series, or crudely designed population studies. Within weeks of the disaster, research programs had been set up by a number of Indian and international bodies: these included the ICMR, the Tata Institute for Social Sciences (Bombay), the World Health Organisation (WHO) and Union Carbide itself, as well as epidemiological and clinical groups from Bhopal University and other academic institutions in India. In all instances, save one, published reports from these have been based on the clinical experience of patients hospitalized in the period immediately after the leak. A large proportion of the long-term studies set up soon after the disaster has inexplicably failed to reach publication. Large cohort studies set up by the ICMR-sponsored Gas Disaster Research Centre have been reported only in the Council's Annual Reports, and even there in very little detail. There are, however, a small number of published studies reporting disease among survivors, though none with a follow-up of more than two or three years. Most of these have examined respiratory disease, as summarized in Table 1.

Subjects		Duration of follow-up	Major findings
n	Source		
500	hospital	72 hours	pulmonary oedema in 41%
978	hospital	2 days	symptoms of respiratory tract irritation; pulmonary oedema on selected CXR
33	hospital	1 week	respiratory tract irritation, pulmonary oedema (11%), 'pneumonia' (79%)
224	hospital	3 months	'obstructive' lung disease at spirometry (44%)
569	hospital	3 months	symptoms but not obstructive spirometry (22%) commoner in those living closer to the factory
1109	community volunteers	3 months	symptoms more frequent in those living closer to the factory. Radiographic changes 'caused by gas exposure' in 4%
82	hospital	6 months	'restrictive' lung disease at spirometry (78%) Interstitial deposits on CXR (95%)
87	hospital	2 years	spirometric evidence of small airways obstruction (grouped data)
459	community	3 years	gradient of symptoms with estimated exposure

Table 1. Studies of respiratory morbidity in survivors of the Bhopal disaster, 1984.

In all but one of these studies, subjects were drawn from a potentially unrepresentative section of the population, and it is questionable how far their findings can be generalized. Few examined both symptoms and respiratory function and none compared these in a systematic manner. The radiographic findings reported, apart from those indicating pulmonary edema in the hours after exposure, are impossible to interpret in the absence of a suitable control group. Several groups interpreted functional findings as evidence for restrictive airways disease though the results, as presented, are more likely to indicate small airways obstruction.

Interestingly, biopsies on three of the heavily exposed subjects revealed alveolar wall thickening, interstitial fibrosis and early changes of bronchiolitis obliterans, a finding also reported in other studies. Consistent with this are the results of a study which charted the progress of a small (initially 113) group of selected subjects with (unquantified) exposure to the gas leak; 87 were followed through a period of two years, after which initial reductions in most parameters of lung function had improved, though there remained evidence of minor small airways obstruction. Unfortunately, only group average values were presented, making it impossible to establish whether there were individual variations in response.

3.2.4. Attributing Disease to Gas Exposure

A weakness of much of the published literature, at least that which refers to outcomes beyond a week or two after the disaster, has been the question of attribution; few studies have examined to what extent abnormal findings can be causally attributed to gas exposure. Admittedly this is difficult where most of the proposed (respiratory) outcomes are distinguishable, if at all, from other, more frequent disease states only by elaborate and invasive investigation—and where individual and community data on respiratory disease rates prior to the disaster are not available. These deficiencies are not unique to Bhopal, though arguably were exacerbated by the scale of the disaster, and indeed are common problems in the assessment of industrial disasters, where specific outcomes are rare. The difficulties in this case are compounded by the continuing uncertainty over exactly which gases were discharged and the lack of previous, human toxicological data on MIC (the probable major constituent).

A common approach to this problem is the comparison of disease frequencies across two or more groups with differing levels of estimated exposure and the establishment, in this way, of an exposure-response relationship. A number of the studies cited above have employed this technique. A higher prevalence of respiratory symptoms has been described among those resident less than two kilometers from the factory at the time of the disaster and an increased frequency of obstructive changes on spirometry has been reported in this group, although this is not consistent with published figures. Another study categorised the subjects into three groups of estimated exposure, again based on distance of residence from the plant, but unfortunately failed to correlate this with morbidity. On the other hand, another study reported findings from an unusually elaborate and carefully followed cohort of survivors, sampled using a cluster technique

and comprising eight exposed and two non-exposed groups. Although this study aimed primarily to examine ophthalmic outcomes, some information on respiratory disease three years after the disaster is available. Reported symptoms of breathlessness, cough and chest pain were each more common across three categories of estimated exposure, although the trend was statistically significant only for the first of these. The gradients were not explained by differences in cigarette smoking. No measurements of lung function were made, and it remains possible that the findings were influenced by differential reporting across the exposure gradient. Irritant eye symptoms and the reported frequency of eye infections showed similar findings. Bronchoalveolar lavage was carried out in 36 exposed survivors, and demonstrated a trend of increasing (macrophage) cellularity across three categories of self-reported exposure intensity. Again, no functional measurements were made and the clinical implications of the findings of this study are unclear.

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