QUESTION BANK (VETERINARY PHARMACOLOGY & TOXICOLOGY)

PAPER No. 25

(METALLOIDS)

I. NAME THE FOLLOWING:

1.An antiparasitic agent causing fluoride toxicity.--(Sodium fluoride)

2.Charecterestic lameness in fluorine toxicity .—(Moving lameness)

3.Few Fluorine alleviators-- (Aluminium salts, calcium carbonate, deflurinated phosphate).

4.Fluorine containing ores—(Fluorspar, cryolite, phosphate rock, apatite.)

5.One element causing lameness in animals in chronic cases.--(Fluorine)

6.Organic form of fluorine which is highly toxic.-(Sodium fluoro acetate (rodenticide), fluoroethyl ester of fluoroacetic acid --(poisonous gas)

7.Organic form of fluorine which is highly toxic.-(Sodium fluoro acetate (rodenticide), fluoroethyl ester of fluoroacetic acid--(poisonous gas)

8.Other name for chronic fluorine toxicity.--(Fluorosis)

9.Poisoning caused due to ammoniated molasses.-- (Bovine bonkers syndrome)

10.The brown black dis-colouration of teeth in fluorine toxicity-.-(Mottling of teeth)

11.The most susceptible species to sodium chloride poisoning.—(poultry)

12.The most susceptible mammal to sodium chloride poisoning.—(pigs)

13.Two poison that selectively deposited in specific tissue.-(fluorine, lead in bones)

14.Two poisons that hinter oxygen transport to tissues.-(Nitrate, Chlorate)

15.Two inorganic form of fluoride._(sodium fluoride ,ammonium fluoride, sodium hexafluorosilicate)

II.STATE TRUE OR FALSE:

1.Acute fluorine poisoning is otherwise known as fluorosis.—(F)

2.Ammonia formation is speeded up by alkaline pH.-(T)

3.Aluminium salts are fluorine alleviators.—(T)

4.Amyl nitrate inhalation can be used to convert haemoglobin to methaemoglobin.-(T)

5.Amyl nitrate can not be used instead of nitrate for the conversion of haemoglobin to met haemoglobin.-- (F)

6.Bone will act as a sink for fluorine.—(T)

7.Burning of nitrogen containing plastics release cyanides.-- (T)

8.Burning of soft coal may produce fluorine contamination.-(T)

9.Cattle will not develop tolerance to urea toxicity.-(F)

10.Cattle with blind staggers exhibits a tendency to wander in circles.-(T)

11.Dietary calcium and iron retard absorption of fluorine from gastro intestinal tract,-(T)

12.Ensiling nitrate rich fodder results in loss of nitrate content.—(T)

13.Nitrite ion is more reactive than nitrate ion.—(T)

14.Excess methylene blue will convert haemoglobin to methaemoglobin .—(T)

15.Foetal tissue can accumulate more ammonia than Mother-(T)

16.Fluorosis can occur in mild form in cattle if diet contain 100 ppm fluoride as rock phosphate for 3-5days , with in one month symptoms will develop.-(T)

17.Fluorine toxicity in young ones causes malformation of enamel and dentin.-(T)

18.Fluorine can act as an anticoagulant.-(T)

19.Fluorine is mainly stored in blood-(F)

20.Fluorine inhibits the production of phosphopyruvate.-(T)

21.Fluorine toxicity causes the formation of 'chalky white bones'-(T)

22.Fluorine is mainly stored in blood.-(F)

23.Fluorine has a strong affinity to calcium , ferrous and aluminium ions._(T)

24.Fluorine alleviators like aluminium salt, calcium carbonate can be given orally.-(T)

25.For estimation of nitrate rumen content is well recommended.(T)

26.For methaemoglobin estimation blood sample must be collected immediately or with in one hour of death.-(T)

27.High sulphur is associated with Crohn's disease.—(T)

28.Ice cold water (0-4 Degree centigrade) up to 40 liters can be given intra-ruminally in urea toxicity.-(T)

29.If the level of fluorine in the diet is above 2-4 ppm it may be toxic.-(T)

30.In phosphorus poisoning gastric lavage with any oil is advisable.-(F)

31.In sheeps feeding of 10-15 gm. Urea at a time may produce toxicity.—(T)

32.In cattle 40 ppm of fluorine in diet can cause chronic toxicity in 3-6 years time.-(T)

33.In cattle urea tolerance will not disappear on withdrawal .-(F)

34.In cattle and sheep urea should not exceed 0.3 to 0.5 gm./kg body weight.—(T)

35.In phosphorus poisoning gastric levage with 0.1 to 0.2 % potassium permanganate is advisable-(T)

36.In wet hay nitrate will be converted to nitrite on exposure to air.-(T)

37.Kerato conjunctivitis is produced in chicken by exposing to ammonia.-(T)

38.Keratoconjunctivitis is produced in chicken by exposing to urea.—(F)

39.Nitrites in the feed is more toxic than nitrates.-(T)

40.Nitrogen fertilizers can enhances the cyanide content of cyanogenic plant.-(T)

41.Red phosphorus is non toxic.-(T)

42.Small amounts of fluorine (1-3) ppm in the drinking water increases the calcification of the teeth.-(T)

43.Safe level of fluorine as rock phosphate in cattle ration is 60—100 ppm.-(T)

44.Silo fillers disease is caused by Sulphur dioxide.-(F)

45.Sodium and potassium oxalate are soluble in water.—(T)

46.Sodium bicarbonate is not toxic in dogs but toxic to birds.-(T)

47.Sodium bicarbonate is toxic in poultry.-(T)

48.Sodium chloride poisoning is otherwise known as water deprivation syndrome.—(T)

49.Sodium fluoride is used as antiparasitic and anticoagulant.—(T)

50.Sodium fluoro acetate blocks the citric acid cycle and produce toxicity.-(T)

51.Sodium fluoro acetate is converted to fluoro citrate and produce toxicity.-(T)

52.Sodium fluoroacetate as such is not toxic.-(T)

53.Sodium fluoride is used as antiparasitic and anticoagulant.—(T)

54.Sodium methylthiouracil reduce BMR.-(T)

55.Sodium potassium and ammonium nitrate and nitrite are important in considering nitrate poisoning.-(T)

56.Soya meal contain a lot of urease enzyme .-(T)

57.Specimen for nitrate estimation must be frozen and packed in plastic container.-(T)

58.Sulphur is an essential element. –(T)

59.Sulphur is an antagonist of copper.—(T)

60.The acute lethal dose of sodium chloride in swine is 2.2 g/kg.-(T)

61.The maximum tolerable level of fluoride for cattle is 40—50 ppm.—(T)

62.The order of susceptibility to nitrate is pigs- cattle—sheep—horse.-(T)

63.Urea mixed with soya meal is advisable in cattle.-(F)

64.Vitamin E deficiency increases selenium toxicity.—(T)

65. Urea tolerant animals can handle as much as 4 times the usual urea level in the feed.-(T)

66.White / yellow phosphorus is highly toxic than other natural forms.-(T)

67.Yellow phosphorus is highly toxic.-(T)

68.Yellow phosphorus on exposure emit white fumes.-(T)

69.Rock phosphate contain high amount of fluorine.-(T)

70.With the development of urea tolerance animals can handle as much as 4 times the usual urea level in the feed.-(T)

III.FILLUP THE BLANKS WITH MOST APPROPRIATE WORDS:

1.Acid burned surfaces must be flooded with water except for burns due to ……..acid.-(Sulphuric)

2.Aluminium manufacturing unit causes mainly .....contamination.-(Fluorine)

3.Among farm animals ……. ……. and……are more susceptible to nitrite poisoning.-(Pigs, Ruminants and Fowls)

4.As a treatment in urea poisoning up to ……. liters of vinegar can be given in cattle.-(8)

5.As an immediate step in the treatment of urea poisoning …….. can be given orally to lower the rumen pH. –(Vinegar)

6.Chronic poisoning with mineral ore 'Apatite' is known as .....—(Fluorosis)

7.Dietary ……and……..ion retard absorption of fluorine ions from G.I. tract-(Calcium and Iron)

8.Dragging of hind feet while walking or knuckling of the fetlock is a characteristic signs of ……poisoning in cattle.(sodium chloride)

9.Even though red phosphorus in nontoxic commercial preparations containing red phosphorus is toxic because it contain traces of …….phosphorus.-(Yellow)

10.Even though chances of acute fluorine poisoning is rare there is a possibility in ……species because of the use of sodium fluoride as anthelmintic in this species.-(Swine)

11.Feed grade phosphate should not contain more than …….part fluorine to hundred part phosphorus.-(one)

12.Fluorine is gradually excreted via ……..-(kidney / urine)

13.Fluorine above ……..ppm in feed on dry matter basis causes toxicosis.-(100 ppm)

14.Fluorine level in urine above ……..ppm is indicative of fluorine toxicosis.—(15 ppm)

15.Fluorine alleviators will combine with fluorine in …….of animals.-(Gastro intestinal tract)

16.Fluorine inhibits the glycolytic utilization of glucose by ……..and that is why fluoride is added as an anticoagulant to blood for ……. estimation.-(RBC, glucose)

17.For estimation of nitrate in blood ……..is preferred as a clinical sample .-(serum)

18.For diagnosis of urea toxicity the post mortem sample for lab. analysis must be preserved in…….condition. (frozen)

19.Hydrolysis of urea to ammonia in the feed is speeded up by …… enzyme present in plants consumed by the animals.-(urease)

20.If ……% of haemoglobin is converted to methaemoglobin animal shows clinical signs of anoxia.-(20-40%)

21.If ……..% of haemoglobin is converted to methaemoglobin animal dies.-(above 60%)

22.In chicken ammonia from excreta causes damage to the eyes, the condition is called as ……-- (Kerato conjunctivitis)

23.In fluorosis the bone ash level of fluorine will be above ……..ppm.-(5000)

24.In …….toxicity tarry bloody exudates from natural orifice may leads to confusion with anthrax.—(chlorate)

25.In nitrate poisoning treatment will be successful if ……..% of the haemoglobin is available for oxygen transport.—(30%)

26.In phosphorus poisoning exhaled air has …… odour and ……..in dark.-(garlic, glow)

27.In …….toxicity tarry bloody exudates from natural orifice may leads to confusion with anthrax.—(chlorate)

28.In pigs ……..toxicity is known as water deprivation syndrome –(Sodium chloride)

29.Leuco……is the actual agent which convert methaemoglobin to haemoglobin.-(methylene blue)

30.Low land abortion is due to ……poisoning.-(nitrate)

31.Lime water can be used in ……ingestion and lime juice can be used in ……ingestion.—(acid, alkali)

32.Maximum tolerable level of fluoride in cattle is …….ppm. –(40-50)

33.Marked ……pulse is a characteristic symptom in urea poisoning in cattle.-(Jugular)

34.Methylene blue is administered intravenously at a concentration of ……..-(1%)

35.Mottling of teeth is seen in .....poisoning.—(Fluorine)

36.Nitrite is …….times more toxic than nitrate .-(10)

37.Nitrate will convert …….to ……..which is unable to transport oxygen.-(haemoglobin to methaemoglobin)

38.Nitrate salts are converted to nitrite in the ……tract.-(Gastro intestinal)

39.Oat hay contain as high as …….% nitrate.-(3—7%)

40.Oily medium ……the gastric absorption of phosphorus.-(increase)

41.On analysis ……ppm or more of ammonia in the rumen liquor is indicative of urea toxicity in cattle.-(800)

42.On analysis ……ppm or more of ammonia in the blood is indicative of urea toxicity.-(20)

43.Palpation of long bones is painful in …….toxicity.-(fluorosis/ fluorine)

44.Phossy jaw is a characteristic symptom in .....poisoning.-(Phosphorus)

45.Phosphorus exist in three forms ……., ……and …….-(White(yellow) , Red and Black)

46.Phosphorus trioxide is having ……..odor.-(Garlic)

47.Smoke bombs cause …….poisoning.-(Phosphorus)

48.Sodium bicarbonate is highly toxic in .....—(Poultry)

49.The lethal dose of nitrate in cattle is ……mg/kg (150 mg)

50.The safe limit of nitrate in feed and plants is……..-(0.5%)

51.The safe limit of urea in the ration of sheep is ……gm/ Kg feed.-(0.3 to 0.5 gm/kg)

52.The safe limit of fluorine level in mineral mixture with salt is……..%.—(0.05%)

53.The recommended urea level in cattle feed is below ……..%. of concentrate or ……..% of total feeds.-(3% , 1%)

54.The safe limit of fluorine level in drinking water is……ppm.-(0.7 to 1 ppm)

55.The usual level of urea permitted in the cattle feed is 1% of total feed or ……..% of the concentrate.—(3)

56.Tolerance to fluorine can be increased by balanced intake of…… (calcium, phosphorus, vitamin-D)

57.Volcanic eruption can cause…….fluorine toxicity in animals.(acute)

58.Volcanic ashes may contain as high as ……..ppm of fluorine –(2000)

59.White phosphorus on exposure to air release……which is luminous in dark.-(white fumes)

60.Yellow discolouration of mucous membrane is seen in .....poisoning—(Phosphorus)

61……is most susceptible species for sodium chloride poisoning .-(poultry)

62……..will act as a natural sink for fluorine.-(Bone)

63……..phosphorus is used only for research purpose.-(black)

64……lameness is characteristic in fluorosis.-(intermittent/ moving)

65……..is a characteristic sign in birds exposed to ammonia --(keratoconjunctivitis)

IV. MATCH THE FOLLOWING:

Match each one in A to matching ones In B and C:

A	B	C
1.Rock phosphate	Bright red blood---9	Fluorine alleviator---3
2. Sodium fluoride	Water deprivation—12	Chalky white bone---1
3. Aluminium salt	Rat poison---6	Low land abortion—8

4. Yellow phosphorus	Silofillers disease---11	Cyanosis---8
5. Red phosphorus	Dark red blood---10	Sodium thiosulphate---9
6. Zinc phosphide	Fluorine---1	Sugar beat tops—8
7. Adamsite	highly toxic phosphorus—4	Poultry—12
8. Nitrate poisoning	Bound to fluorine----3	Garlic odour---4
9. Cyanide	Oat hay poisoning---8	Antiparasitic---2
10. Hydrogen sulphide	Anticoagulant---2	Match sticks---5
11.Nitrogen dioxide	Non toxic phosphorus---5	Phosphine---6
12. Sodium chloride	Phossy jaw—4	Mottling of teeth---1

V. CHOOSE THE CORRECT ANSWERS FROM THE GIVEN ONES:

1.Acute fluoride toxicity is generally seen in a) Cattle b) Dog c) Pigs d) Poultry.-(C)

2.Acute fluorine toxicity is seen in a) Swine b) Cattle c) Sheep d) all the above.-(D)

3.All the following are characteristic symptoms of fluorine toxicity except. a) Mottled teeth b) exostosis at bone extrimities c) moving lameness d) chalky white bone e) Phossy jaw.-(E)

4.Black tarry blood that fails to clot is a characteristic finding in the following poisoning. A) fluorine b) Phosphorus c) Nitrate d) Hydrogen sulphide.—(B)

5.Dark chocolate coloured blood is indicative of toxicity with the following agents. a) Nitrites b) Nitrates c) Chlorates d) all the above.—(E)

6.Dragging of hind feet of cattle while walking is a characteristic symptom in toxicity due to a) Fluorine b) Sodium chloride c) Nitrite d) Ammonia .-(B)

7.Death due to sodium chloride poisoning is mainly due to a) Liver damage b) Kidney damage c) disturbance in water and electrolyte balanced) brain damage .-(C)

8.Following oares are fluorine containing ore. a) fluorspar b) Cryolite c) Apatite d) all the above.(D)

9.For superficial burns with the following agents the area must be flooded with water except a) Sulphuric acid b) Hot water c) Hydrochloric acid d) Nitric acid.-(A)

10.For burns with one of the following acid application of alcoholic pads followed by oil pad is recommended. a) Sulphuric b) Carbolic c) Nitric d) Acetic.-(B)

11.Fluorine has a high affinity to a) Ferrous b) Aluminium c) Calcium d) all the above.-(D)

12.Fluoride salt is added as anticoagulant to blood for estimation of glucose. A) It is better than sodium EDTA b) prevent glycolytic utilization of glucose by RBC c) highly soluble in blood d) nontoxic.-(B)

13.Fluorine toxicity is indicated if the urine level of fl. is above a) 25 ppm b) 75 ppm c) 2ppm d) none of the above.-(D)

14.Following plants are very rich in nitrate content a) immature oats b) Corn grass c) Sorghum d) all the above.-(D)

15.Following compounds are fluorine alleviators. a) Aluminium salt b) Calcium carbonate c) deflurinated phosphate d) all the above.-(D)

16. Industries causing fluorine contamination are a) aluminium manufacturing by electrolyte process. b) brick and tile factory using fluorine contaminated clay. c) enamel factory d) manufacturing of acid phosphate from rock phosphate. e) all the above.-(E)

17.In alkali poisoning all the following therapeutic measures are advisable except one .a) Acetic acid 5% b) Citric acid c) Lime juice d) Limewater.-(A)

18.In acid poisoning all the following therapeutic measures are advisable except one .a) Sodium bicarbonate b) Calcium carbonate c) Lime juice d) Limewater.-(C)

19.In nitrate poisoning treatment will be successful if the following percent of haemoglobin is still available for oxygen transport.--a) 50% or above b)70 % c) 30% d) at least 10%.—(C)

20.Low land abortion is due to a) fluorine poisoning b) phosphorus poisoning c) Nitrate poisoning d) all the above.-(C)

21. 'Mad dog running' is a symptom of a) sodium fluoroacetate b) zinc phosphide c) ANTU d) warfarin -(B)

22.Moving lameness is characteristic in a) rickets b) fluoride toxicity c) lead toxicity d) phosphorus toxicity.-(B)

23.Mottling of teeth is seen in . a)Lead poisoning b) nitrite poisoning c) fluorine poisoning d) all the above.-(C)

24.Natural sink for fluorine is a) Blood b)muscle c) bone d) liver.-(C)

25.One of the following is not a fluorine containing ore. a) fluorspar b) cryolite c) apatite d) sodium fluoroacetate.-(D)

26.One of the following is highly toxic form of phosphorus. A) Red phosphorus b) White phosphorus c) Black phosphorus d) Phosphorus trioxide.-(B)

27.One of the following statement is correct.- a) red phosphorus is nontoxic and is prepared by heating white phosphorus b)Red phosphorus is non toxic and is prepared by mixing black and yellow phosphorus and heating. c) Red phosphorus is moderately toxic and is prepared by heating yellow phosphorus. d) Red phosphorus is highly toxic prepared as such in lab.-(A)

28.One of the following mineral in the diet retard fluorine absorption a) Calcium b) sodium c) Potassium d) Magnesium.-(A)

29.Oat hay poisoning is a) nitrate poisoning b) Fluorine poisoning c) Phosphorus poisoning d) none of the above,-(A)

30.One of the following species is highly susceptible to urea toxicity. A) Ruminants b) Dogs c) Pigs d) Birds .-(A)

31.Sodium bicarbonate is highly toxic in a) poultry b) pigs c) cattle d) sheep.-(A)

32.Safe limit of fluorine in drinking water is a) 1-5 ppm b) Above 10ppm c) Below 5 ppm d) 0.7-1ppm –(D)

33.The chocolate colourd blood due to methaemoglobin formation is seen in nitrite poisoning b) Cadmium poisoning c) Arsenic toxicity.—(A)

34.The colour of the venous blood in nitrite poisoning will be a) dark chocolate b) cherry red c) bright red d) brown.-(A)

35.The colour of the venous blood in hydrogen sulphide poisoning will be a) dark chocolate b) dark red c) bright red d) light red.-(B)

36.The decreasing order of susceptibility among following species to fluorine toxicity is a)Cattle , Sheep , Horse , Pigs, Poultry. b)Cattle, pig, sheep, horse, poultry. c) Horse, pig, poultry, cattle, sheep. d) Poultry, horse, pig, sheep, cattle.---(A)

37.The main clinical symptoms in chronic phosphorus poisoning are a) Tooth ache b) offensive discharge from socket c) Sequestrum formation d) all the above.-(D)

38.The most susceptible species to sodium chloride poisoning is a) Cattle b)Horse c)Dogs d)Poultry.-(D)

39.The post mortem specimen for laboratory examination in nitrate poisoning must be preserved in a) saturated saline b) alcohol c) formalin d) frozen state.-(D)

40.The safe fluoride level in mineral mixture with salt is a) 0.1 % b) 0.05% c) 0.7 to 1ppm d) none of the above.-(B)

41.Water deprivation syndrome is poisoning due to a)Sodium chloride b)sun burn c)high temperature d)nitrites.-(A)

42.Yellow phosphorus on exposure to air a) emit white fumes b) emit fumes with garlic odor c) luminous in dark d) all the above.-(D)

43.Alkali disease is toxicity due to a) copper b) mercury c) selenium d) molybdenum --(C)

VI.ANSWER THE FOLLOWING:

1.How can we do the differential diagnosis of urea poisoning with arsenic, OP and OC ? In urea poisoning –colic without diarrhea, violent struggling, incordination, twitching and tonic convulsions. Arsenic and other heavy metal –colic with diarrhea-without nervous or motor signs. In organochlorine poisoning high rise of body temperature- intermittent convulsion . in organophosphorus poisoning –similar to urea poisoning but alleviate with atropine.

2.How vinegar will help in the treatment of urea poisoning : If acetic acid 5% (vinegar) is administered in urea poisoning .It combines with ammonia to form ammonium acetate, which is utilized immediately by the microbs and thus ammonia is removed . Acetic acid treatment can be repeated.

3.How nitrate and nitrite act as toxic agent? Nitrite interact with haemoglobin to form methaemoglobin by oxidation of ferrous haemoglobin to ferric haemoglobin which can not carry oxygen. Body can tolerate up to 20-40 % methaemoglobin, if it exceeds 60 % animals will die. Nitrate ion directly relaxes the smooth muscles (vascular) result in hypotension and decrease cardiac output –tissue oxygen starvation. Primary nitrate poisoning is similar to common salt poisoning.

4.In chlorate poisoning methylene blue will act as an antidote in small amount and in excess amount it will worsen the condition .Why? Methylene blue is converted to leuco methylene blue in the body with

the healp of an enzyme NADPH2 system. Leuco methylene blue so produced will act as the reducing agent to act on methemoglobin. This leucomethylene blue is converted back to methylene blue with the help of this enzyme and this process happen as long as free enzyme is available. When too much methylene blue is administered enzyme will not be sufficient to convert to leucomethylene blue. This excess methylene blue will act like chlorate- convert haemoglobin to methemoglobin and worsen the condition

5.In ammonia toxicity some times dam may die and foetus may escape. Why? foetus can store large amount of ammonia compare to dam and urea can not crosses the BBB of foetus.

6.Monogastric animals are less susceptible to ammonia toxicity from urea why? Because to release ammonia from urea either urease enzyme or alkaline pH of stomach is essential . In mono gastric animal both these conditions are absent so no ammonia release and subsequent toxicity.

7.Symptoms of chronic phosphorus poisoning: In chronic phosphorus poisoning the main clinical symptoms is 'Phossy jaw' . First tooth ache leads to extraction and socket fails to heal-offensive discharge-bone is exposed and show necrosis with sequestrum formation-finally death.

8.Urea itself will stimulate the production of ammonia How? Urea liberate sufficient ammonium ions to make the rumen content more alkaline. Alkaline pH will stimulate the release of ammonia from urea

9. what is the treatment for burns due to phenol? Wash the area with alcohol or apply alcohol pads 3-4 times to extract phenol in to alcohol, then apply some oily dressing.

10.What are the toxic action of fluorine ? lack of definition at epiphysis, suppress the enzymes formation of bone phosphates , impaire calcium metabolism, impaire glycolytic utilization of glucose by RBC, affect anaerobic glycolysis, act as anticoagulant, disrupts ionic balance .

11.What are the mechanisms of urea toxicity? It suppress citric acid cycle, it stimulate anaerobic glycolysis, increase blood glucose, increase blood lactate.

12.Why blood serum is not preferred over plasma in lab. test for nitrate analysis ? Some amount of nitrate will bound to plasma protein which will be removed along with the blood clot ,hence serum will give only false value of blood.

13.Why urea is not mixed with soya meal ? soya meal contain a lot of urease enzyme which will act on urea to release ammonia and produce toxicity.

VII.WRITE SHORT NOTES ON:

1.Bovine Bonkers syndrome: feeding of ammoniated molasses,(urea molasses) wheat straw and hay are fed to cattle .Interactions of reducing sugar with ammonia results in a bye-product called '4-methyl imidazole' which is the toxic agent to cattle-symptoms stampeding, ear twitching, trembling, champing, convulsion- totally called as bonkers syndrome.

2.Diagnosis and treatment of fluorine toxicity. By symptoms, determination of fl. in the bones. Bone ash may contain above 5000 ppm ,urine level above 15ppm, on post mortem examination ivory colour of bone changes to chalky white, exostosis of long bones and mandibles, mottled teeth. Treatment includes – remove the animal from the source or source from the animals-symptomatic, fluorine alleviators- calcium intravenously- increase tolerance to fluorine by balanced intake of calcium phosphorus, vitamin D.

3.Fluorine alleviators: are agents which reduce the fluorine load in the G. I tract by binding with it, complex become insoluble and can not be absorbed. As a result less fluorine is available for absorption so that toxicity can be reduced. Eg. aluminium salts, calcium carbonate, deflurinated phosphates.

4.Mechanism of action of fluoride toxicity (acute) : After ingestion large amount of soluble inorganic fluoride form hydrofluoric acid in stomach leads to gastro intestinal irritation or corrosive effect . Fluorine binds to several cations such as calcium, manganese and magnesium in the body and disrupts ionic balance . it interfere with calcium metabolism , form insoluble complex of calcium cause hypocalcemia affecting several physiological functions in the body. Fluoride act as anticoagulant because of its calcium precipitation

5.Phossy jaw: it is a condition produced due to chronic phosphorus poisoning- main symptoms are necrosis of jaw bones-tooth ache leads to its extraction. The socket fail to heal and there will be offensive discharge from it – bone is exposed and show necrosis with formation of sequestrum. The condition may progress and animal may die of debility.

6.Pathogenesis of urea/ammonia toxicity: Normally some ammonia is liberated in the rumen which is in the ionic form(NH_4) and this is soluble but its charge prevent its absorption across rumen wall. If rumen pH is increased to 11 or more(when too much urea is ingested or too much ammonia is liberated) ammonia exist in ammonia form –soluble –lack the ionic charge –readily absorbed to blood. Normally ammonia is removed from blood by liver converted to urea or incorporated in glutamic acid for glutamine synthesis which is depended on substrate provided by citric acid cycle. When absorption rate is more than these conversion rate ammonia accumulate. Primarily it inhibit the citric acid cycle ,increase anaerobic glycolysis, increase blood glucose and lactate level, reduce energy production and cellular respiration- nervous signs--leads to convulsion.

7.Sources of fluorine in animals: Ores containing fluoride eg, Cryolite, apatite- Feed supplement /mineral mixture high in rock phosphate/ super phosphate. Contamination of drinking water /forage. fl.rich soil, deep well water. Consumption of forage rich in fluorine .Environmental contamination from factories-aluminium manufacturing units by electrolytic proces, fertilizer plant, units manufacturing of acid phosphate from rock phosphate, mineral supplement plant, enamel factories ,glass etching units and clay factories. Accidental ingestion of sodium fluoride as acaricides. Dust and gas from volcano eruption.

8.Source of nitrate poisoning: Nitrogen fertilizer increase the nitrate level in plants , hormone type weed killer 2-4 D also increases the nitrate content of plants. Well water high in nitrate. Plants growing in nitrate rich soil. Whey containing nitrate. Use of well water containing high amount of nitrate. Potassium nitrate (used for the manufacture of gun powder) and ammonium nitrite (used for dynamite

production) will act as a source when they contaminte. Some plants like immature oats, corn(maize), sorghum, sugar beat top, rye, sorghum contain high level of nitrate. Organic nitrite (nitroglycerine , polynitrate, vasodilators antihypertensive agents.

9.Sources of phosphorus poisoning : accidental ingestion of phosphate fertilizer, factories (fire works, gun powder, smoke bombs, match sticks, military ammunition factories.) contaminating the environment. Finely divided phosphorus is mixed with fats and oil to promote their absorption , over consumption of these causes toxicity, use of phosphorus as rodenticides accidental consumption of this can cause poisoning- feeding of excess wheat bran rich in phosphorus and low in calcium may cause toxicity.

10.Symptoms of acute phosphorus toxicity: It is a protoplasmic poison. Corrosive and necrotizing effect on stomach, main symptoms are vomition, diarrhea, colic, acute hepatic failure, jaundice, hypotension, shock. Reduce prothrombin level- breath will glow in dark – have garlic odour. Following the initial symptoms recovery lasting for 2-4 days- abdominal pain, vomition etc. recur with jaundice. Nervous signs continue for several days and finally death . Contact with skin causes burns which heals very slowly.

11.Symptoms of fluorine toxicity: In young ones malformation of enamel and dentin, in adult excessive wear of molars, incisors, mottling of teeth. On skeleton- disrupted osteogenesis, defective mineralisation, exostosis on extremities, osteoporosis, chalky white bones. Inhibits enzyme required for bone, teeth formation. Intermittent lameness (periodic and moving lameness) enlargement of sternum and jaw bones, exostosis of long bones palpation of affected bones cause severe pain, painful stiff gait and posture, spontaneous fracture, mottled teeth, chalky white enamel . Decrease milk yield and feed intake- anorexia and emaciation. Symptoms of acute toxicity. acute toxicity is rare –ingestion of high amount of sodium fluoride as anthelmintic in pigs or volcanic eruption can cause acute toxicity –inhaled as gas- pulmonary oedema. Orally symptoms are gastro enteritis, vomiting, urination, defecation, muscular weakness, delayed clotting, clonic excitability, muscle tremors ,convulsion, coma and death.

12.Symptoms of urea toxicity: Symptoms of urea toxicity may appear in 10min. to 4 hrs. Initially salivation, rumen atony, bloat grinding of teeth, severe colic, kicking at abdomen ,groaming (deep sound due to pain) forced rapid breathing, marked jugular pulse, death after violent struggling and bellowing may occur in few hours to 3-4 day. Some animals die of cardiac arrest(increase potassium level) ventricular fibrillation/respiratory paralysis.

13.Treatment of nitrate poisoning: methylene blue is one of the drug of choice- It is a thiazine dye and is an oxidizing agent after ingestion converted to a reducing agent leucomethylene blue by NADPH2 depended system- the leucomethylene blue then reduces methhaemoglobin back to haemoglobin. In this the leucomethylene blue is oxidized back to methylene blue which in turn reconverted to leucomerthylene blue as long as there is sufficient NADPH2 in the system. Too much methylene blue which can not be handled by NADPH2 (NADPH2 is limited in the system) will act as an oxidizing agent convert haemoglobin to met. haemoglobin) so use with in the dose limit. (Cattle and sheep 4-8 mg/kg. slow i/v as 1% sol) maximum up to22 mg/kg .Lower dose may be repeated in 20-30min in severe cases.

Ascorbic acid is a reducing agent can be used. Gastric lavage, blood transfusuion, oxygen therapy , saline purg, plenty of water. Treatment will be successful if 30% of haemoglobin is still available for oxygen transport.

14.Water deprivation syndrome: Sodium chloride poisoning is otherwise called as water deprivation syndrome. The most susceptible mammal is pigs. The toxic dose vary between species. higher salt will upset the tissue water balance, unable to remove excess water from blood by kidney and intestine. Hyper tonicity of blood-shrink the capillary vascular endothelium in brain meninges and other organs-increase capillary permeability- oedema predominate in brain-nervous signs results. Symptoms- thirst, pruritus, salivation, nasal discharge, polyurea followed by anurea and diarrhea. hyperesthesia, blindness deafness moving in circles, shivering and twitching. In cattle dragging of hind feet while walking-knuckling of the fetlock. Treatment -- give fresh water little by little.

IX. WRITE ESSAYS ON:

1.Enumerate the chances of Ammonia poisoning in cattle, Explain the pathogenesis, symptoms and treatment of urea poisoning.
2.Write in detail ethiology, mechanism of toxicity, symptoms and treatment of fluorosis in cattle.
3.Fluorosis in cattle.
4.Water deprivation syndrome.
5.Etiology,pathogenesis, symptoms and treatment of urea toxicity in ruminants.
6.Nitrate poisoning in cattle.

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