ASBESTOS AS A MODERN URBAN HAZARD

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A preliminary report by one of us has been published on the association between mesothelioma of pleura or peritoneum and a limited pulmonary asbestosis, confined to the basal 1 cm. or so of the lower lobes, in people who had no history of occupational or industrial exposure to asbestos.[1] It was stressed that these cases had no clinical or radiological evidence of asbestosis, and that the limited basal asbestosis was usually missed at autopsy. Even if the lungs were examined microscopically the local asbestosis would again be missed unless sections were taken from the bases of the lower lobes. The association was regarded as an aetiological one in that there was locally an asbestosis of carcinogenic concentration, effective on the pleura or on the peritoneum, which is no more than a few millimetres distant over the diaphragm. No bronchi of any size are present in this thin strip of basal lung, only bronchioles without glands; and consequently a bronchial carcinoma, the more usual result in classical industrial asbestosis, is not to be expected.

Asbestos fibres in the lung, or some of them, are converted into asbestos bodies by the deposition of yellow globules of protein and iron, producing an easily recognized and quite characteristic structure. But before this happens many, if not most, of the very thin and sharp asbestos fibres tend to move down towards the bases, owing to lung movements and gravity. Once the fibre has become an asbestos body it is unlikely to move further; it is too large to be transported by lymphatics and its sharp points are now blunted by the protein globules.

The enormously increased world consumption of asbestos, from 300,000 tons in 1934 to 2,400,000 tons in 1961, is used in a wide variety of industrial products, from obvious ones such as asbestos roofing, roof tiles, ceilings, floor tiles, pipe insulation and electrical insulation, to less obvious ones where asbestos is added to all sorts of plastic materials, cement, etc. It is possible for those manufacturing, selling and using these products to inhale asbestos fibres, although their occupations are in general not labelled or recognized as ones in which asbestos is a hazard to be anticipated. We do not know what percentage of the world's consumption of asbestos is used in factories other than those labelled asbestos factories, but suspect that it is becoming more and more significant.

Further, it is possible that the inhalation of a relatively small number of asbestos fibres over a long period, several years or indeed a lifetime, may result in a focal concentration at the lung bases, in people who have no occupational risk from asbestos, merely because asbestos is mainly used in towns and cities and is a likely contaminant of the air.

We were unable to find any figures in the literature on the frequency of asbestos bodies in the lungs in routine autopsies, and in 1960 an investigation was started to see if the inhalation of asbestos was indeed an urban hazard, and to try and establish some sort of standard frequency for comparison with any change in the future.

MATERIALS AND METHODS

From each lung a smear was made on a 3 x 1 inch (7·5 by 2·5 cm.) microscope slide, usually consisting mainly of blood, and quite thick. This was air-dried, flooded with xylol, and mounted with balsam or balsam substitute under a large coverglass. Initially the smears were made from apex to base of lung, but the majority were from the bases of the lower lobes only. The golden-yellow asbestos bodies were readily seen with the low-power of the microscope (16 mm. objective), and confirmed by the 4 mm. objective. Counting (on one slide only) was done by low-power fields, and in those recorded as negative 300 low-power fields were examined. Rarely we saw a small black carbon particle with a yellow coating, usually circular in form and readily distinguished from the true asbestos body, and nothing was seen in these smears giving any real resemblance to a genuine asbestos body.

Smears were taken from consecutive autopsies from the Groote Schuur Hospital, Cape Town, on subjects aged 15 years and over to a total of 500, of which 60% were males.

RESULTS

The method used to demonstrate these asbestos bodies is subject to certain variables, the most important of which is the extent to which pulmonary oedema is present. When it is marked enough, fluid from a small number of air vesicles is sufficient to cover the slide, and a severe degree of basal pulmonary congestion will have a similar but less marked effect. None of the lungs thus examined was from medicolegal autopsies (many of these were for people dying suddenly), and few lungs in this series were without some pulmonary oedema and congestion. The amount of lung thus examined for asbestos bodies was trifling, and the positive findings should be assessed in that light. We suspect that the percentage of lungs showing these asbestos bodies could be brought to much higher figures if larger samples of lung juice were examined. Even so, the percentage positive is surprisingly high, and we can regard our suspicions that inhalation of asbestos is a hazard of modern urban life as having been confirmed.

Over one-quarter of the lungs of 500 consecutive autopsies on subjects of 15 years and over showed asbestos bodies by the method used (26·4% — 132 cases) — a much higher figure than anticipated when the investigation was started. We expected a higher incidence in the male, and this was found (30%), but the proportion of females whose lungs were positive was also strikingly high (20%) (Table I).

Further differences were evident in the three racial groups in Cape Town, from 16% in the Whites to 58% in the Africans, with the Coloureds occupying an intermediate position with 23·7% of autopsies positive for asbestos bodies. In males only (306 cases), these racial differences in frequency were more evident — Whites 17·9%, Coloureds 29·2% and Africans 61·7%.

In both sexes and in all three racial groups there is the anticipated rise in frequency with age (Table II), ranging from one positive in the under-25-years group (4·3%) to approximately 30% in those over 65 years of age. One surprising feature is a lower frequency in the 45 - 54 year age group, present in both sexes, as shown in Fig. 1.

TABLE I. THE FREQUENCY OF ASBESTOS BODIES IN BASAL LUNG SMEARS IN THE THREE RACIAL GROUPS

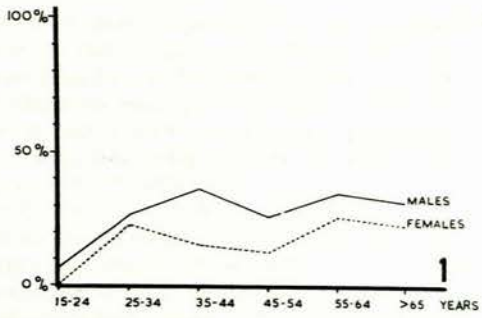
	Whites			*Coloureds*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
Cases examined	140	84	224	106	84	190
Cases positive	17·9% (25)	14·3% (12)	16·5% (37)	29·2% (31)	16·7% (14)	23·7% (45)

	Africans			*All races*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
Cases examined	60	26	86	306	194	500
Cases positive	61·7% (37)	50% (13)	58·1% (50)	30·4% (93)	20·0% (39)	26·4% (132)

TABLE II. THE FREQUENCY OF ASBESTOS BODIES IN BASAL LUNG SMEARS IN AGE GROUPS

Age groups (years)		*Whites*			*Coloureds*		
		Male	*Female*	*Total*	*Male*	*Female*	*Total*
15—24	Positive	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
	Negative	100% (4)	100% (2)	100% (6)	100% (7)	100% (5)	100% (12)
25—34	Positive	28·6% (2)	33·3% (2)	30·8% (4)	13·3% (2)	12·5% (1)	13·0% (3)
	Negative	71·4% (5)	66·7% (4)	69·2% (9)	86·7% (13)	87·5% (7)	87·0% (20)
35—44	Positive	22·2% (2)	0% (0)	13·3% (2)	38·2% (8)	9·5% (2)	23·8% (10)
	Negative	77·8% (7)	100% (6)	86·7% (13)	61·8% (13)	90·5% (19)	76·2% (32)
45—54	Positive	9·1% (3)	11·1% (1)	9·5% (4)	20·0% (5)	6·7% (1)	15·0% (6)
	Negative	90·9% (30)	88·9% (8)	90·5% (38)	80·0% (20)	93·3% (14)	85·0% (34)
55—64	Positive	19·4% (7)	16% (4)	18·0% (11)	36·8% (7)	29·4% (5)	33·3% (12)
	Negative	80·6% (29)	84% (21)	82·0% (50)	63·2% (12)	70·6% (12)	66·7% (24)
Over 65	Positive	21·6% (11)	13·9% (5)	20·8% (16)	47·4% (9)	27·8% (5)	37·8% (14)
	Negative	78·4% (40)	86·1% (31)	79·2% (71)	52·6% (10)	72·2% (13)	62·2% (23)

Age groups (years)		*Africans*			*All races*		
		Male	*Female*	*Total*	*Male*	*Female*	*Total*
15—24	Positive	25·0% (1)	0% (0)	20·0% (1)	6·7% (1)	0% (0)	4·3% (1)
	Negative	75·0% (3)	100% (1)	80·0% (4)	93·3% (14)	100% (8)	95·6% (22)
25—34	Positive	50% (4)	33·3% (1)	45·5% (5)	26·6% (8)	23·5% (4)	25·6% (12)
	Negative	50% (4)	66·7% (2)	54·5% (6)	73·4% (22)	76·5% (13)	74·4% (35)
35—44	Positive	43·8% (7)	60·0% (3)	47·6% (10)	37·1% (17)	15·6% (5)	28·2% (22)
	Negative	56·2% (9)	40·0% (2)	52·4% (11)	62·9% (29)	84·4% (27)	71·8% (56)
45—54	Positive	78·6% (11)	28·6% (2)	61·9% (13)	26·4% (19)	12·9% (4)	22·4% (23)
	Negative	21·4% (3)	71·4% (5)	38·1% (8)	73·6% (53)	87·1% (27)	77·6% (80)
55—64	Positive	77·0% (10)	57·2% (4)	70·0% (14)	35·3% (24)	26·5% (13)	31·6% (37)
	Negative	23·0% (3)	42·8% (3)	30·0% (6)	64·7% (44)	73·5% (36)	68·4% (80)
Over 65	Positive	80·0% (4)	100% (3)	87·5% (7)	32·0% (24)	22·8% (13)	28·0% (37)
	Negative	20·0% (1)	0% (0)	12·5% (1)	68·0% (51)	77·2% (44)	72·0% (95)



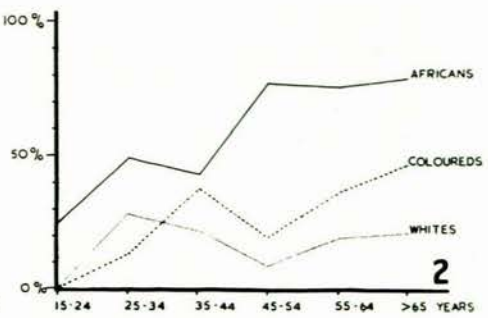


Fig. 1. Percentage of cases positive for asbestos bodies in age groups (all races).
Fig. 2. Percentage of cases positive for asbestos bodies in age groups (males only).

In males only (306 cases) this is striking in Whites and Coloureds, but in Africans the drop appears one decade earlier (Fig. 2), and is based on smaller numbers. The appearances suggest that there are two curves: that of the young which, if extrapolated, would reach 100% by the age of 60, and that of those two decades older who were less exposed to the inhalation of asbestos fibres in their youth and avoided the undue exposure from 45 years onwards. Otherwise we would have to presume that after the age of 35 a significant number of formed asbestos bodies lose their encrustation of protein and iron and return to the state of asbestos fibres, in which form they would not be seen or recognized in smears. Another possibility, just as difficult to establish or to refute, is that in the older age groups there is a less marked or slower response to the presence of asbestos fibres, and a smaller proportion of those inhaled is converted into asbestos bodies.

There can be little doubt that those under the age of 35 have had better opportunities of inhaling asbestos fibres than the older age groups in their first 35 years, owing to the increased and more widespread use of asbestos. The world consumption of asbestos 35 years ago was less than one-eighth of what it is today, and the houses and public buildings in Cape Town generally contained relatively little asbestos. The city has since more than doubled in size and population, and the new buildings, public and private, contain much larger quantities of asbestos in various forms. In addition, much of the industry using asbestos in this area is of new growth or recent development, as it is in almost every part of the westernized world, in conformity with the marked increase in world consumption of asbestos in the last 30 years.

It is not difficult to suggest reasons why those of 45 years and older at death did not inhale as many fibres as did those in the younger age groups in the same period of time. A higher proportion of these older people would not be employed in occupations involving hard physical work as is required in the manufacture, handling or use of many asbestos-containing products, more would be in administrative posts, and some would be retired.

What are the possible means by which asbestos fibres are inhaled to produce the asbestos bodies? Firstly, we have those employed in asbestos factories or, in South Africa at least, as pointed out recently by Wagner *et al.*,[2] those who work in asbestos mines or who live in the vicinity of the mines or where the asbestos from the mines is treated or handled. This is of necessity a restricted group. Secondly, we have those who may be exposed to asbestos fibres in factories not designated asbestos factories, where asbestos is used. These constitute a surprisingly large and ever-increasing group, such as cement works (asbestos cement), and all the factories where asbestos is used in the manufacture of asbestos roofing, tiles, millboard, friction materials such as brake linings, ceiling boards, insulation and packing for pipes and boilers, for electrical insulation, and the many other ways in which asbestos is employed as an addition to all sorts of plastic materials. A much larger group of people who deal with or handle such materials may be similarly exposed to varying degrees, ranging from the occupational exposure of builders and builders' labourers to the handyman in his home workshop. For example, a garage worker would not ordinarily be regarded as a candidate for asbestosis of any degree, yet Brugsch and Bavley[3] reported recently pulmonary asbestosis in a garage worker employed in spraying underbody coating containing 50% of asbestos. We suspect that the number of people in a modern urban community who are thus theoretically or practically liable to inhale asbestos fibres is a very

TABLE III. THE CONCENTRATION OF ASBESTOS BODIES IN BASAL LUNG SMEARS

	Whites			*Coloureds*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
1 Asbestos body in 31–300 L.P.Fs.	16	9	25	21	10	31
1 Asbestos body in 10–30 L.P.Fs.	4	2	6	4	1	5
1 Asbestos body in 2–9 L.P.Fs.	4	1	5	3	3	6
More than 1 asbestos body per 2 L.P.Fs.	1	0	1	3	0	3
	Africans			*All races*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
1 Asbestos body in 31–300 L.P.Fs.	21	11	32	58	30	88
1 Asbestos body in 10–30 L.P.Fs.	9	2	11	17	5	22
1 Asbestos body in 2–9 L.P.Fs.	5	0	5	12	4	16
More than 1 asbestos body per 2 L.P.Fs.	2	0	2	6	0	6

L.P.Fs. = Low-power fields.

significant percentage of the population, but cannot even put forward a guess as to what the figure is.

Lastly, we have those with employments not obviously associated with asbestos. These constitute presumably the largest group of all, yet the results of this investigation clearly indicate that they also inhale asbestos fibres.

To try and place our positive cases into these three groups would appear an impossible task, since details of occupation are meagre, and in any case many of the Coloureds and Africans have had varied occupations and only the last is noted. But in Cape Town the occupations of the three racial groups are restricted to a greater extent than in most cities. There are virtually no labourers who are White, and the occupations of the African males have a relatively narrow range; those of African females are narrower still.

A crude assessment of the relative number of asbestos bodies is given in Table III. It should be stressed that these are quantitative only to the bases of the lungs and not to the lungs as a whole. In the great majority, over 80% of the positive cases, the bodies were scanty, one body in 10 - 300 low-power fields. In 22 cases, 4·4% of those examined and 6% of the males, the asbestos bodies were numerous, from more than one per low-power field to 1 in 9 low-power fields. These are likely to be cases in which industrial or occupational exposure to asbestos has been present, and if that inference is correct, it is disturbing that 1 in 17 of the males of the hospital population of Cape Town of 15 years and over should be thus affected. The admissions to the Groote Schuur Hospital are fully representative of all ethnic groups in Cape Town, but not perhaps of all social groups in the Whites. In Cape Town African females are rarely employed except as domestic servants, and, as expected, none are found in the group with abundant asbestos bodies. Only 4 were females, while 18 were males, a finding again in favour of occupational exposure as the explanation.

Unfortunately tissue for microscopical examination was not taken as a routine from the bases of the lungs, and a precise correlation between the presence of asbestos bodies and a limited basal asbestosis is not possible. But in all cases sections from the lungs were available for study, and 3 examples of ordinary classical asbestosis were found, 1 mild and 2 of moderate severity. In only 1 of these was a history of exposure to asbestos obtained, one year's employment in an asbestos factory 15 years before his death from lobar pneumonia, and that was obtained from relatives as a result of persistent enquiry. In 2 further cases sections had been taken from the lung bases and both showed a limited basal asbestosis with bilateral basal pleural thickening, but the lungs elsewhere were free from fibrosis, and showed very scanty asbestos bodies. Of the remaining 17 cases in this group we do not know how many would have shown this basal asbestosis had sections from the lung bases been available for histological examination. All one can say is that in the sections of lung taken at autopsy from areas other than the bases, asbestos bodies, usually scanty, were demonstrated in 9, but in none was there fibrosis or pulmonary asbestosis. The fact that lung smears from these 17 cases showed asbestos bodies to be as numerous as in the smears from 3 cases of classical asbestosis and 2 of basal asbestosis, leads us to suspect that most would have shown the basal asbestosis had sections been taken from the lung bases.

We find it difficult to believe that the majority of those with abundant asbestos bodies worked in asbestos factories, though no investigation of their occupations during their lifetime was undertaken. Only 1 was a builder, and no other of the occupations given indicated clearly the possibility of exposure to asbestos. We presume that most of them at some time or other came into the second group, and had contact with asbestos or asbestos-containing products in some of the varied occupations already mentioned — a conclusion that is significant enough. If they were not so exposed occupationally, the findings are even more significant, and even more disturbing, though it should be emphasized that only in the 2 cases of classical asbestosis of moderate severity pulmonary disability was present, and in none of the others were the pulmonary lesions in any way responsible for death.

But perhaps the most significant finding is the large proportion of cases in which fewer asbestos bodies were demonstrated — 1 in 4 of all the remaining autopsies. In most of these the bodies were scanty, but quite characteristic, and we wish to stress that there are no grounds for regarding these as pseudo-asbestos bodies merely because their presence may be regarded as difficult to explain. We do not think that there is any difficulty in explaining their presence, and indeed would go so far as to say that their absence would be the more remarkable finding in the average urban dweller of adult age in westernized communities. In recent years the term 'asbestos body' has been altered by some to 'asbestosis body', an unfortunate and unwarranted change, because the great majority of lungs which show asbestos bodies have no pulmonary asbestosis at all, as these findings clearly demonstrate.

The varying frequencies in the three racial groups and the two sexes give some indication of the means by which the asbestos fibres are inhaled. The overall positive figures for the sexes are 30% male to 20% female. If we exclude those with abundant asbestos bodies as being the result of occupational exposure, the resulting figures show little variation between the sexes in Whites and Africans: White males 14·8%, White females 13·3%; African males 57%, African females 50%, but the difference in the Coloureds is increased; Coloured males 25%, Coloured females 13·6%.

We find almost identical figures for the Whites, male and female, and the female Coloureds — 14·8%, 13·3% and 13·6% respectively. Are we justified in presuming that this is the environmental base-line in Cape Town, not resulting from occupation, but determined entirely by asbestos fibres in the air? The numbers on which these figures are based are not large, but together are significantly lower than the other 3 groups with higher frequencies ($P = < 0{\cdot}01$). And what are the explanations of the much higher rates in Coloured males and Africans of both sexes? Occupations with possible exposure to asbestos, such as building, may account for both the male groups, but not for the African females. Their environment, largely restricted to native locations, is the only explanation left to us.

If we assume, as is likely, that most of those with scanty asbestos bodies did not develop them as a result of their employment, we are left with the home and the air of

the city as the only possible sources for the asbestos fibres.

In the modern home there is no lack of theoretical possibilities. Asbestos roofing or roof tiles, ceilings and floor tiles are common enough, and the last at least must be subjected to wear and liberation of asbestos fibres. Asbestos-insulated central heating pipes, so common in the northern hemisphere, are virtually absent in Cape Town homes, but the asbestos-insulating pad in domestic irons is almost universal and usually is made in a soft, friable and readily disintegrating form. But under ordinary conditions it would seem unlikely that many asbestos fibres are inhaled in the average home. The air of the street seems a more probable source. The average motor vehicle wears out 3 or 4 sets of brake linings in its lifetime, and they consist mainly of asbestos, which is ground to dust as the linings wear. This alone would involve in most cities the discharge of tons of asbestos dust in the streets every year. The disintegration by natural weathering of asbestos-containing products from car underbody coating to asbestos cement roofing must again result in asbestos fibres getting in the air of towns and cities.

But perhaps the most important factor is that asbestos is virtually indestructible. It resists heat, alkalis, acids and other means by which materials are altered chemically or physically, and mechanical disintegration merely results in the production of finer fibres, more liable to be inhaled, and more dangerous when inhaled than larger fibres. However one assesses the hazard of inhaling asbestos fibres, as it exists today, as a trifling matter or as a potentially significant one, it must become greater with the years. The half-life of strontium 90 is 28 years, but the half-life of the asbestos fibre is an infinity of years. The 2,400,000 tons of asbestos used each year are added to the millions of tons used in previous years, and even if a small proportion becomes available as an air-contaminant in towns, the actual amount will increase, as more asbestos accumulates on the surface of the earth. The natural weathering and disintegration of some asbestos-containing products is likely to be very slow, but many are relatively new, and may not be as long-lasting as expected. We can only guess what proportion of air-borne asbestos fibres is rendered innocuous by dispersal or removed by fresh or salt water, but whatever the proportion, it is likely to be one that is more or less fixed, and we do not think that the total amount of air-borne asbestos fibres in cities will do other than increase.

We do not know of any published figures from other parts of the world with which our findings can be compared, but the high frequency of asbestos bodies in routine autopsies is not confined to Cape Town. One of us initiated a similar investigation in 1961 in Miami, Florida, USA, and even higher positive figures are being obtained. This is almost to be expected since proportionately more of that city is of recent growth with a high asbestos content of the buildings, and the United States accounts for roughly over a third of the world's consumption of asbestos. We would suspect that even higher figures would be obtained in Los Angeles.

It is all too easy to go to extremes in discussing these findings. They could be dismissed as a pathological curiosity of no practical significance, supported by the fact that in only 3 of the 132 cases positive for asbestos bodies was there associated pulmonary fibrosis and disability, and in none was there an associated mesothelioma of pleura or peritoneum. But 5 such cases were seen in the same autopsy service in the previous 3 years, and, going to the other extreme, a lurid picture could be painted of what conditions might be in 30 years' time, when the accumulation of so much asbestos in towns would lead to much higher concentrations of asbestos fibres in the air. The results of life-long inhalations of these fibres might be almost universal basal asbestosis in adults, and a frequency of mesothelioma of pleura or peritoneum higher than that of bronchial carcinoma today. It could be added that even if the hazard were then appreciated, and all mining and utilization of asbestos ceased, there might be no significant reduction of the air contamination, owing to the indestructibility of the asbestos already mined and used, and its inevitable weathering and disintegration with time.

The present findings clearly demonstrate that asbestos fibres are being inhaled by urban dwellers, but not in amounts sufficient to produce pulmonary lesions or disability. In 6% of hospital autopsies in males aged 15 years and over, a high basal concentration of asbestos bodies is demonstrated, suggesting that industrial exposure to asbestos is more widespread than expected. It would seem almost inevitable that in the future the urban dweller will be exposed to a higher concentration of asbestos fibres. We do not anticipate that this will lead to an increase in classical pulmonary asbestosis, but more cases of basal asbestosis would seem almost inevitable. Indeed, even if there is no significant increase in the amount of asbestos fibres in the air, the younger urban dweller will have a lifetime's exposure available for concentration at the lung bases, unlike most of those of over 50 years of age today, who, in their earlier years, had little opportunity of inhaling asbestos fibres. If we are correct in regarding basal asbestosis as the main aetiological factor in mesothelioma of pleura or peritoneum,[1] an increase in this tumour would also follow. This possibility is the main justification for the title of this paper.

SUMMARY

In smears from lung bases from 500 consecutive autopsies in subjects of 15 years or over, asbestos bodies were found in 30% of the males and 20% of the females.

In most cases this would appear to be the result of contamination of the urban atmosphere, and the bodies were scanty and not associated with pulmonary changes.

In 6% of the males the bodies were numerous and were presumably of occupational origin.

An increase in the amount of urban air contamination would seem inevitable in view of the increasing consumption and diversity of uses of asbestos, and of its virtual indestructibility.

The tendency for asbestos fibres to concentrate in the bases of the lower lobes of the lungs produces a cumulative effect, and an increase in the frequency of basal asbestosis is probable.

This basal asbestosis does not result in pulmonary disease or disability, but appears to be of aetiological significance in mesothelioma of pleura or peritoneum. An increase in this tumour is forecast, and this is suggested as the main ground for regarding asbestos as an urban hazard.

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