VIGS in Suid-Afrika – wie, wat en waar?

Aan die Redakteur: Gerhard Ras[1] het in sy onlangse brief verduidelik dat die krediet vir die diagnose van die eerste VIGS-gevalle in the RSA nie aan hom òf professor Prozesky nie, maar aan drie kollegas toekom. In sy meegaande kommentaar het professor Prozesky geskryf dat hy die viroloog in die span was wat die eerste geval in Suid-Afrika gediagnoseer het. Dit sou miskien die indruk kon skep dat 'n spesifieke virologiese diagnose bevestig was.

Die HIV oftewel VIGS-virus is egter vir die eerste keer 'n jaar later in 1983 in Frankryk gekweek. In Suid-Afrika is HIV vir die eerste keer in November 1984 in die Departement Geneeskundige Virologie by die Tygerberg-hospitaal se opleidingskompleks gekweek, wat dit toe moontlik gemaak het om 'n baie doeltreffende diagnostiese immunofluoressensietoets te ontwikkel wat vir etlike jare in die departement gebruik is totdat kommersiële toetsstelle beskikbaar geword het.[2,3]

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1. Ras G. VIGS in Suid-Afrika – wie, wat en waar? (Brief). *S Afr Med J* 1991; **79:** 224.
2. Spracklen FHN, Whittaker RG, Becker WB, Becker MLB, Holmes CM, Potter PC. The acquired immune deficiency syndrome and related complex. *S Afr Med J* 1985; **68:** 139-143.
3. Becker MLB, Spracklen FHN, Becker WB. Isolation of a lymphadenopathy-associated virus from a patient with the acquired immune deficiency syndrome. *S Afr Med J* 1985; **68;** 144-147.

Aan die Redakteur: Ek wil graag u aandag daarop vestig dat die persoon wat werklik verantwoordelik was vir die diagnose van die eerste geval van VIGS in Suid-Afrika, professor I. W. Simson, hoof van die Departement Anatomiese Patologie, Universiteit van Pretoria, is.

Volgens alle inligting tot my beskikking was die diagnose nooit tydens lewe in die eerste pasiënt gemaak nie. Enkele weke na die voltooiing van die lykskouing het die oorledene se familie professor Simson geskakel om te verneem wat die lykskouingbevindings en oorsaak van dood was. Tydens die gesprek het hy besef dat die pasiënt homoseksueel was, die spreekwoordelike 'pennie' het geval en professor Simson het die diagnose van VIGS gemaak en dit ook so aan dr. T. Hamersma oorgedra.

Na my mening kom die eer dus professor Simson toe.

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Non-cirrhotic portal hypertension

To the Editor: In their paper entitled 'Non-cirrhotic portal hypertension – a new entity in South Africa?', Jaskiewicz and Robson[1] report on 6 patients. I would like to point out that non-cirrhotic portal hypertension (NCPH) is *not* uncommon in this country, especially in the Transvaal, where schistosomiasis is endemic in the north-eastern region. We published a paper[2] on this problem in 1988, emphasising that the two important causes of NCPH in South Africa are bilharzia (7 of our patients) and portal vein thrombosis (8 patients). These 15 patients were treated between 1 June 1986 and 31 May 1987.

Splenectomy is not recommended, since this precludes the performance of a distal splenorenal shunt (DSRS), which should cure the condition permanently. These patients have a good prognosis because the portal hypertension is pre-sinusoidal and liver function is normal. For this reason, obliteration of the varices with sclerotherapy is also an effective form of treatment.

Splenectomy is not recommended unless the patient has hypersplenism with an isolated splenic vein thrombosis, which precludes a DSRS. If the splenic vein is patent, DSRS will decompress both the varices and the spleen, thus treating the bleeding and the hypersplenism with a 'once-only' procedure.

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1. Jaskiewicz K, Robson SC. Non-cirrhotic portal hypertension – a new entity in South Africa?: a report of 6 cases. *S Afr Med J* 1991; **79:** 268-270.
2. Modiba MCM, Pantanowitz D, Franklin J. Presinusoidal portal hypertension at Baragwanath Hospital. *S Afr J Surg* 1988; **26:** 53-55.

Drs Jaskiewicz and Robson reply: We thank Professor Pantanowitz for his interest and comments. The term 'non-cirrhotic portal hypertension' (NCPH) was not used in a descriptive context but in fact referred to a specific clinical entity diagnosed on the basis of characteristic histological findings with or without portal vein thrombosis. This condition was not addressed by Modiba *et al.*[1] in their paper. A major cause of extrahepatic presinusoidal portal hypertension at Groote Schuur Hospital is exclusive portal vein thrombosis. Preconditions for the diagnosis of NCPH are the exclusion of schistosomiasis, other causes of intrahepatic portal hypertension such as nodular regenerative hyperplasia and perisinusoidal fibrosis secondary to toxins or alcohol. In our paper we discussed the possible causation of this syndrome, which remains obscure.

Splenectomy was performed in 2 patients years before assessment at Groote Schuur Hospital. A further patient underwent splenectomy for diagnostic purposes because of the possibility of underlying lymphoma.

Our surgeons do not recommend or perform distal splenorenal shunts for patients with extrahepatic presinusoidal portal hypertension following variceal haemorrhage. We agree with Professor Pantanowitz that patients fare very well with regular sclerotherapy and ablation of varices alone.[2]

1. Modiba MCM, Pantanowitz D, Franklin J. Presinusoidal portal hypertension at Baragwanath Hospital. *S Afr J Surg* 1988; **26:** 53-55.
2. Kahn D, Terblanche J, Kitano S, Berman P. Injection sclerotherapy in adult patients with extrahepatic portal venous obstruction. *Br J Surg* 1987; **74:** 600-602.

Proteinuria and the acute mononucleosis-like illness associated with seroconversion in HIV infection

To the Editor: During December 1988 a 32-year-old woman was admitted to Somerset Hospital with a pyrexial illnes of 1 day's duration. Examination revealed conjunctivitis, mild neck stiffness, a diffuse erythematous macular rash and tender cervical lymphadenopathy. A progressive lymphocytosis developed, peaking in the 2nd week of illness at 74% of the total white cell count of 26,1 x 10^9/l. Many atypical lymphocytes were noted. Mild proteinuria (1+ on dipstick examination) was present on admission. The serum creatinine value was normal at 87 µmol/l.

During the next 2 weeks the patient developed splenomegaly, generalised lymphadenopathy and oral and vaginal candidiasis. At this stage the 24-hour urinary protein excretion was 2,9 g. During the 5th week of illness the serum creatinine value peaked at 185 µmol/1, occasional granular casts were seen in the sediment, and 24-hour urinary protein excretion was 0,83 g.

The patient was initially assessed as having a septicaemic illness, but all blood cultures were negative and she was found to be HIV-positive (Wellcozyme HIV Recombinant enzyme-linked immunosorbent assay (ELISA)) during the 3rd week of illness. Although Western blot testing at that stage was negative, the p24 antibody was shown to be present a week later. A third specimen 17 days later was strongly positive on ELISA and positive on Western blot testing (all viral bands except p17 were present – LavBlot; Pasteur Diagnostics).

The illness, characterised by persistent pyrexia and lassitude, began to resolve during the 4th week, by which time the serum creatinine value had returned to normal. The patient had muscle pain for 2 weeks with a persistently raised creatine kinase (CK) value, but did not have other biochemical features of rhabdomyo-