Editorial

Anthracosis in Iran, Un-answered Questions

See the pages: 128 – 130

Cite this article as: Heidarnazhad H. Anthracosis in Iran, Un-answered Questions. *Arch Iran Med*. 2012; **15(3):** 124 – 127.

During the last few years, anthracosis and anthraco-tuberculosis have found great interest between pulmonologists of Iran and some other parts of the world as a newly emerging medical condition associated with high tuberculous burden. A recent report from World Health Organization (WHO) consider indoor air pollution from old fashion household cooking fires as the most important environmental cause of mortality and morbidity which is the etiology of at least 2 million deaths across the world each year.[1] This primitive household cooking which uses biomass (dung, wood, charcoal and crop residues) results in old fashion homes filled with dense smoke, blacking walls and ceilings and sickening women and children in low income countries. The risk of these types of exposures is equal to lifelong smokers of tobacco and result in annual deaths of 872,000 children under age 5 from acute lower respiratory infections , 1,057,000 adults from COPD, 36,000 adults from lung and upper respiratory cancers.[2,3]

Pazoki et al. work about anthracosis and tuberculosis in this issue of Archives of Iranian Medicine, is 17th article published by Iranians either in local and international journals.[4–20] (see table one for summary of these works). We have other related publications from other parts of the world.[21–29]

We are facing with increasing number of anthracosis from simple mucosal to severe anthracofibrosis with or without associated tuberculosis. Since the introduction of fiberoptic bronchoscopy and availability of respiratory specialist all over the country, what we have seen 25 – 30 years ago in coal miners or household rural baker women, now is reported with increasing frequency from various states of Iran even in peoples without evidence of occupational exposure. Carbon is essential element of combustion of biomasses and fossil fuels (e.g. diesel exhaust) and increasing prevalence of anthracosis especially in air polluted and dirty areas of the world is related to air quality.[30] Studies on carbon deposition and clearance have showed that if the quantity of deposition became more than the clearance capacity of the lungs, carbon particle retained in the alveoli and transferred to lymphatics with some fibrotic reaction and may distribute through the lymphatics to other organs.[31–35] Surprisingly, in smokers, due to increased mucociliary clearance, the deposition of carbon was less than non-smokers.[36] The role of air pollution in producing anthracosis regardless of human being[27] has been documented in animals, which have no occupational exposure, no traditional bakery, and no smoking history. In Dhaka zoo collections, Ahasan et al in necropsy of animals have showed that between 36 samples of 24 different species, 27 cases had anthracosis, which was related to Dhaka city air pollution.[37] In another study by Beytut from Turkey, the presence of anthracosis was also established in 2.25% of 2000 slaughtered sheeps.[38]

During last 30 years, we have witnessed a dramatic change in socioeconomic status of low income people in Iran with improve housing and special replacement of natural gas as the most common source of energy for cooking and warming across the country which include almost all urban population and a significant portion of rural population. This has resulted in significant reduction in indoor air pollution from old fashion household cooking fires.[39] This event coincided with industrialization of our country and increasing numbers of car which is now the main source of air pollution. This will affect both the old and the new generation of people living and working in Iran and other low and middle income countries.So the increasing prevalence of this kind of deposition disease is related to increasing air pollution and industrialization of our country. We should anticipate its adverse effect on our children's health who are living in polluted areas with vulnerable lungs and what will happen in coming decades.

Studies in anthracosis have shown a spectrum of disease from simple mucosal anthracosis to advanced fibrotic disease with distortion of bronchi associated with mild to severe ventilatory disturbance of obstructive, restrictive, or mixed types. Regarding published studies, the small amounts of carbon particles have no fibrogenic effect, but large ones may give rise to fibrosis especially in lymphatics and nodes and this may distort the lung architecture. But even in these cases the role of other particles and minerals should not be ignored. Carbon particles are rarely pure and usually combined with various minerals and metals like iron, nickel, silica and mica. Thus, the fibrosis which occurs in some patients may be related to mixed particles inhalation. No study from Iran answered this question, but scattered reports especially in rural bakery women from Africa, Latin/South America and India[40–42] have revealed the nature of particles. Some of our patient·s clinical and radiologic characteristics are similar to these cases, so we cannot rule out the role of mixed dust inhalation. We watch only the black color of carbon at bronchoscopy but other minerals and metals were not detected by usual methods and they should be studied by other methods like polarized light and transmission electron microscopy. Grobbelaar et al have studied 25 rural bakery women from Transki of South Africa with no history of tuberculosis, and other occupational exposure or mining. Histological picture of these mostly asymptomatic patients were in favor of simple anthracosis (12 patients), anthracosis with macules (6 patients), and mixed dust fibrosis (7 patients). In this study, silica (quartz) was attributed to fibrotic process.[40] The contribution of other particles in anthracosis and potentiating of mixed dust theory also has been revealed by Naccache et al who reported three cases of anthracofibrosis without tuberculosis due to mixed dust. These three patients have had free crystalline silica, mica, kaolin and other silicates on transmission electron microscopy in addition to black pigment of carbon.[41]

Studies on anthracosis particularly in coal mine workers about 100 years ago, when the prevalence of tuberculosis was high in the community, have revealed protective and bordering effect of fibrosis of anthracotic lesions for tuberculosis. Epidemiologic work Wainwright et al.[43] in 19th century in USA and UK found

Table 1. Summary of Iranian publications about anthracosis and tuberculosis.

Author /city	Date of article	Study period	Anthra-cosis No	FOB No	Smoking %	Presence of Occupation Risk %	M/F No	TB No (%)	Comment
Amoli[5] /Tehran	1994	85–90	10	---	Neg	Bakery /100	1/9	2	Smear + Culture -
Amoli [6] /Tehran	1998	86–95	10	891	Neg	Bakery /100	0/10	Neg	----
Towhidi[7]/Mashhad	2002	98–20	29	1118	----	----	9/20	29 (100)	17 granuloma 15 AFB+
Aslani [8]/Tehran	2002	98–01	96	919	----	38.5%	44/52	26 (27)	Tb in Control?
Najafizade[9] /Tehran	2003	4 months	47	290	10.6	30%	24/23	13/(27.7)	73% urban S+ = 11.2%, c+ = 13.5% Path = 5(10.6)
Amoli [10] /Teheran	2004	75–20	819	11664	----	91	430/319	23/(2.8)	S+ = 14 C+ = 9
Mirsadraee[11,12]/Mash-had	2005	2003	----	189	19	91% house 46%Farmer	95/94	52/172(30)	S+ = 22 C+ = 42/163 Path = 18/102
Rezaeitalab[13]/Mashhad	2006	2002–2004	225	1000	----	14%	93/132	(25.3)	Tb = 5.7% in non-anthracotics
Hemmati [14]/Zahedan	2008	2003–2006	34	207	----	33% Bakery F Smoking M	15/19	15/34 (44)	Tb in non-anthracotic = 30/173(17.4)
Najafizadeh[15]/Tehran	2008	7 months 2006	87	87	11.5	6.9% 50% urban 30% fire work	57/30	23/87 (26)	MT EAI = 11 CAS = 7 Haarlem = 4 T clade = 1
Sigari [16]/Tehran , Sanandaj	2009	1982–2006	778	14300	----	98%F housework 41% M farmer 30%M manual work 7.5% miner 86% rural	399/379	----	No work about TB
Amoli [17]/Tehran	2009	1975–2000	102	205	64	50% Housewife 6% farmer 30% Worker Rustic ,F 100%, M 48% Urban M 52%	42/60	27/102(26.4)	19/103 non-anthracot-ics(18.4) had TB
Fekri[18]/Kerman	2010	2003–2007	333	1594	----	----	148/158	(6.9)	2.7% of non-anthracot-ics had TB
Ghaffari[19]/Tabriz	2010	2004–2006	9	9		42 Baker women	0/42		Not mentioned
Ghanei [20]/Tehran	2011	1998–2001	71	919	----	Occ = 42% Bakery = 27% Miner = 11.5%	32/39	41/71 (58)	10.6% of non-anthra-cotics had TB
Pazoki [4]/Tehran	2011	----	150	----	58 smoker	35 bakery	88/62	42/150	TB s/c + = 32 Path = 10 Smear+ =10 BAL + =17 C+ =5 Close contact = 16, 12 with active TB

that prevalence and mortality of tuberculosis in coal miners was lower between all other occupations. For example, TB death rate/100000 people was 540.5 for marble and stone cutters compared to 120.9 for miners and quarrymen. In this study the rate of tuberculosis in miners and quarrymen was less than lawyers and physicians (168.8/100000)but slightly higher than farmers (110/100000).[43] A few years later, Haythorn in his experience on anthracosis proved that, both carbon granule and tubercle bacilli engulfed by alveolar macrophage (endothelial leukocyte), other cells has negligible role in phagocytosis.Moreover the presence of carbon within these cells did not interfere with granuloma reaction to mycobacterium, and pigment bearing cell around the granuloma promote fibrosis and encapsulation of the lesion, and then obliteration of lymphatics by anthracotic process can prevent spread of bacilli and localize the lesion.[44]

It seems that other factors instead of carbon alone are important for predisposition to tuberculosis.Behera et al have found that Indian Women using domestic cooking fuels, have high Odd ratio for getting tuberculosis due to low socioeconomic state, poor ventilation of cooking place(cooking inside v/s open place), however, type of fuels has not significant effect on involving with tuberculosis.[45] In a recent study in China, Hong-Min Fan et al have reported TNF-α- 308 G/A Gene polymorphisms were associated with pulmonary tuberculosis in coal miner pneumoconiosis.[46] According to Chaudhry et al.[36] if increasing mucociliary clearance by smoking can prevent or decrease the process of anthracosis, we assume that tuberculosis by impairment in clearance mechanisms may increase the deposition and remaining of carbon particles in the lung . Overall, it seems that, not carbon alone but presence of other predisposing factors like quartz (silica), living and working

in crowdy , poor ventilated or polluted areas and possibly genetic factors are important for predilection of anthracotic patients to tuberculosis.. Further epidemiologic and pathologic surveys are needed to answer clearly these questions.

Retrospective studies have limitations due to lack of sufficient and effective data for good conclusion . In these 17 articles, only a few have noticed the standards of isolation , culture of mycobacteria and correlation of smear and culture to sure what is(are) reported is (are) Mycobacterium Tuberculosis.[11–13,15,18] There is a lot of reports about the contamination of fiberoptic bronchoscopes and washing machines with mycobacteria including saprophytic ones. Every research which includes bronchoscopy and BAL for Mycobacterium tuberculosis and other Mycobacteria should consider the methodology and standards of isolation, culture of Mycobactria and its pitfalls. In conditions that AFB smear is positive and culture is negative , for example, in Amoli's study,[5] how we can sure that we are confronting with only Mycobacterium tuberculosis. The importance of looking for non-tuberculous mycobacteria have been mentioned previously by Korean team[43] and others.[47–49] Prospective population based and controlled studies with good design are needed to resolve the answers which are not solved by previous studies. In these 17 reports only five studies had control patients.[13,14,17,18,20] and only in last study[4] the history of close contact to tuberculosis as a contributory factor has been clearly notified.[4]

In conclusion, we need more controlled studies to fill the knowledge gap regarding prevalence, trend, complicating factor(s), and associations that we have found up to this time in anthracosis.

Hassan Heidarnazhad MD[1]

[1]Tobacco prevention research center, Masih Daneshvari Hospital, National institute of tuberculosis and lung disease, Darabad, Tehran, Iran. E-mail: heidarnazhad@nritld.ac.ir

References:

1. Global Health Risks: Mortality and Burden of Disease Attributable to Selected Major Risks. WHO report 2009, Geneva: World Health Organization; Available from URL: www.who.int/healthinfo/global_burden_disease/ GlobalHealthRisks_report_Front.pdf.
2. Quantifying Environmental Health Impacts: Global Estimates of Burden of Disease Caused by Environmental Risks .WHO report 2009, Geneva: World Health Organization ; Available from URL: www.who.int/quantifying_ ehimpacts/global/globalair2004/en/index.html.
3. Martin WJ , Glass RI, Balbus JM, Collins FS . A major environmental cause of death .*Science* .2011; **334:** 180 – 181.
4. Pazoki M, Goodarzi HM, Taheri AH, et al. Prevalence of tuberculosis in patients with anthracosis: study on 150 subjects. *Arch Iran Med*. 2012; **15(2):** 128 – 130.
5. Amoli K. Brochopulmonary complications of indoor pollution in iranian rustic population. *Acta Medica Iranica*. 1994; **32:** 35 – 41.
6. Amoli K. Brochopulmonary disease in Iranian housewives chronically exposed to indoor smoke. *Eur Respir J*. 1998; **11:** 659 – 663.
7. Touhidi M, Keshmiri M, Ataran D, Ghiasi Moghadam T, Azarian A. Tuberculus bronchostenosis presenting as anthracofibrosis. *Med J of Mashhad University of Medical Sciences*. 2002; **45:** 73 – 76.
8. Aslani J, Ganei M. khosravi L. Relationship between anthracosis and tuberculosis. *Med J of Tehran University of Medical Sciences*. 2002; **60:** 460 – 464.
9. Najafizadeh K, Zahirifard S, Mohammadi F, Farnia P, Shah-Ghasempour S, Hasanzadeh N, et al. Bronchial anthrcofibrosis or anthracotic bronchitis. *Tanaffos*. 2003; **2:** 7 – 11.
10. Amoli K. Study of 819 cases of anthracotic airway disease. *J Med Council of I R Iran*.2003; **21:** 259 – 265.
11. Mirsadraee M , Saeedi P. Anthracosis of the lung: evaluation of potential causes. *Iran J Med Sci*. 2005; **30:** 190 – 193.
12. Mirsadraee M, Saeedi P. Anthracosis of the lung: evaluation of potential causes. *J Bronchol*. 2005; **12:** 84 – 87.
13. Resaei Talab F, Akbari H. Relationship between anthracosis and pulmonary tuberculosis in patients examined through bronchoscopy. *Birjand University Medical journal*. 2006; **14:** 48 – 55.
14. Hemmati SH, Shahriar M, Molaei NA. What causes anthracofibrosis? Either tuberculosis or smoke. *Pak J Med Sci*. 2008; **24:** 395 – 398.
15. Najafizadeh K, Ghorbani F, Farnia P, Shiehmorteza M, Jamali M. Spoligotyping of mycobacterium tuberculosis in anthracotic bronchitis. *Int J Tuberc Lung Dis*. 2008; **12:** 962 – 966.
16. Sigari N, Mohammadi S. Anthracosis and anthracofibrosis. *Saudi Med J*. 2009; **30:** 1063 – 1066.
17. Amoli K. Anthracotic airway disease: report of 102 cases. *Tannafos*. 2009; **8:** 14 – 22.
18. Fekri MS, Lashkarizadeh MR, Kardoost AH, Shokoohi M. Bronchial anthracosis and pulmonary tuberculosis. *Tannafos*. 2010; **9:** 21 – 25.
19. Gaffari MR, Tagizadieh A. A description of the lung disease found in Irranian women exposed to dung smoke.*J Cardiovas Thorac Res*. 2010; **2:** 29 – 32.
20. Ganei M, Aslani J, Peyman MR, Asl MA, Pirnazar OR. Bronchial anthracosis: A potent clue for diagnosis of pulmonary tuberculosis. *Oman Medical Journal*. 2011; **26:** 19 – 22.
21. Chung MP, Lee KS, Han J, Kim H, Rhee CH, Han YC, et al. Bronchial stenosis due to anthracofibrosis. *Chest*. 1998; **113:** 344 – 350.
22. Torun T, Gungor G, Ozmen I, Maden M, Bolukbasi Y, Tahaoglu K, et al. Bronchial anthracostenosis in patients exposed to biomass smoke. *Turkish Respiratory Journal*. 2007; **8:** 48 – 51.
23. Wynn GJ, Turkington PM, O,Driscoll BR. Anthracofibrosis, bronchial stenosis with overlying anthracotic mucosa: Possibly a new occupational lung disorder: A series of seven cases from one UK hospital. *Chest*. 2008; **134:** 1069 – 1073.
24. Jung SW, Kim YJ, Kim GH, Kim MS, Son HS, Kim JC, et al. Ventilatory dynamics according to bronchial stenosis in bronchial anthracosis. *Tuberc Respir Dis*. 2006; **59:** 368 – 373.
25. Kim SW, Kim IS, Park DH, No TM, Joeng JK, Jung SW, et al. The clinical significance of bronchial anthracofibrosis in the patients with endobronchial tuberculosis. *Tuberc Respir Dis*. 2004; **56:** 495 – 504.
26. Hwang J, Puttagunta L, Green F, Shimanovsky A, Barrie J, Long R, et al. Bronchial anthracofibrosis and tuberculosis in immigrants to Canada from Indian subcontinent. *Int J Tuberc Lung Dis*. 2010; **14:** 231 – 237.
27. Onitilo AA, Engel JM, Nguyen TT, Tanimu SB. Anthracosis and large mediastinal mass in a patient with healed pulmonary tuberculosis. *Clin Med & Res*. 2010; **2:** 99 – 103.
28. Bekci TT, Maden E, Emre L. Bronchial anthracofibrosis case with endobronchial tuberculosis. *Int J Med Sci*. 2011; **8:** 84 – 87.
29. Gupta A, Shah A. Bronchial anthracofibrosis: an emerging pulmonary disease due to biomass fuel exposure. *Int J Tuberc Lung Dis*. 2011; **15:** 602 – 612.
30. Klotz O. Pulmonary anthracosis- A community disease. *Am J Public Health*.1914; 887 – 916.
31. Adamson IYR, Prieditis HL. Response of mouse lung to carbon deposition during injury and repair. *Envirorn Health Perspect*. 1995; **103:** 72 – 76.
32. Baris YI, Hoskins JA, Seyfikli Z, Demir A. Biomass lung: Primitive biomass combustion and lung disease. *Indoor Built Environ*. 2002; **11:** 351 – 358.
33. Moran-Mendosa O, Perez- Padilla JR, Salazar- Flores M, Vazquez-Alfaro F. Wood smoke –associated lung disease: A clinical, functional , radiological and pathological description. *Int J Tuberc Lung Dis*. 2008; **12:** 1092 – 1098.
34. Souza MB, Saldiva PHN, Pope CA, Capellozi VL. Respiratory change due to long-term exposure to urban levels of air pollution. A histopathologic study in humans. *Chest*. 1998; **113:** 1312 – 1318.
35. Bilici A, Erdem T, Boysan SN, Acbay O, Oz B, Besirli K, et al. A case of anthracosis presenting with mediastinal lymph nodes mimicking tuberculous lymphadenitis or malignancy. *Eur J Int Med*. 2003; **14:** 444 – 446.
36. Chaudhry NA, Shah SS, Khan SA, Tayyab M. Carbon deposition in tracheobronchial tree of smokers and non-smokers: An autopsy study. *Biomedica*.1999; **15:** 73 – 76.
37. Ahasan SA, Chowdhury EH, Azam SU, Parvin R, Rahaman AZ, Bhuyan AR, et al. Pulmonary anthracosis in Dhaka Zoo collections-a public health forecasting for city dwellers. *Journal of Threatened Taxa*. 2010; **2:** 1303 – 1308.
38. Beytut E. Anthracosis in the lungs and associated lymph nodes in

sheep and its potential role in the occurrence of pneumonia. *Small Ruminant Research*. 2002; **46:** 15 – 21.
39. Pourshams A, Saadatian-Elahi M, Nouraie M, Malekshah AF, Rakhshani N, Salahi, et al. Golestan cohort study of oesophageal cancer: feasibility and first results. *Br J Cancer*. 2005; **92:** 176 – 181.
40. Grobbelaar JP, Bateman ED. Hut lung: a domestically acquired pneumoconiosis of mixed aetiology in rural women. *Thorax*.1991; **46:** 334 – 340.
41. Naccache JM, Monnet I, Nunes H, Billon-Galland MA, Pairon JC, Guillon F, et al. Anthracofibrosis attributable to mixed mineral dust exposure: report of 3 cases. *Thorax*. 2008; **63:** 655 – 657.
42. Dhar SN, Pathania AGS. Bronchitis due to biomass fuel burning in North India; Gujjar Lung, an extreme effect. *Semin Respir Med*. 1991; **12:** 69 – 73.
43. Wainwright JM. The relation between anthracosis and tuberculosis. *Am J Med Science*. 1905; **130:** 403 – 415.
44. Haythorn SR. Some histological evidence of disease importance of pulmonary anthracosis. *J Med Research*. 1913; **29:** 259 – 278.
45. Behera D, Aggarwal G. Domestic cooking fuel exposure and tuberculosis in Indian women. *Indian J Chest Dis Allied Sci*. 2010; **52:** 139 – 143.
46. 46-Fan HM, Wang Z, Feng FM, Zhang KL, Yuan JX, Sui H, et al. Association of TNF-α-238 G/A and 308 G/A gene polymorphism with pulmonary tuberculosis among patients with coal workers pneumonconiosis. *Biomedical and Environmental Science*. 2010; **23:** 137 – 145.
47. Kim YM, Kim M, Kim SK, Park K, Jin SH, Lee US, et al. Mycobacterial infections in coal workers' pneumoconiosis patients in South Korea. *Scand J Infect Dis*. 2009; **41:** 656 – 662.
48. Ra SW, Lee KH, Jung JY, Kang HS, Park IN, Choi HS, et al. Mycobacterium Kansasii disease presenting as a lung mass and bronchial anthracofibrosis. *Tuberc Respir Dis*. 2006; **60:** 464 – 468.
49. Van Hest R, van der Zanden A, Boeree M, Kremer K, Dessens M, Westenend P, et al. Mycobacterium heckeshornense Infection in an Immunocompetent Patient and Identification by 16S rRNA Sequence Analysis of Culture Material and a Histopathology Tissue Specimen. *J Clin Microbiol*. 2004 ; **42:** 4386 – 4389.

A view of the Rayen Mudbrick Castle in Kerman Province, Iran, Sassanid era. Hazar Mountain is seen in the background (Photo by S. Borzabadi Msc, 2012).