Zoonotic helminthiasis: A challenge unabated

Pulmonary helminthiasis has been reported from antiquity, particularly from areas with significant pastoralistic population. However, the diseases have received scant attention, and research in these areas has largely been limited. Echinococcosis is one such zoonotic infestation caused predominantly by the post-larval metacestode stage of the dog tapeworm, *Echinococcus granulosus*; the life cycle of which requires two mammalian hosts, a definitive host and an intermediate host. Cystic echinococcosis (CE) caused by *E.granulosus* accounts for >95% of the estimated 2–3 million cases worldwide.[1] The disease is endemic in many parts of the world like Mediterranean countries, southern America, Australia, Africa and also in some regions of Asia. The infestation leads to the development of hydatid cysts in various organ systems, the liver and lung being the commonest. Pulmonary CE (PCE) may be an accidental diagnosis in an asymptomatic patient or may present with nonspecific chest symptoms. Incessant bouts of cough with expectoration of salt like tasting fluid may be the presenting symptom of a ruptured cyst.

The current issue of Lung India carries an article where a patient of hydatidosis is shown to present with multiple pulmonary cysts,[2] akin to a bunch of marbles. Radiological features of PCE can be varied and can include single or multiple sharply defined, round-to-oval homogenous opacity of variable size, with several characteristic features on radiology.[3,4] CT features have been well defined and differ in intact and ruptured cysts. While intact cysts present as well-defined cystic masses with variable CT density, ruptured cysts can present various imaging features depending on the extent of the rupture and the remaining contents of the cyst.[5] These include 'signet ring' sign, 'inverse crescent sign', water lily sign, Cumbo sign, empty cyst sign, etc. Calcification and daughter cyst formation is rare in PCE.[5,6] Detached or collapsed endocyst membranes, collapsed daughter cyst membranes and intact daughter cysts are the pathognomic computer tomographic features of ruptured hydatid cysts.[5] Evaluation of extrapulmonary intrathoracic hydatid cysts like chest wall, mediastinal, pericardial, fissural and pleural localization may necessitate MR imaging. While cysts usually have cystic/fluid attenuation, higher attenuation, usually suggestive of solid tissue, has been reported too. Further thick cyst wall has also been reported, mostly in infected cysts.[5] Imaging features are complemented by serological evidence of echinococcosis like immunoelectrophoresis, indirect immunofluorescence, ELISA or hemagglutination.[7] Bronchoscopy may aid in the diagnosis at times.[8] Molecular methods, such as polymerase chain reaction (PCR) and sequencing, can play an important role in the surveillance, diagnosis, epidemiology and management of hydatid disease by ability to detect parasite DNA in various clinical samples, including cyst fluid, blood and faeces.

Surgery has remained the mainstay of management of PCE and has, in recent times, been supplemented by medical therapy and percutaneous aspiration.[9-11]

Variable periods of anti-helminth chemotherapeutic agents are prescribed preoperatively to prevent disease recurrence due to spillage at the time of surgery.[12,13] An 8-week combination of praziquantel and albendazole was most effective in effecting a near-total sterilization of the cysts.[14] Post-operative use of chemotherapeutic agents is used to prevent recurrence.

Echinococcus has a history of taxonomic and nomenclatural confusion, resulting from lack of morphological characters.[15] Increasing use of recent molecular methods has led parasitologists to suggest that *Echinococcus* should be split into 10 species, based on not only molecular but also morphological and ecological criteria.[16] The life cycles and host range of all 10 species are now documented and represent an essential foundation for control efforts.

Molecular epidemiological studies have also been instrumental in identifying and describing sympatric cycles of transmission involving more than one species of *Echinococcus*.[16] Although morphology can be used to differentiate between adult worms of different species, this is not practical given the difficulties in recovering adult specimens and the associated public health risks. However, molecular tools allow species identification from eggs in faeces. For example, a study of wild canids on both sides of the US–Canada found wolves and coyotes infected with *Echinococcus canadensis*.[17] This species comprises two genotypes, G8 and G10, and single and mixed infections are reported.

In areas where multiple species of *Echinococcus* are endemic, it is important to note that clinical infections of humans can be caused by multiple species which is important to inform public health measures. In a recent study in China, 22 isolates of *E. granulosus* and *E. multilocularis* were recovered from one patient and *Echinococcus intermedius* in another.[18] Similar observations have been found in Iran where in a study of 42 patients, 18 were infected with E. granulosus and 24 with the *Echinococcus intermedius* (G6 genotype)[19] In one of the patients in the study, a mixed infection of *E. granulosus* in the liver and right lung, and *E. intermedius* in the left lung, was detected. These studies demonstrate

the value of using molecular diagnostic procedures to ensure appropriate therapeutic interventions.

E. granulosus, throughout most of its geographical range, exists almost invariably under conditions modified by humans and perpetuated by domestic animals with infection of humans principally a result of domestic dogs having access to infected livestock.[20]

Anthropogenic activities allowed spillover from the domestic sheep/dog cycle, to a cycle involving native wildlife, macropod marsupials and dingoes in Australia, that not only impacts on public health and livestock industries but also on wildlife health.[21,22] This artificial, man-made cycle also represents a barrier to the control or eradication of *E. granulosus* in sheep on the Australian mainland due to infected dingoes contaminating sheep pasture[21] Interestingly, no evidence existed that *E. granulosus* was present before the arrival of European settlers, with whom *E. granulosus* was introduced in sheep.[22] Similarly, *Echinococcus canadensis* (formerly termed as genotypes G8 and G10) is also maintained in a wild animal cycle involving wolves and large cervids (moose and caribou), originally referred to as the 'northern form' of E. granulosus, and principally occurring in Alaska, Canada, parts of Scandinavia, Eastern Europe and the USA.[23,24] Domestic or free-roaming dogs have long been recognized as important 'bridging hosts' between the wild, sylvatic cycle of E. canadensis and people.[25] In northern Canada, interaction between the wildlife cycle and indigenous communities occurs due to subsistence hunting within indigenous communities where domestic dogs have access to offal and carcasses leading to infection with E. canadensis.[17,26]

Echinococcus felidis is confined to Africa and may represent an indigenous form perpetuated in wildlife cycles involving the lion as the principal definitive host and the warthog as an intermediate host. With the domestication of livestock in Africa, *E. granulosus* has become widespread in domestic livestock with *E. intermedius* also occurring in camels. Over 18 species of wild herbivores from different parts of Africa have been found to be infected with hydatid cysts.[27] These include the most common prey species of the lion. The existence of an independent cycle in wild mammals in Africa has been proposed,[28] and there is a speculation that *E. felidis* is responsible for infections in domestic livestock and other wildlife intermediate hosts of *Echinococcus* in Africa.

Helminth zoonoses remain a global problem to public health and the economy of many countries. PCR-based techniques and sequencing have resolved many taxonomic issues and are now essential to understanding the epidemiology of helminth zoonotic infections and the ecology of the causative agents. Thus, today, we have the molecular tools to characterize helminth zoonotic infection, and we better understand the epidemiology, ecology and transmission patterns of these parasites. While theoretically we are in the best position possible to control helminth zoonoses, practically, this is not the case. Vaccines have been introduced but their full impact on controlling helminth zoonoses is unclear at present as they would still have to be administered at a significant expense in LMIC regions where their administration would be most effective. Anthropogenic factors like culture, poverty, poor hygiene, dietary factors, climate factors and urbanization have all been linked to the perpetuation of zoonotic helminthiasis. In terms of zoonotic helminths, effective control must take a one-health approach. This would need to come from governments and aid agencies, given the likely lack of commercial interest for private organizations. We need to act now, with well utilized, successful approaches that require education and government support. Otherwise, the situation will only worsen due to anthropogenic factors that are perpetually exacerbating the problem of helminth zoonoses.

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