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Lassa, Marburg and Ebola: newly described African fevers

As a result of rapid travel by jet, people can arrive in North America from any part of the world within the incubation period of any infectious disease. While malaria is the most common cause of fever in Africa, there are other newly described fevers that are more contagious and more lethal. Lassa fever is one of them.

Most Canadians had never heard of Lassa fever until August 1976, when the news media described in detail a presumed case in a woman hospitalized in metropolitan Toronto.¹ The woman had never visited Africa; she was returning to Canada after a visit to Europe when she became ill. About the same time a case of Lassa fever was diagnosed in London. The woman from Toronto had had no contact with this individual or any other sick person. However, because her fever and pneumonia did not respond to treatment with antibiotics and because none of the investigations proved diagnostic, a serum sample was sent to the Center for Disease Control in Atlanta, Georgia. The indirect fluorescent test for antibody to Lassa virus was positive; therefore the woman was presumed to have Lassa fever. The hospital was closed temporarily and all contacts were traced and kept under surveillance. Although local, provincial and federal health officials worked hard and cooperated closely on this case, subsequent detailed viral studies failed to isolate the Lassa virus and the diagnosis was never confirmed. As a result, a procedure has been developed for the isolation of the patient and the surveillance of the contacts.

On at least two previous occasions Canadian volunteer workers in rural Africa had been sick with suspected Lassa fever. The decision as to whether to bring the patient home is a difficult one. Because treatment is only support-

ive and rural African hospitals do not have sophisticated life-supporting facilities it seems humane to bring the patient home to a well equipped hospital. However, by having a patient with a highly contagious condition in Canada the whole population is exposed to a dangerous disease for which there is no preventive vaccine or treatment. On balance it appears wise to transport the sick person in a specially equipped military aircraft to a specially equipped isolation facility at home. Health and Welfare Canada has installed such an isolation unit at the National Defence Medical Centre in Ottawa for patients with dangerous contagious diseases.

Because several thousand Canadians are working in various parts of West Africa at any one time, many Canadian physicians will be called upon to treat febrile patients returning from Africa. It is therefore useful to review the newly identified viral fevers that are generally endemic only in Africa.

In 1969 three missionary nurses contracted a fever that did not respond to treatment with antimalarial agents and antibiotics. They had contracted the disease while nursing febrile patients in Jos, Nigeria. Two of the nurses died and one recovered after a long illness.² A virus isolated from the nurses was demonstrated to be a new member of the arenaviruses, of which the lymphocytic choriomeningitis (LCM) virus is the prototype. The arenaviruses are ribonucleic acid viruses and include 10 morphologically similar and serologically related viruses. Four of the arenaviruses are known to cause human disease: LCM, aseptic meningitis; Juno, Argentinian hemorrhagic fever; Machupo, Bolivian hemorrhagic fever; and Lassa, Lassa fever.³

Lassa fever is probably not a new disease. Only modern virologic techniques have made the isolation of this

Zyloprim* (allopurinol)

Indications: ZYLOPRIM is intended for the treatment of gout as well as primary and secondary hyperuricaemia. ZYLOPRIM is indicated in the treatment of primary or secondary uric acid nephropathy. ZYLOPRIM is especially useful in patients with gouty nephropathy, in those who form renal urate stones, and those with unusually severe disease. ZYLOPRIM is effective in preventing the occurrence and recurrence of uric acid stones and gravel. ZYLOPRIM is useful in the therapy and prophylaxis of tissue urate deposition, renal calculi and for acute urate nephropathy in patients with neoplastic disease who are particularly susceptible to hyperuricaemia and uric acid stone formation, especially after radiation therapy or the use of antineoplastic drugs.

Contraindications: Zyloprim should not be given to patients who are hypersensitive or who have had a severe reaction to this drug.

Precautions and Warnings: Acute gouty attacks may be precipitated at the start of treatment with Zyloprim in new patients, and these may continue even after serum uric acid levels begin to fall. Prophylactic administration of colchicine and a low dosage of Zyloprim are advisable, particularly in new patients and in those where the previous attack rate has been high. Zyloprim is not recommended for use during pregnancy or in women of child-bearing potential unless in the judgement of the physician, the potential benefits outweigh the possible risks to the fetus. Zyloprim should not be given to children except those with hyperuricaemia secondary to malignancy or with Lesch-Nyhan syndrome. Patients with impaired renal or hepatic functions should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in hepatic or renal functions appear.

Uricosurics and Zyloprim: Combined therapy of Zyloprim and uricosurics will result often in a reduction in dosage of both agents.

Purinethol or Imuran with Zyloprim: In patients receiving PURINETHOL* (mercaptapurine) or IMURAN* (azathioprine), the concomitant administration of 300-600 mg of ZYLOPRIM per day will require a reduction in dose to approximately 1/2 to 1/4 of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of PURINETHOL or IMURAN should be based on therapeutic response and any toxic effects.

Chlorpropamide with Zyloprim: In the presence of allopurinol, there may be competition in the renal tubule for the excretion of chlorpropamide. When renal function is poor, the recognised risk of prolonged hypoglycaemic activity of chlorpropamide may be increased if ZYLOPRIM is given concomitantly.

Coumarin anticoagulants with Zyloprim: It has been reported that under experimental conditions allopurinol prolongs the half-life of the anticoagulant, dicumarol. The clinical significance of this has not been established, but this interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

Adverse reactions: Skin reactions associated with exfoliation, fever, chills, nausea and vomiting, lymphadenopathy, arthralgia and/or eosinophilia are the most common and may occur at any time during treatment. Gastrointestinal disorders were reported but may diminish if Zyloprim is taken after meals.

Symptoms and treatment of overdosage: Overdosage of allopurinol is usually manifested by nausea and vomiting. No treatment is normally required, provided the drug is withdrawn and adequate hydration is maintained to facilitate excretion of the drug. If, however, other forms of acute distress are observed, gastric lavage should be considered, otherwise the treatment is symptomatic.

Pharmacology: When taken orally, allopurinol is rapidly metabolized. The main metabolite is oxypurinol, which is itself a xanthine oxidase inhibitor. Allopurinol and its metabolites are excreted by the kidney, but the renal handling is such that allopurinol has a plasma half-life of about one hour, whereas that of oxypurinol exceeds 18 hours. Thus, the therapeutic effect can be achieved by a once-a-day dosage of ZYLOPRIM in patients taking 300 mg or less per day.

Dosage and administration: ZYLOPRIM, administered orally should be divided into 1 to 3 daily doses. Daily doses up to and including 300 mg may be taken once daily after a meal. Divided doses should not exceed 300 mg. The minimum effective dose is 100 to 200 mg. The average is 200 to 300 mg/day for patients with mild gout, 400 to 600 mg/day for moderately severe tophaceous gout, and 700 to 800 mg/day in severe conditions. The maximal recommended dose is 800 mg per day in patients with normal renal function.

Treatment with 600 to 800 mg daily for two or three days prior to chemotherapy or x-irradiation is advisable to prevent uric acid nephropathy. Treatment should be continued at a dosage adjusted to the serum uric acid level until there is no longer a threat of hyperuricaemia and hyperuricosuria. It is essential that a daily urinary output of two litres or more be maintained during ZYLOPRIM therapy, and neutral or alkaline urine is desirable.

Children: For the treatment of secondary hyperuricaemia associated with malignancies and in the Lesch-Nyhan syndrome, ZYLOPRIM should be given in doses of 10 mg/kg/day. The response should be evaluated after approximately 48 hours by monitoring serum uric acid and/or urinary uric acid levels and adjusting the dose if necessary.

Presentation: ZYLOPRIM 100 mg scored white tablets. Bottles of 100 and 500 tablets; Code: Wellcome U4A. ZYLOPRIM 300 mg scored peach coloured tablets. Bottles of 100 tablets. Code: Wellcome C9B.

Product Monograph available on request.

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LaSalle, Qué.

virus possible, but it may be that previous cases of unexplained fever epidemics have been of Lassa fever.⁴ Having acquired the virus from rodents, humans spread it by means of blood, secretions and body fluids; hospital personnel have the highest risk of acquiring the infection.

The clinical features of Lassa fever are fever, pharyngitis, abdominal symptoms and, in half the cases, lymphadenopathy, leukopenia and albuminuria.⁵ These clinical features are not specific enough to be diagnostic, so a presumptive diagnosis can be made only when there is a history of travel in rural Africa and contact with febrile patients, and when other causes of fever have been excluded. Definitive diagnosis depends on isolation of the virus from blood, urine or other body fluids.⁵

The demonstration of complement-fixing antibodies to Lassa virus antigen is almost certainly diagnostic of Lassa fever. This is especially true when there is a conversion from negative to positive in a paired serum sample. The complement-fixation test, however, is not sensitive enough and may become positive only when the infection is at an advanced stage. The indirect fluorescent antibody test may be more sensitive and may detect antibodies earlier in the course of infection.⁶ Increased sensitivity, however, may be accompanied by increased nonspecificity, and antibodies to other arenaviruses, such as the LCM virus, may crossreact with Lassa viral antigen.

In earlier outbreaks the mortality of Lassa fever was reported to be approximately 50%,⁷ a figure that subsequently has been reduced. Serologic surveys have shown that there are many subclinical and clinically benign infections, and that Lassa fever is more widespread than has previously been reported.^{8,9} In the Sierra Leone outbreak it was estimated that only 1 of 30 patients with infections needed hospitalization, and that the mortality in infected persons was less than 1 in 60.¹⁰ In the most recently published case of Lassa fever imported to the United States the patient had a very mild illness. In none of the 552 contacts kept under surveillance did the illness develop. A serologic survey of the persons at high risk showed no evidence of infection.¹¹ Hence it can be assumed that Lassa fever is not as lethal and contagious as originally thought. Infections acquired in the laboratory appear to be more virulent. In the United States two persons acquired infection in a laboratory, one of whom died.¹²

The principal reservoir of Lassa virus is rodents, particularly the species *Mastomys natalensis*.¹³ This multimammate mouse, the most common and

most widespread rodent in Africa south of the Sahara Desert, is an ideal carrier of animal diseases to humans because of its preference for living near human habitations and its exceptionally high propagation rate.¹⁴ This rodent is also an important link in the plague cycle in southern Africa. Lassa virus has been isolated from other rodents, such as *Rattus rattus* and *Mus minutoides*.¹⁵

In 1967 three simultaneous outbreaks of a severe febrile illness occurred in laboratory workers in Marburg, West Germany, and Belgrade, Yugoslavia. There were 25 primary and 6 secondary cases of infection. The primary infections were all acquired by contact with a consignment of green velvet monkeys from Uganda. The etiologic agent was found to be a virus, previously unknown, that has been named Marburg virus.¹⁵ Subsequent intensive investigations did not reveal the monkeys to be a reservoir of this virus; the reservoir remains unknown.

In late 1976 there was an outbreak of a severe febrile illness with a high mortality in southern Sudan and northern Zaire. It was estimated that in this outbreak there were more than 600 cases and more than 450 deaths. A World Health Organization international commission operating in the endemic areas sent specimens to high security laboratories in England, Belgium and the Center for Disease Control in Atlanta. The virus isolated by all these laboratories was morphologically similar but serologically distinct from Marburg virus. The name Ebola (Ebola is a small river in Zaire) was given to this virus.¹⁶⁻¹⁸

Clinically patients with Ebola virus infections have a high fever, sore throat, muscular pain, vomiting and diarrhea. Some patients have a skin rash and, in severe cases, hemorrhagic tendencies. The infection is transmitted by direct contact with sick patients and particularly their body fluids. Health workers are at the highest risk. At Maridi Hospital, Sudan, in late 1976, 76 hospital staff became ill with this disease and 41 died. Ebola virus infection is currently believed to be highly contagious and lethal. It is, from preliminary reports, more virulent than Lassa fever. To date no reservoir or vector for Ebola virus has been found, but it is generally accepted that monkeys are not the reservoir.

There is no specific treatment for Lassa, Marburg or Ebola fever. Plasma from patients who have recovered from Lassa fever has been used with some success in Lassa patients. Interferon given to a patient with Ebola virus infection acquired in the laboratory may have helped in the patient's recovery.¹⁹

Valium[®] Roche[®] (diazepam)

Classification: anxiolytic/muscle relaxant

Rx summary:

Indications Symptomatic management of mild to moderate degrees of anxiety in conditions dominated by tension, excitation, agitation, fear or aggressiveness. Spasm in muscular disorders of central or peripheral origin.

Contraindications Myasthenia gravis, glaucoma, known hypersensitivity to the drug, and because of lack of sufficient evidence, in children under six months.

Adverse reactions Drowsiness, ataxia, fatigue, dizziness, slurred speech, tremors, hypotension, tachycardia and phlebitis. Paradoxical reactions in psychiatric patients.

Precautions Abstinence from alcohol during treatment. Caution wherever mental alertness or physical coordination is required. Periodic blood counts and liver function tests advisable in long-term use. Caution in severely depressed patients or those with suicidal tendencies.

Dosage (oral) Depending upon severity of symptoms. Adults 2 mg to 10 mg, 2 to 4 times daily. Elderly and debilitated patients - 2 mg, 1 or 2 times daily initially; increase gradually as needed and tolerated. Children - 1 mg to 2½ mg, 3 or 4 times daily initially; increase gradually as needed and tolerated.

Parenteral dosage Acute anxiety or tension states or non-psychotic emotional disorders: 2 mg to 10 mg i.m. or i.v. Repeat in 3 to 4 hours if necessary.

Acute alcoholic withdrawal: 10 mg i.m. or i.v. initially then 5 to 10 mg in 3 to 4 hours if necessary. Relief of muscle spasm: 5 mg to 10 mg i.m. or i.v. initially then 5 mg to 10 mg in 3 to 4 hours if necessary.

Status Epilepticus and severe recurrent seizures: 5 mg to 10 mg i.v. or i.m. and repeat in 2 to 4 hours if necessary. Children: 2 mg to 10 mg i.m. or i.v. or 0.25 mg/kg.

Elderly and debilitated: 2 mg to 5 mg i.m. or i.v.

Supply Tablets, 2, 5, 10 mg; 100, 1000. Suspension, 5 mg/5 ml; 100, 400 ml. Ampoules 10 mg/2 ml; 5, 25. Disposable Syringe 10 mg/2 ml, individually packed, 10.

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'Roche' endorses the sound medical practice that no medication should be taken unless prescribed.



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Motrin (ibuprofen)

Action: Ibuprofen has demonstrated anti-inflammatory, analgesic, and antipyretic activity in special animal studies designed to specifically demonstrate these effects. Ibuprofen has no demonstrable glucocorticoid effect.

Ibuprofen has been found to be less likely to cause gastro-intestinal bleeding in doses usually used than is acetylsalicylic acid.

Clinical trials in man have shown the clinical activity of a dose of 1200-1800 mg of ibuprofen daily to be similar to that of 3600 mg of acetylsalicylic acid daily.

Indications and Clinical Uses: Ibuprofen is indicated for the treatment of osteoarthritis and rheumatoid arthritis.

Contraindications: Ibuprofen should not be used during pregnancy or in paediatric patients because its safety under these conditions has not been established. Ibuprofen should not be used in patients with a history of acetylsalicylic acid-induced bronchospasm.

Precautions: Ibuprofen should be used with caution in patients with a history of gastrointestinal ulceration.

Ibuprofen has been reported to be associated with toxic amblyopia. Therefore, precautions should be taken to ensure that patients on ibuprofen therapy report to their physicians for full ophthalmological examination if they experience any visual difficulty. Medication should be discontinued if there is any evidence of toxic amblyopia.

Adverse Reactions: The following adverse reactions have been noted in patients treated with ibuprofen.

Gastrointestinal: Nausea, vomiting, diarrhoea, constipation, dyspepsia, epigastric pain and guaiac positive stools have been noted. A few cases of gastric or duodenal ulceration, including some complicated by bleeding or perforation have occurred.

Central Nervous System: Dizziness, light-headedness, headache, anxiety, mental confusion and depression were noted in some patients treated with ibuprofen.

Ophthalmological: Blurred vision was noted in some patients and rarely a sensation of moving lights was observed following administration of ibuprofen. In addition there are three published cases of toxic amblyopia associated with the use of ibuprofen. Although a definite cause and effect relationship was not established, the attending physicians considered them to be drug-related. The condition was characterised by reduced visual acuity and difficulty in colour discrimination. Defects (usually centrocaecal) were observed on visual field examination. Symptoms were reversible on discontinuation of treatment.

Skin: Maculopapular rashes and generalised pruritus have been reported with ibuprofen therapy. Occasional cases of oedema have also been reported.

Laboratory Tests: Sporadic abnormalities of liver function tests have occurred in patients on ibuprofen therapy (SGOT, serum bilirubin and alkaline phosphatase) but no definite trend was seen indicating toxicity. Similar abnormalities of white blood count and blood urea determinations were noted. A slight fall in haemoglobin and haematocrit has been noted in some patients.

Symptoms and Treatment of Overdosage: One case of overdosage has been reported. A one-year-old child ingested 1200 mg ibuprofen and suffered no ill effects other than being drowsy the next day. Blood levels of ibuprofen reached 711 mcg/ml, which is considerably above the 90 mcg/ml previously recorded as the highest level seen in adults after a single oral dose of 800 mg. The SGPT level, nine days post-ingestion, was 72. No specific antidote is known. Standard measures to stop further absorption and maintain urine output should be implemented at once. The drug is excreted rapidly and excretion is almost complete in six hours.

Dosage and Administration: To obtain rapid response at the start of treatment, particularly when transferring from other anti-inflammatory therapy, Motrin should be given at a dose of 1200 mg per day in four divided doses. Depending on the therapeutic response, the dose may be adjusted downward or upward keeping the four-times-a-day dosage schedule. The daily dose should not exceed 2400 mg. Maintenance therapy, once maximum response is obtained, will range from 800 to 1200 mg per day. Due to lack of clinical experience, ibuprofen is not indicated for use in children under 12 years of age.

Supplied: 200 mg yellow coated tablets, 300 mg white coated tablets and 400 mg orange coated tablets in bottles of 100 and 1000.

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Hyperimmune plasma is in limited supply and should be given only to patients proven to have the disease and patients who are critically ill and suspected of having the disease. There is no vaccine for these viral infections.

Experience has shown that malaria is the most common cause of fever in patients who have recently been in Africa. The other common causes, in decreasing likelihood, are respiratory infection, preicteric hepatitis, infective enteritis (caused by *Salmonella* or *Shigella*) and invasive amebiasis.²⁰ Physicians must now consider these newly described viral infections in their differential diagnosis. It is suggested that all febrile patients who have been in West or Central Africa in the preceding 2 to 3 weeks be considered to have potential Lassa or Ebola fever. Blood smears should be done immediately, and if they are negative for malaria the patient should be isolated in a single room until the probable diagnosis is more certain. Leukopenia (leukocyte count of less than $4.0 \times 10^9/L$) in a febrile patient from West Africa should suggest typhoid or Lassa fever. In either instance the patient should be isolated. If the patient is likely to have Lassa fever it is important to remember that health workers such as physicians, nurses and laboratory technologists in rural West Africa are at the highest risk. A business man or tourist in a large city in Africa who has no contact with sick people is unlikely to have Lassa or Ebola virus infection.

There are, in large Canadian cities, an increasing number of physicians knowledgeable in importation medicine; family physicians and physicians in the emergency rooms of hospitals should not hesitate to seek advice from them. If the diagnosis of Lassa or Ebola fever is considered likely the hospital microbiologist and district public health officer should be notified immediately. They should then arrange for specimens to be sent to a specialized laboratory for serologic tests and viral isolation. At this time consultation should begin with provincial and federal health authorities (such as the Laboratory Centre for Disease Control, health protection branch, Health and Welfare Canada) concerning more strict isolation of the patient, such as transfer to the isolation unit at the National Defence Medical Centre in Ottawa, and surveillance of contacts. At present the definitive diagnosis of Lassa or Ebola viral infection is difficult to make because we do not have a maximum security laboratory. It is hoped that such a facility will be established so that we do not waste valuable time in sending samples to the Center for Disease Control in Atlanta. In the meantime, in-

activated viral antigen should be made available to large hospitals for rapid serologic diagnosis.

In addition to the improvement of diagnostic ability, the development of a body of physicians knowledgeable in such exotic diseases and the provision for isolation facilities, plans should be made for the collection and storage of plasma from convalescing patients. The latter should be done with the cooperation of an international agency such as the World Health Organization because it will continue to coordinate efforts in controlling future outbreaks of Lassa, Marburg and Ebola fever. The most urgent need, however, is for the development of effective vaccines.²¹ It is hoped that national, international and private charitable agencies will encourage and support scientists to work towards this end.

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