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Urinary apparatus tumours and asbestos: The Ramazzini Institute caseload

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Summary

Many studies have drawn attention to the possible association between occupational exposure to asbestos and tumours of the urinary apparatus. Besides the main etiological agents recognised today – such as smoking, obesity and hypertension – experimental and epidemiological evidence converges on the view that tumours of the kidney and bladder are largely due to occupational exposure to industrial agents: these and their transformation products linger in the body and are eventually eliminated by those organs. That one such agent targeting the urinary system is asbestos has found confirmation in the discovery of asbestos fibres in the urine of populations at risk. We here present 23 cases of work exposure to asbestos in a range of exposure scenarios where the workers developed tumours of the kidney and bladder. The cases came to the attention of the Ramazzini Institute casually.

Key words: Asbestos; Renal cancer; Occupational exposure.

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Tumours of the urinary apparatus in workers exposed to asbestos

Renal excretion is one of the main elimination pathways for the toxic and carcinogenic chemical agents and metabolites to which man may be exposed. The urinary system is naturally often a target of such agents, the kidney being the filter in which they may be trapped, and the bladder a place where they may linger for a long time before being excreted.

The likelihood that asbestos has a carcinogenic effect on urinary tract tissues is supported by many studies which have shown the presence of asbestos fibre in the urine, caused by the occasional migration of such fibres from the gastro-intestinal wall to the bloodstream then into the urinary pathways (1-4). Asbestos formations have been detected in the kidneys and increased risk of kidney tumours was noted among miners from the Quebec asbestos mine (5). The presence of asbestos fibres in the urine both of occupationally exposed subjects (6-8) and of people who had drunk water contaminated with amphibolic asbestiform fibres (9, 10) has been documented on various occasions. Evidence of asbestos fibre lodging in the kidneys has come from experimental and epidemiological studies based on autopsy (11-14). It is thought by some researchers that asbestos formations start in the lungs and then migrate to the kidney. Once the asbestos fibres reach they form around asbestos fibres that had migrated to the kidneys in their own right. This is based on the demonstration that, once they reach the lungs by inhalation, they may pass the alveolar barrier, cross the interstitium and reach the lymph vessels and blood circulation; from there the fibres get distributed throughout the organs and tissues in concentrations that vary with local conditions (15). The highest concentration of asbestos fibres, outside the lungs, has been found in the kidney (high pressure and fast flow) and in the liver (high micro-vascular permeability); it is lower in the brain (owing to the blood-brain barrier) (16).
1. Renal tumours

- **Epidemiology**
Renal tumours occupy ninth place in the list of neoplasias commonly found in industrialised countries. They include carcinoma of the renal parenchyma (92%), carcinoma of the transition cells of the renal pelvis and ureter (7%) and Wilms' infancy tumour (1%) (17-19). Although often were considered as renal tumours, renal pelvis and ureter tumours are urothelial neoplasms, therefore they are affected by the same risk factors of bladder cancer, the most frequent urothelial malignancy. Roughly 80% of renal parenchymal tumours are clear-cell (non-papillary) adenocarcinomas, the remainder are papillary/chromophilic (~ 15%) or chromophobic (~ 5%) tumours and carcinomas of the collector ducts (< 1%) (18).

It is estimated that some 273,500 new cases of renal carcinoma are diagnosed every year in the world, accounting to 2% of all tumours (20). The highest incidence is recorded in North America, the lowest in Asia and Africa. In Europe 86,000 new cases are observed every year, 8,200 of them in Italy which occupies nineteenth place among the European countries, the incidence being considerably higher in men (5,600) than in women (2,600) (17, 21). As well as differences between continents, there are considerable differences within one and the same continent or even the same nation. In Europe, for example, 15.2 cases per 100,000 inhabitants are found in the Czech Republic and 2.9 in Serbia (22). In Italy the incidence of renal tumour observed in the north-east (9.6/100,000 men and 3.9/100,000 women) is three times higher than in a southern province like Salerno (3.6/100,000 men and 1.6/100,000 women) (23).

From 1973 to 2000 the incidence of tumours to the kidney and renal pelvis increased by 47% in men and 65% in women, a rate of about 2% per year (24). In the subsequent decade (1999-2008) this increase rose to 3% (2.8% men and 3.1% women) with 20.4 cases per 100,000 in the age bracket 50-54 years, 43.5 cases per 100,000 in that between 60 and 64 years, and 70.0 per 100,000 for the population between 75 and 79 years of age (25).

- **Situations, risk factors and exogenous agents**
We here list the agents and general factors, as well as occupational situations, which are likely to increase the risk of renal carcinoma in man, going by the epidemiological evidence.

- Smoking: it doubles the risk of carcinoma of the renal parenchyma, triples the risk of tumours of the renal pelvis. An increased risk has been observed to correlate with the number of cigarettes: 1.3-1.6 for a 20-40 packets/year consumption, 9.3 for consumption over 40 packets/year (26, 27).
- Obesity: the risk is thought to grow by 24% in men and 34% in women with each 5 kg/m² increase in the body mass (28).
- Hypertension: an increased dose-response risk has been observed, correlating with high blood pressure values (23).
- Diet: diets rich in fruit and vegetables correlate inversely with increased risk of renal cancer (29).
- Occupational situations: as recognised to date, the risk factors and agents mentioned above seem to be causally involved in the onset of about half the renal tumours observed (18). This lends all the greater importance to epidemiological and experimental evidence connecting occupational exposure to the aetiology of renal cancer. The agents most frequently associated with renal tumours in the literature are: 1,3-butadiene, vinyl chloride, vinylidene chloride, trichloroethylene, tetra-chloroethylene, aspartame and asbestos (23, 30-33).

- **Renal tumours in workers exposed to asbestos**
One of the experimentally and epidemiologically identified carcinogens for the kidney is asbestos. More and more experimental and epidemiological studies are now confirming the causal link between cancer of the kidney and occupational exposure to asbestos, as discovered back in the 1970s (34). One experimental study performed in 1976 on Wistar rats orally treated with a suspension of chrysotile showed an increase in the incidence of renal carcinoma (35). Medium-length asbestos fibres (chrysotile) were administered to Fisher F344 rats in their food on its own and with 1,2- dimethylxazine dichlorohydrate (DMH), a known carcinogen: among the females treated with asbestos a significant increase (P < 0.05) was noted in the incidence of mixed tumours of the kidney (34/175, 19%) as compared to DMH alone (13/125, 10%) (36). In a more recent study Wistar rats were treated with asbestos fibre (amosite) via intratracheal instillation. After six months’ treatment no neoplastic lesions were found in the kidneys, but significant glomerulosclerosis and interstitial tubulo-fibrosis were found (37).

Increased incidence of renal carcinoma was observed in a cohort study on 17,800 insulation workers in the United States and Canada over the period from 1967 to 1986 (38, 39). Here 37 deaths by renal neoplasm were recorded, a relative risk (RR) of 1.96 which is statistically significant (P < 0.01) against expectation (Table 1). A second epidemiological study on 1,074 American workers employed in various sectors including textiles and asbestos-cement-based products, showed a statistically significant increase in renal tumour deaths: 7 cases observed versus 2.54 expected, with a Standardized Mortality Ratio (SMR) of 2.76 (Confidence Interval (CI) 95%: 1.29-5.18) (40). Another cohort study on 1,500 workers exposed to asbestos brought to light malignancies of the kidney that the authors considered related with asbestos exposure (41). Yet another case-control study based on 518 cases spread over 37 hospitals in Massachusetts, as identified between 1981 and 1984, showed increased incidence of renal adenocarcinoma induced by asbestos (42). McCredie and Stewart’s case-control study in Australia evinced an RR of 1.62 (CI 95%: 1.04-2.53) for renal neoplasias, versus a sample of the general unexposed population (43). High incidence of kidney tumour was observed in workers with a cumulative exposure to asbestos of 300 mpcy (millions of particles per cubic foot per year) (44). In 1994 a high number of cases of renal tumour in Denmark were correlated with occupational exposure to asbestos (45). Another case-control study on workers exposed to asbestos and belonging to various job categories in vari-
ous countries (Australia, Denmark, Germany, Sweden and the USA) confirmed the significant increase in renal neoplasias (RR = 1.4; CI 95%: 1.1-1.8) (46). In 2000 Gamble and Lewis published the results of two studies on deaths among three cohorts of American refinery and petrochemical industry workers. The studies showed that renal tumour decease increased in all three cohorts. The relative risk (RR = 1.86) in one cohort, especially, was of statistical significance (47, 48).

2. Tumours of the bladder

➢ Epidemiology

Malignant tumours of the bladder (MTB) are the fourth neoplasia in order of frequency, above all in men where the ratio vis-a-vis women is 3.5:1. There are estimated to be around 200,000 new cases per year worldwide (49). The elderly are at greater risk than the young after the age of 70, the risk is about 30 times higher than in more youthful age brackets (21, 50).

According to recent data, MTB represents 4.8% of all tumours in the two sexes (6.9% in men and 2.5% in women) with a mortality rate of 3.4% and 1.5% in men and women respectively (50). In the United States the incidence tended to rise until 1990 in both sexes, but mortality is now diminishing especially among men (51). Over the period 2002-2006 the incidence among males in the United States was 37.1 per 100,000 inhabitants and 9.3 per 100,000 women. Over the same period mortality was 7.5 and 2.2 per 100,000 among men and women respectively (52). The highest rates were recorded in North America, North Africa, the Middle East and Europe - particularly Italy, Spain and French Switzerland with incidences higher than 30 per 100,000 men. The highest death rates were recorded in Denmark, Spain, Poland, Malta and Ireland. In Europe mortality increased up to the Eighties, above all in the South and East, and then tapered between 1988 and 2000 (53). In women the highest death rate found is around 2-3 per 100,000 in Denmark and Great Britain (54).

MTBs also reflect racial differences. For example, in the USA the Filipinos’ and Afro-Americans’ risk of developing a vesical carcinoma is respectively one-fifth and half that of whites. This finding with MTBs runs counter to the trend with other kinds of tumour where the incidence and mortality of Afro-Americans is always higher than among whites (55). In developed countries the incidence is twice as high in urban and industrialised areas as it is in the countryside. The differences are more marked among men than women. In the United States between 80 and 95% of MTBs are transition-cell carcinomas (56). The remaining malignant tumours are basically squamouscellular carcinomas and adenocarcinomas. Some authors think the percentage of squamouscellular carcinomas is growing steeply among blacks as compared to whites (12% vs 2%) and again, more in women than in men (18.3% vs 7.6%) (57).

The latency period of MTBs (the interval between start of exposure and onset of neoplasia pathology) varies according to type and intensity of exposure to factors and agents responsible. The latency period for cancer of the bladder ranges between 6 and 20 years, with a maximum of 45 years. The most commonly reported mean time is 20 years (58).

➢ Situations, risk factors and exogenous agents

Clinical-pathology findings from a hundred years ago (i.e. the historical dawn of bladder carcinogenesis) down to today’s epidemiological surveys and experimental research.
enable us to identify the exogenous carcinogen risks for the bladder.

We here list the agents and general factors, as well as occupational situations, which entail an adequately proven risk of bladder cancer in man, according to epidemiology (51, 59):

- Smoking: a relation has been found between the number of cigarettes smoked and the risk run.
- Schistosoma haematobium: the high incidence of vesical carcinoma among patients with schistosomiasis is a problem for some areas of Africa and the Middle East, especially Egypt.
- Occupational situations: colouring industry (aromatic amines and related agents), aluminium industry, coal distillation, combustion products from engines, shoe, furniture, rubber and textile industries.

**Tumours of the bladder in workers exposed to asbestos**

It is thought that a sizable part of MBs (5-25%) can be put down to occupational origin (56). There is mounting evidence of a causal link between tumours of the urinary apparatus, including bladder cancer, and occupational exposure to asbestos. This is hardly surprising if one thinks that many carcinogens, and especially their active biontransformation products, are excreted in the urine. The literature shows a heightened risk of MB in at least 40 different job categories, including paint-sprayers, truck drivers, electricians, mechanics, turners, barmen and waiters. A case-control study carried out in Spain between 1978 and 1982 examined 406 patients of the La Paz Hospital, Madrid, whose diagnosis of bladder cancer was associated with occupational exposure to asbestos (60). An increase in urinary tract tumours was observed in a German case-control study for 1977-1985 focusing on asbestos-exposed workers (Odds Ratio (OR) = 1.3, IC 95% = 0.7-2.5) from various categories, including foundries where the environment was heavily contaminat-ed with asbestos (OR = 1.6, IC 95% = 0.8-2.9) (61). An epidemiological study performed in Finland on 33 patients from a surgical clinic over the period October-December 1988, revealed that out of 28 MB sufferers, as many as 17 (61%) had been occupationally exposed to asbestos, and in 94% the exposure was certain (OR = 2.4, IC 95% = 0.9-8.4) (62). A cohort study on worker deaths at a shipyard in Genoa demonstrated an increase in MB in relation to asbestos fibre associated with aromatic polycyclic hydrocarbons, industrial oils and welding fumes. One notes especially that the bladder cancer latency among the workers involved proved to be about 25 years of continuous exposure, an important fact for follow-ups on people exposed (63). Another case-control study in four Canadian provinces evidenced a further confirmation of an association between MTB and exposure to certain chemical agents such as: asbestos (OR = 1.69, IC 95% = 1.07-2.65), mineral lubricant oils (OR = 1.64, IC 95% = 1.06-2.55), benzidine (OR = 2.20, IC 95% = 1.00-4.87) (64). The cohort study on workers at the Porto Torres petrochemical works in Sardinia (5,350 males were included in the follow-up) showed an increased risk of MB (RR = 1.46; 95% IC 1.09-1.96) in the sub-cohort of workers potentially exposed to asbestos (65).

To the best of our knowledge, the first study pointing to increased risk of MB dates from 1965, a survey on electrical industry workers which is a sector much polluted by asbestos – see the specific exposure scenario that follows – given the almost ubiquitous use of asbestos in electric-cables (66). This observation of electrical workers at increased risk of MTB was subsequently confirmed by many papers (67-69), including one that shows the combined results of 11 case-control studies (OR = 3.99, IC 95% = 1.10-14.51) (70). One Spanish study covering from 1998-2000 on 1,219 bladder cancer patients and 1,271 control patients found a statistically significant affect, all the more marked in exposures of ten years and more: the workers involved were from the medical gas-electricity sector (OR = 3.94, IC 95% = 1.49-10.44) and the electrical sector (OR = 1.31, IC 95% = 0.62-2.77) (71). The risk of MTB appears significantly greater when the exposure is a lasting one (over 10 years): this is confirmed by the study published by Cassidy et al. in 2009 (OR = 4.37, IC 95% = 1.62-11.77) and that by Colt et al. in 2011 which points to statistically significant increased risks run by car electricians (OR = 1.5, IC 95% = 1.02-2.3) and an increase among other electricians as well (OR = 1.1, IC 95% = 0.6-2.1) (72,73).

Several studies in the literature point to an increased risk of MTB in foundry workers, another category heavily exposed to asbestos, as described in Case 1 below. Claude et al. published a case-control study on 531 urinary tract patients from a range of different jobs including foundries (OR = 1.56, IC 95% = 0.84-2.91) as well as thermo-electric power stations and kilns (OR = 2.17, IC 95% = 0.84-5.57) (69).

**The Ramazzini Institute caseload on urinary apparatus tumours and asbestos**

In 1995 Professor Cesare Malloni and his group at the Ramazzini Institute first encountered a renal tumour in a worker occupationally exposed to asbestos. In a quite random way since then our attention has been drawn to another 22 cases, all men (the most gender exposed in the past) who had tumours of the urinary apparatus. The 23 cases include 18 kidney cancers, 2 of the ureter, and 3 of the bladder, traceable to asbestos exposure at work in various exposure scenarios recounted at first hand. In what follows we present an account of these cases in the order of presentation. The data are summarised in Table 2.

Appendix with individual case descriptions is available at http://architrulo.it/index.php/iit/supplements.

**Discussion and conclusions**

Tumours of the urinary apparatus are on the increase in the most industrialised countries and regions. The many epidemiological and experimental studies show that many agents can cause such tumours, and asbestos is emerging as one of them.

Asbestos is the generic name for a series of natural fibrous silicates which are widespread in nature and were extensively used in the last century in a range of manufacturing sectors: building, railways, shipyards, textile firms, the
Table 2.
Asbestos-related tumours of the urinary system: the Ramazzini Institute caseload.

<table>
<thead>
<tr>
<th>Case N.</th>
<th>Patient initials</th>
<th>Place of work</th>
<th>Exposure scenario: exposure to asbestos</th>
<th>Period of exposure (length)</th>
<th>Diagnosis</th>
<th>Year of diagnosis</th>
<th>Age at diagnosis</th>
<th>Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>MZ</td>
<td>BO, MO</td>
<td>Foundry: thermal and electrical insulation of kilns, protective gear</td>
<td>1964-1991 (27)</td>
<td>Renal carcinoma</td>
<td>1990</td>
<td>46</td>
<td>26</td>
</tr>
<tr>
<td>2</td>
<td>PN</td>
<td>PD</td>
<td>OFFICE STANGA (Firema): lining and insulation of railway carriages</td>
<td>1969-1998 (29)</td>
<td>Renal oncocytoma, asbestosis</td>
<td>1999 (48)</td>
<td>51</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>MM</td>
<td>BO</td>
<td>State railways: lining and insulation of railway carriages</td>
<td>1941-1974 (33)</td>
<td>Renal carcinoma</td>
<td>1994</td>
<td>70</td>
<td>53</td>
</tr>
<tr>
<td>5</td>
<td>GS</td>
<td>BO</td>
<td>State railways: lining and insulation of railway carriages</td>
<td>-</td>
<td>Renal carcinoma</td>
<td>1988</td>
<td>72</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>GP</td>
<td>BO</td>
<td>Casaralta Componenti (Firema): lining and insulation of railway carriages</td>
<td>1977-1987 (10)</td>
<td>Renal carcinoma</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>SE</td>
<td>NA</td>
<td>Metalworking firm: lining and insulation of railway carriages</td>
<td>-</td>
<td>Renal neoplasia</td>
<td>1985</td>
<td>79</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>CN</td>
<td>BO</td>
<td>Sugar refinery (home exposure): lining and insulation</td>
<td>1938-1960 (22)</td>
<td>Renal carcinoma</td>
<td>1995</td>
<td>59</td>
<td>57</td>
</tr>
<tr>
<td>20</td>
<td>EB</td>
<td>MO</td>
<td>Magneti Marelli: thermal and electric-cable insulation for kilns, protective gear</td>
<td>1974-2012 (38)</td>
<td>Renal carcinoma</td>
<td>2001</td>
<td>49</td>
<td>27</td>
</tr>
<tr>
<td>23</td>
<td>DG</td>
<td>FE</td>
<td>Magneti Marelli: lining, insulation, thermal and electric-cable insulation for kilns, protective gear</td>
<td>1980-present (32)</td>
<td>Bladder carcinoma</td>
<td>2008</td>
<td>52</td>
<td>28</td>
</tr>
</tbody>
</table>

Ceramics industry, brickworks, foundries and sugar refineries. Its main uses are as: 1) asbestos-cement for building materials (roofing, walls, panels, ceilings, floors, conduits), pipelines (aqueducts, oil lines), basins and tanks; 2) insulating material (heat, electricity, sound) in various types of building (factories, public buildings, homes) and in pipes, boilers, vehicles, rolling stock, ships, etc.; 3) friction parts (brakes); 4) paper and board; 5) textiles; 6) plastics; 7) gaskets; 8) filters. Apart from these main uses, asbestos has been employed in innumerable ways. One recent estimate is that about 3,000 uses have been listed (4).
In Italy about 75% of asbestoses went into asbestos-cement. The remaining 25% was almost entirely used in the textile sector, gaskets, paper and board. Asbestos spread more or less everywhere because of its multiple properties: resistance, non-flammability, insulation, durability, low cost. Such qualities made it non-degradable. It lingers indefinitely in the environment; once it penetrates the organism, it is partly retained by tissues along its route. Its carcinogenic effects on the human organism are irreversible, all the evidence suggests. Again, nowadays asbestoses, though ubiquitous, is at its highest and most dangerous concentration in certain work and living conditions. It is difficult to list all the work conditions at risk. Exposure to asbestoses occurs to: those who extract it, those who make asbestos-based products, those who handle such materials, repairers and demolishers of asbestos-containing materials, port stevedores, transporters and all whose work environment is polluted by asbestoses. One occupational category thought to be most exposed and at the greatest carcinogenic risk from asbestoses are the technicians making, checking, cleaning and demolishing rolling stock containing asbestoses. In Italy asbestoses was extensively used by the railways from the 1930s to the 1970s in the form of panels and lagging strips for various parts of locomotives, infrastructure, and also making asbestos-cement sleepers for rail-track. From the places where it is employed, movement, wear and tear and vibration spread it inside rolling stock, in depots and in outdoor and indoor repair workshops, in and around railway stations, and also in workers’ homes, since they carry asbestoses fibre on their hair and work clothes. Another work category at high risk of asbestoses-related cancer is that of dockers heavily and permanently exposed as large quantities of the material are shifted, loaded, unloaded and stored. At least until the late-Eighties, the lack of collective or individual protection measures, as well as lack of information about the risk of inhaling the fibres, further increased the degree of environmental exposure.

The IARC has classified asbestoses as a carcinogen for man (Group 1) and states that it gives rise to mesothelioma, lung, larynx and ovary tumour and – somewhat less proven – tumours of the pharynx, stomach and colon-rectum (74). INAIL sees neoplasias of the lung, pleura, peritoneum, pericardium and the tunica vaginalis testis as highly attributable to asbestoses exposure, and (to a lesser degree of probability) neoplasias of the larynx and gastrointestinal tract.

Greater controversy still surrounds the aetiological role of asbestoses in tumours of the urinary apparatus, despite the existence of wide scientific documentation of a causal link (not least, the finding of asbestoses fibres in exposed workers’ urine and kidneys). The 23 cases from the RI caseload here published give grounds for thought on various counts. First, they comprise workers whose asbestoses exposure was certain and recognised: many of them worked directly or indirectly on the railways, or were dockers, shipyard workers, foundry workers or from sugar refineries where the massive asbestoses pollution is proven. Among them are cases recognised by INAIL as heavily and continuously exposed to asbestoses, or suffering from asbestosis which is a specific marker for asbestoses exposure. The second reason is the length of exposure and time of latency (the period from when exposure starts to when the neoplasia sets in): respectively 26.2 years (range 7-57) and 33.1 years (range 16-57). Such time-frames are most frequently observed in occupational tumours and may be seen as expressing the high environmental oncogenic potential to which the workers were exposed. Again, the early mean age at onset (59.6 years: range 37-79) points to an occupational origin. The 43.5% of these tumours set in below 55 years of age, 10 years earlier than the incidence rates observed in men over the period 2006-2008 (20).

Lastly, the RI caseload came to our notice quite randomly and is not the result of a systematic epidemiological survey detailing the exposure scenario of the worker categories involved: if anything, it underestimates the situation. Let us not forget that it took decades before official recognition was given to pleural mesothelioma – nowadays considered the most specific asbestoses-related tumour. Bearing in mind the wide range of risk categories and the picture painted by the scientific literature, we urgently need to implement measures to protect workers and their families, as well as the population at large.

Nationwide and internationally, it is to be hoped that new epidemiological efforts will be made to quantify the global cancer risk from asbestoses – especially in view of all the “unexpected” malignancies that have occurred – and improve prevention strategies as well as obtaining legislative recognition of the occupational origin behind these neoplasias. Greater light might be shed on this if the categories exposed to asbestoses were regularly monitored by oncologists – a project the Ramazzini Institute has in mind to undertake, responding to pressure from workers and as a way of standing by them in this difficult phase.

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