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Possible hepatotoxic effect of rooibos tea: a case report

Marjatta Sinisalo · Anna-Liisa Enkovaara ·
Kari T. Kivistö

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Sirs,

Rooibos (*Aspalathus linearis*) is a popular indigenous South African herbal tea with a growing worldwide market. Traditional medicinal uses of rooibos have included alleviation of infantile colic, asthma, allergies and dermatological problems. It has also been used to treat certain malignancies and inflammatory disorders [1]. Rooibos tea is consumed for enjoyment, as an alternative to Oriental tea, but also for its possible medicinal properties and, to date, no adverse effects have been associated with its consumption [2]. This is noteworthy as the total production of rooibos exceeded 14,000 tons in 2007 [1]. We describe here a patient who temporarily experienced elevated liver enzymes after having consumed relatively large amounts of rooibos tea.

The patient was a 42-year-old woman who had been diagnosed with a low-grade B-cell malignancy, Waldenström's macroglobulinemia, in 2004. Her disease was in a stable stage, with a plasma immunoglobulin M concentration of about 10 g/l (reference range, 0.47–2.84 g/l). She was receiving maintenance treatment with rituximab (Mabthera) every 3 months and prednisolone 5 mg daily. Because her total lymphocyte cell counts had been low during rituximab treatment, prophylaxis for *Pneumocystis jirovecii* pneumonia and toxoplasmosis with co-trimoxazole was given. She used alendronic acid and calcium carbonate with vitamin D as

prophylaxis for osteoporosis. She also took daily supplements of potassium chloride. The patient had no medical history or clinical evidence of viral or autoimmune hepatitis or hemochromatosis, and she did not use any other drugs. She came to our hematology outpatient clinic for rituximab infusion in an excellent physical condition. She had experienced no fatigue, abdominal pain, jaundice, fever or pruritus. Routine blood tests unexpectedly showed elevated plasma levels of alanine transaminase (ALT) (246 U/l; reference range 10–45 U/l), gamma-glutamyl transferase (GGT) (267 U/l; reference range 10–75 U/l) and alkaline phosphatase (ALP) (107 U/l; reference range 35–105 U/l). Her hemoglobin, total leucocytes, platelets, thromboplastin time and plasma bilirubin, albumin, ferritin and C-reactive protein were all in a normal range. The results of the clinical examination were unremarkable. Rituximab was not given, and treatment with co-trimoxazole and alendronic acid was stopped. Tests for viral hepatitis (A, B and C), herpes viruses (cytomegalovirus, Epstein–Barr virus) and toxoplasmosis were negative. An abdominal ultrasound examination revealed mild steatosis in the liver; the spleen was normal. Within 1 week, her ALT value rose up to 1226 U/l, the GGT value to 324 U/l and the ALP value to 129 U/l. The patient denied any excessive alcohol consumption or any excessive use of additional medications, such as paracetamol, but stated that she had begun to drink rooibos tea about 2 weeks prior to the current clinical examination for medicinal purposes. She had bought the tea (Forsman Rooibos Tea; Aaro Forsman Oy, Vantaa, Finland) at a local supermarket and made it according to the directions given on the packet, infusing 1 teaspoonful per cup with boiled water for 10 min. This rooibos preparation was flavoured with small amounts of strawberry, chamomile and petals of daisy, but there is no evidence to suggest that these plants could be hepatotoxic. Her daily consumption of the tea had been about 1 l. After

M. Sinisalo · A.-L. Enkovaara · K. T. Kivistö
Department of Internal Medicine, Tampere University Hospital,
Tampere, Finland

M. Sinisalo · A.-L. Enkovaara · K. T. Kivistö (✉)
Department of Pharmacological Sciences, Medical School,
University of Tampere,
Tampere 33014, Finland
e-mail: kari.kivistö@uta.fi

the patient stopped drinking rooibos tea, her liver enzyme levels normalised within 1 week. Co-trimoxazole and alendronic acid were re-introduced, into her therapeutic regimen, with liver enzymes remaining normal. For safety reasons, re-exposure to rooibos to confirm it as a cause of the observed hepatotoxicity was not considered.

To the best of our knowledge, this is the first report of possible hepatotoxicity due to rooibos ingestion in humans. Based on in vitro studies and studies conducted using animal models, rooibos possesses antioxidant, antimutagenic, immune-modulating and anticancer activities [1, 2]. Evidence of any health benefits in humans is, however, scanty. Rooibos tea (extract of 2 g leaves/100 ml boiled water) administered as sole drinking fluid (ad libitum) to Fischer rats for 10 weeks did not influence liver function as determined by, for example, serum transaminase and bilirubin concentrations [3]. However, the extent of exposure of rats to rooibos in this study may be considered to be “therapeutic” in that one rooibos teabag contains about 2 g of rooibos. As rooibos tea has been shown to induce intestinal CYP3A in rats [4], the possibility that this effect contributed to the elevation of liver enzymes in our patient cannot be excluded.

Interestingly, the results of a controlled study in rats suggest that rooibos may possess hepatoprotective effects [5]. In this study, one group of ten Wistar rats served as a control group, one group was treated with tetrachloromethane (CCl₄) (intraperitoneally twice a week for 10 weeks), one group with CCl₄ and rooibos tea and one group with CCl₄ and N-acetyl-L-cysteine. The animals in the CCl₄ + rooibos group had free access to rooibos tea solution as drinking water, starting 7 days before CCl₄ administration. In addition, they were given 5 ml/kg tea once daily by gavage. Rooibos tea was found to be protective against CCl₄-induced hepatic injury in causing

histological regression of liver cirrhosis and suppressing increases in plasma transaminase, ALP and bilirubin concentrations, as compared to the control group exposed to CCl₄ only.

Although our single case suggests that rooibos tea may have adverse hepatic effects, the tea has an excellent safety record, and further study is therefore required to resolve this question. It could also be that the rooibos used by our patient was contaminated by some hepatotoxic compound, such as a pyrrolizidine alkaloid, or that she was genetically predisposed to react adversely to one of the many bioactive rooibos constituents, which include a number of flavonoids and other polyphenols as well as minerals [1, 2]. Finally, our case underlines the importance of encouraging patients to report their use of dietary supplements, including herbal products, in case of a suspected adverse drug reaction or interaction.

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