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SECTION 6
EXTRATHORACIC EFFECTS OF ASBESTOS

The extrathoracic effects of asbestos are summarized in Table 2-22.

ASBESTOS WARTS

In the past, asbestos warts were common in persons who handled asbestos fibers and textile. They occurred on the knuckles, fingertips, and forearms. Asbestos fibers, especially amphiboles rather than chrysotile, are responsible. The warts are tender and may persist for years unless the asbestos fiber is removed. They are rare today because manual handling of fibers is no longer done.[1]

GASTROINTESTINAL CANCER

Some disorders such as ulcerative colitis, familial polyposis, and to a lesser extent, regional enteritis predispose to the development of gastrointestinal malignancy. Exogenous effects such as diet and constipation also play a part. Asbestos may be an additional exogenous factor. Epidemiologic studies of cohorts of asbestos workers have shown an increased risk for gastrointestinal cancer. Data from recent series are summarized in Table 2-23. It may be that the true incidence of gastrointestinal cancer is understated, since some cancer patients will die from unrelated causes.

With the exception of the Finnish group exposed to anthophyllite, an excess of gastrointestinal malignancies is indicated in the other series. To gain further detailed information, the American Cancer Society has underway a prospective study of 16,000 asbestos workers.[11] The study will follow the workers for many years, and in the event of development of colon or rectal cancer, the surgical or autopsy specimens will be examined for the presence of asbestos fibers in the bowel epithelium and wall at the site of the lesion and in adjacent areas.

There are occasional observations of an excess of pancreatic, kidney,

Table 2-22
Extrathoracic Effects of Asbestos

1. *Known to be due to asbestos exposure*
   - Asbestos warts
   - Peritoneal mesothelioma
2. *Probably due to asbestos exposure*
   - Gastrointestinal cancer
   - Laryngeal cancer (only in smokers)
   - Splenic and hepatic capsular hyaline sclerosis
3. *Sporadic reports suggesting relationship with asbestos exposure*
   - Hemopoetic system malignancy
   - Breast cancer
   - Ovarian cancer
   - Renal cancer

Modified from Parkes.[1]

prostate, and brain cancers in communities occupationally or environmentally exposed to asbestos. Because of sex and exposure differences in the observed excesses of some of these cancers, a cause-and-effect relationship for asbestos has not yet been accepted.* In vitro experiments have not demonstrated a teratogenic effect for chrysotile.**

Source of Ingested Asbestos

Asbestos Workers

Some 99 percent of inhaled dust is passed back to the oropharynx by the tracheobronchopulmonary clearance mechanism and subsequently swallowed. Thus, about 100 times the amount of dust stored in the lungs will end up in the gastrointestinal tract. The significance is clear for those living or working in an asbestos-contaminated atmosphere.

General population. Asbestos fibers may reach the gastrointestinal tract from contaminated water, beverages, food, and air. Industrial wastes containing asbestoslike fibers has been emptied into Lake Superior since 1955. The adjacent population of Duluth has shown an increasing incidence of rectal carcinoma but not of cancer of the esophagus, stomach, and colon.[12] The

*Cancer mortality in relation to asbestos in municipal water supplies. Arch Environ Health 32:185–190, 1977.

**Schneider U, Maurer RR. Asbestos and embryonic development. Teratology 15:273–280, 1977.

Table 2-23

Author	Date	Site	Standardized Mortality Ratio (Observed/Expected)	Comment
Enterline and Wolff[3]	1973	Digestive tract	1.5	No difference between asbestos cement pipe workers (crocidolite exposure) and asbestos shingle and sheet workers (no crocidolite exposure).
McDonald[3]	1973	Digestive tract	Not given	Data suggested probable link with chrysotile exposure in Quebec miners and millers. Risk is probably dose-related. In the earlier deaths, increased risk for colorectal carcinoma was apparent; in later deaths, cancer of the esophagus and stomach became more common.
Newman, Kiviluoto, and Hakama[4]	1974	Digestive tract	No increased risk	Group too small and follow-up too short to be of definite statistical value. Exposure was to anthophyllite only.
Newhouse[5]	1974	Gastrointestinal tract	1.4	Exposure had been to crocidolite, amosite, and chrysotile. Histological verification of cause of death was poor.
Selikoff, Hammond, and Seidman[6]	1974	Esophagus, colon, and rectum	4.0	(a) Data based on 632 New York–New Jersey insulators. Cause of death when dying 20 or more years after first

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Table 2-23 (continued)

Author	Date	Site	Standardized Mortality Ratio (Observed/Expected)	Comment
				exposure. Standardized mortality ratio increased with elapsed time since first exposure, being approximately 1.7 in first decade after exposure and 4.0 more than 20 years after exposure. These workers had amosite and chrysotile but no crocidolite exposure.
		Esophagus stomach colon and rectum	1.4 2.6 1.5	(b) Data based on 17,800 asbestos insulation workers in U.S. and Canada exposed to chrysotile and amosite only. Standardized mortality ratios when dying 20 years or more after first exposure. Risk of GI malignancies was slightly less if dying earlier than 20 years after first exposure.
		Esophagus stomach rectum	No increase 3.1 2.8	(c) Data based on 933 amosite insulation workers in eastern U.S. Standardized mortality ratios when dying 20 or more years after first exposure. The absence of increased risk of esophageal cancer is stated to [illegible]

Enterline, Decoufle, and Henderson[7]	1972	Digestive tract	1.3	Data based on 1404 retired asbestos workers, average length of service 25 years. Some had very high exposures, i.e., more than 50 million particles per cubic foot of air. The standardized mortality ratio was dose-related, ranging from 1.24 to 1.8 according to calculated cumulative dust exposure. There was little difference in standardized mortality ratio between production and maintenance service workers. There was exposure to chrysotile, amosite, and crocidolite.
Anspach[8]	1974	Stomach, colon, rectum	2.3 (approx)	Data derived from 397 deaths among Dresden asbestos workers and extrapolated from author's data. Standardized mortality ratio not quoted in original article.
Doniach, Swettenham, and Hathorn[9]	1973	Stomach	1.6	East London workers with occupational exposure in shipping, insulation.
Lumsley[10]	1976	Digestive tract	Increasing SMR after 1960 onwards but not yet reached statistical significance	Data based on cancer registrations of dockyard workers, not cause of death.

predilection for a rectal site is at variance with what might be expected ingested carcinogen, but the period of follow-up is short. An average water supply may contain 2–9 million asbestos fibers per liter. occur when, as in Thetford, Canada, the source is a lake in an asbes area and counts of 173 million asbestos fibers per liter are obtained. fibers may originate from asbestos-cement pipes used for water main counts are 400–1400 fibers per liter, which are considered to be approaching zero risk to health." Chrysotile filters are used in the fil beer, wines, spirits, and soft drinks because of the ability of asbestos bacteria, thus imparting a permanent clarity to the liquid. No health known to exist,[14] even though asbestos particle counts up to 4 million (beer) and 12 million per liter (ginger ale) have been recorded.

Talc-treated rice has obtained notoriety as a cause of gastric cancer especially when intended for Japanese consumers may have talc added after the milling stage to increase its whiteness and to retain its storage. Although United Kingdom samples of talc are not reported to asbestos, many U.S. samples contain varying amounts of chrysotile and phyllite, since both minerals may be present in the same rock vein presence of asbestos is said to explain in part the high mortality from cancer in Japan. The age-adjusted mortality resulting from this dis Japanese males age 62–63 is 68 per 100,000 male population, abou times the U.S. rate. Furthermore, the rate is less in those areas of Japa cereals and soya beans are used as rice substitutes and in Japanese Hawaii or the continental U.S.A. who use fewer rice products.[15]

For most people, asbestos in the air is the more important than as contaminated water and rice.

Pathogenesis

Accepting that much inhaled asbestos may eventually find its way gastrointestinal tract and that there is an increase in incidence of intestinal cancer in those working or living in an asbestos-contaminat ronment, the actual method of induction of these neoplasms is obscu particles in general could penetrate gastrointestinal mucosa with any would expect that the wall would become anthracite-colored in coal and fibrotic in hard rock miners, while mesenteric lymph nodes would of become stony hard, respectively. This is not the case with regard wall, although occasionally calcified mesenteric nodes are seen abdomen in chest radiographs of miners. Asbestos fibers must act diff entering the gastrointestinal mucosa and inducing carcinoma. In rats

opened abdominal wall, intragastric injection of asbestos fibers has resulted in later accumulation of these fibers in lungs, brain, omentum, and other organs via the bloodstream. If one accepts that asbestos fibers reached these sources by penetration of the gastrointestinal mucosa (rather than by leakage through the injection site), then one needs to consider the mode of penetration. Long fibers might penetrate the gastrointestinal mucosa, but most of the fibers are small because gastric acidity will disintegrate chrysotile fibers into small fibers. Engulfment by pinocytosis would need to be postulated. The omentum had the highest proportion of asbestos fibers in this experiment, and since the omentum contained peritoneum and small lymph nodes, this may have a bearing on the induction of mesothelioma. Whether the omentum has a specific affinity for asbestos fibers or whether lymph nodes filter out and retain the fibers is conjectural.[12] In mice fed a diet containing 6 percent by weight of chrysotile electron microscopy shows uncoated chrysotile fibers in the colonic mucosa.[16] Transmission electron microscopy has revealed the presence of amosite fibers in the jejunal epithelium and lamina propria of rats after three months of asbestos feeding (see Fig. 2-94C.)

In most experiments where asbestos has been given by natural feeding (not by injection), short-term tests have revealed no appreciable penetration of fibers through the gut wall (considered by most a prerequisite for peritoneal mesothelioma), and long-term feeding has not led to any gastrointestinal tract cancer.[14]

The poor correlations between these animal experiments and epidemiologic experience with asbestos fibers has posed many questions. Analogous to the multiplicative effect of smoking in asbestos and induction of lung cancer, organic additives such as nitroso compounds may play a synergistic role.[17] Another question relates to the difference in the mucosal barrier between species and individuals. Mucosal barrier differences are related to blood groups and susceptibility to gastric ulcer. Do certain mucosal barrier differences increase absorption of asbestos particles and lead to gastrointestinal neoplasm?

Pathology

Stimulated by the high incidence of gastric cancer in the Japanese, surgical specimens received from Japanese stomach cancer patients have been examined for asbestos fibers. Chrysotile and amphibole fibers (crocidolite or amosite) have been found in only three out of seven specimens in one series (one colloid cancer and two adenocarcinomas), but talc was present in all seven. A search for ferruginous bodies (as opposed to asbestos fibers) in U.S. colon



Fig. 2-92. *Adenocarcinoma of the stomach in a 49-year-old asbestos worker. (A) The flattening of the upper part of the lesser curvature was missed. (B) Three months later, the tumor was nonresectable. An incidental duodenal diverticulum is present; displacement of a duodenal diverticulum may be an early sign of cancer of the head of the pancreas.*

neoplasm specimens examined by light microscopy was unrewarding. In 16 patients, none had typical ferruginous bodies in their colonic mucosa. Eight patients, however, mainly those with cancer of the right colon, had atypical ferruginous bodies. It is conjectured that electron microscopy might yield greater counts, since chrysotile is broken up by an acid medium (such as stomach acid) into submicroscopic particles.[16]

Radiology

Examples of an esophageal and colonic cancer in prior asbestos workers are seen in Figures 2-92 to 2-94; there are no distinguishing features. Both patients had radiographic evidence of pleural plaques. Some patients with asbestosis may have multiple primary neoplasms developing synchronously or metachronously; two neoplasms in the same patient are not rare, and three neoplasms have been known to occur. The most frequent association is colorectal cancer and bronchogenic carcinoma, both in asbestos workers and in the control population.[17] With this possibility in mind, solitary masses or even two masses in the lung of asbestos-exposed patients with colorectal or other cancer need not be metastatic, and the possibility of synchronous or metachronous lung malignancy needs to be considered.



Fig. 2-93. *Esophageal cancer in a shipyard worker. Those exposed to chromate and arsenic have a heartbreak burden of cancer of the esophagus.*

LARYNGEAL CANCER

There is no conclusive evidence linking asbestos exposure and laryngeal cancer in those who do not smoke. Morgan and Shettigara concluded from a study of asbestos workers, matched for their smoking habits, that the risk of laryngeal cancer is confined to those who smoke.[20] It is postulated that carcinogens in smoke may be absorbed onto asbestos fibers and play a part in induction of laryngeal cancer.[21]

Earlier reports[8,22] which were not matched for smoking habits[23] did assert an increased susceptibility to laryngeal and other head and neck cancers. Of



Fig. 2-94. A: Apple-core deformity due to cancer of the transverse colon. B: Pleural plaques in the same patient shown during air contrast fluoroscopy. C: Scanning electron micrograph. Amosite asbestos fiber penetrating epithelial cell of jejunal mucosa. Bar = 1 μm long is shown at lower right. × 2 000. (See facing page: From penetration of the small intestinal mucosa by asbestos fibers. by Shivers, R. M. and Brown, M. Mayo Clin. Proc. 52:609–614, 1977. Reproduced with permission of the author and the publisher.)



4000 asbesto- factory workers, 2 developed laryngeal cancer compared with an expected incidence of 2 per 100,000 of the general population,[24] i.e., a 25-fold incidence. Libshitz et al. described 3 patients with laryngeal cancer, all smokers and all with a history of asbestos exposure, whose radiographs show typical pleural plaques and, in one instance, pulmonary fibrosis.[25]

HEMOPOIETIC SYSTEM MALIGNANCY

An association between asbestos exposure and subsequent development of malignant disorders of the hemopoietic system is suggested by the following observation. A fivefold incidence of such malignancies has been found in asbestos workers who had typical radiographic findings of plaques and pulmonary fibrosis. The malignancies include multiple myeloma, lymphosarcoma, polycythemia vera, and Waldenstrom's macroglobulin anemia.[26] Other surveys of asbestos workers have shown 2 leukemias in 58 neoplasms;[27] 1 lymphosarcoma in 96 neoplasms;[28] 3 leukemias, 1 lymphoblastoma, and 1 patient with multiple myeloma in 21 neoplasms;[29] and 2 lymphosarcomas and 1 leukemia in 380 consecutive deaths.[26]

Because of these epidemiologic findings, bone marrow aspirates have been examined in groups of otherwise healthy asbestos workers. In one group with long exposure to crocidolite, amosite, and chrysotile, there was a generalized hyperplasia in 16 out of 17 workers, especially of the myeloid series, reticulum cells, and plasma cells.[30] Despite this hyperplasia, peripheral blood samples showed a total leukocyte count in the low normal level or leukopenia. There is presumably a phagocytosis of the excess leukocytes in the reticuloendothelial system of the lungs. The marrow changes did not show any



Fig. 2-95. *Illustration of metastatic lung disease from breast cancer in a male asbestos worker. Tomographic section shows multiple metastases (horizontal arrows) and calcified and noncalcified plaques (vertical arrows).*

correlation with the lung changes. Despite this data, a causal effect by asbestos in the induction of hemopoietic system malignancy is not yet accepted.

BREAST CANCER

In a study of East London necropsies of patients with known high occupational and or environmental exposure, there was 1.4 times the expected incidence of breast cancer in women.[3] This report is the only one suggesting any association between asbestos exposure and breast cancer. If asbestos is incriminated the fibers would need to be carried into the breast by direct extension from pleural plaques or by lymphatics. The former would not be surprising when one remembers how deeply into the diaphragm and intercostal muscles calcified and noncalcified plaques penetrate.

Figure 2-95 illustrates metastatic lung disease from breast cancer in a male asbestos worker. Carcinoma of the male breast differs from that in women by several features.[31] The stage of the disease is more advanced with some 65 percent of patients in TNM stage III or IV on presentation; a comparable figure for women is about 45 to 50 percent. The age of onset is on the average 5 to 10 years later in men than in women. Double primaries are more common in male patients with breast cancer than in women—13 percent in men compared with 3 percent in women; in men there is a disproportionate involvement of the large bowel and rectum. The histological pattern in male breast cancer compared with women shows a higher proportion of intraductal carcinoma, less comedocarcinoma, and no lobular medullary or mucoid carcinoma. An association exists between male breast cancer and Klinefelter's syndrome, and clinical assessment, together with a search for Barr positive cells and additional X chromosomes, is warranted. A causal effect by asbestos in induction of breast cancer is not yet accepted.

OVARIAN CARCINOMA

The role of asbestos in the induction of ovarian cancer is conjectural, and not yet accepted. There is experimental evidence that intraperitoneal injection of tremolite asbestos is associated with subsequent development of ovarian cancer in experimental animals.[32] Furthermore, in one group of 15 female asbestos workers, there was an increase in the incidence of ovarian malignancy.[33] This is not confirmed by other epidemiologists. It is often difficult to differentiate mesothelioma and papillary adenocarcinoma of the ovary on a microscopic basis. In addition, a large malignant mass arising from the pelvis of a female asbestos worker may present a problem in diagnosing the site of origin, which may not be apparent even on most careful palpation. Histological diagnosis may be in doubt even when specialized histochemical stains and assays for acid mucopolysaccharide are used.[33,34] The ovary is the most common origin of undifferentiated peritoneal carcinoma in females, and this organ is therefore frequently stated to be the primary site in carcinomatosis peritonei, while some such cases may be diagnosed as mesothelioma by other observers. Finally, there is the fact that anthophyllite may be converted naturally to talc, and talc has been found in female genital malignancies. Talc crystals were found in 10 out of 13 ovarian cancers, in 12 out of 25 cervical cancers, and in 1 primary endometrial cancer but not in a secondary ovarian malignancy in the same patient.[35]

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