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AN OUTBREAK OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS IN AN ABORIGINAL COMMUNITY

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Introductions and methods

Outbreaks of acute poststreptococcal glomerulonephritis (APSGN) occurred in one Aboriginal community in the Top End of the Northern Territory in 1980 and 1987[1,2]. APSGN outbreaks are recognised to occur in six to eight year cycles in some places[3], so it was predicted that a further outbreak would occur in this community in 1994. Informal conversations between health care professionals in early June 1994 alerted staff at the Menzies School of Health Research (MSHR) that a number of cases of APSGN had apparently been noted in the community in March and April. The District Medical Officer (DMO) for the community confirmed that there had been five cases admitted to Royal Darwin Hospital (RDH) and a further four cases incompletely investigated, but which were highly suspected of being APSGN. Following discussions with the Disease Control Centre at the Northern Territory Department of Health and Community Services, it was decided to conduct an on-site investigation, although it seemed likely that the outbreak was over.

The objectives of the outbreak investigation were:

- to ascertain individual cases of APSGN which had occurred in this community in 1994,
- to describe the illnesses suffered by these individuals and to ensure that appropriate medical follow-up had occurred,
- to describe the extent of the outbreak,
- to determine if there remained a risk of further cases stemming from this outbreak and to decide on appropriate interventions to reduce the risk of this happening.

The community involved has a population of 1046 residents according to the Australian Bureau of Statistics' 1991 Census. Case finding was undertaken using details of confirmed or suspected cases of APSGN provided by the community DMO and health centre staff. The case definitions used were:

Confirmed case

1. clinical picture consistent with the diagnosis (for example puffy face),
2. abnormal urinary sediment (10×10^6/L red blood cells with 40% dysmorphic cells),
3. evidence of a recent Group A streptococcal infection (elevated ASOT or anti-DNAase B), and
4. reduced complement C3 level.

Possible case

1. clinical picture consistent with the diagnosis,
2. abnormal urinary sediment *or* heavy haematuria with or without proteinuria on urinalysis,
3. absence of results for C3 level and streptococcal serology.

Results

Between 7 March and 27 April 1994 there were five confirmed cases of APSGN and four possible cases (Figure). There was one further possible case in mid-June, seven weeks after the previous last case. Most of the possible cases were unable to be confirmed due to incomplete investigation at the time, particularly the absence of formal urine microscopy and C3 levels.

Figure. Confirmed and possible APSGN cases, 7 March to 27 April 1994, by week

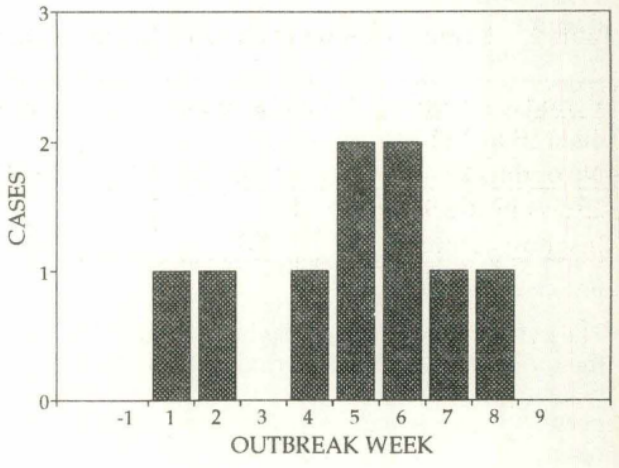

Table 1. Clinical features of the ten confirmed and possible APSGN cases

Clinical feature	Cases
Facial puffiness only symptom	7
Hypertension	4[1]
Elevated urea or creatinine	4[1]

1. All of these were hospitalised.

Of the ten confirmed and possible cases, nine were female. The age range was three to 21 years, with four cases being older than ten years. Six cases were admitted to Royal Darwin Hospital. Seven patients had facial puffiness as the only symptom (Table 1). One required antihypertensive treatment and none required dialysis.

During the outbreak investigation, hypertension was found in only one young adolescent. At subsequent follow-up of this individual, the blood pressure had normalised. In addition, this person and another adolescent case were found to have markedly elevated urinary albumin:creatinine ratios. This measure of proteinuria is thought to be a marker of underlying renal disease. These two were first cousins and had a strong family history of end stage renal disease. They were referred for ongoing review.

On follow-up, all other individuals were well and appeared to have fully recovered from their illness. Of the eight cases whose urine was re-checked, persistent glomerular haematuria was found in six.

Discussion

Because of the retrospective nature of the investigation, the true extent of this outbreak, in particular the number of mild clinical cases (which may not present to the clinic or be noted by health staff) and subclinical cases is not known. This lack of information on these cases may explain the outbreak curve which demonstrates an even spread of cases over the weeks of the outbreak instead of the more usual rapid increase in the early weeks followed by a prolonged tail of secondary cases.

Previous outbreaks of APSGN in this community were documented as more extensive. In 1987 there were 57 cases and in 1980 over 40 cases were found. The reports of these outbreaks do not detail the proportions of symptomatic and asymptomatic cases. During epidemics of APSGN, particularly among families, asymptomatic APSGN episodes outnumber symptomatic episodes by a factor three or four to one[4].

As one means of attempting to assess the true extent of this outbreak, the MSHR Renal Disease Survey team kindly allowed us to scrutinise their records of urine dipstick screening of children from the local school of the affected community. Although this survey has always found a number of children at any one time with blood in their urine, examination of the results revealed an apparent increased rate of haematuria in the paediatric population of this community between November-December 1993 and May-June 1994 (Table 2). This screening did not take place during the period of the detected outbreak, but the results may indicate a number of children in the active or recovery phase of APSGN.

As expected, it was concluded that the outbreak was over by the time of the investigation, and no program of widespread screening or administration of prophylactic benzathine penicillin was carried out. However, as it was possible that the responsible Group A streptococcus (GAS) was still circulating in the community, the health centre staff were encouraged to be extra vigilant about detecting and treating scabies and skin sores.

An interesting feature of this outbreak was the relatively large proportion of cases over the age of ten years. This has been noted in previous studies of APSGN outbreaks in Aboriginal communities[2], whereas the literature implicates the three to ten year old age group as being the most at risk[3,5,6]. Whilst the reasons for this age distribution in Aboriginal communities are not clear, it must be taken into account when planning screening and intervention programs for future outbreaks.

Table 2. Urine screening of children in the community

Period of screening	Total screened	Blood 2+	Blood 1+ or less	Total blood > trace
10.11.93 to 7.12.93 No outbreak	60	6 (10%)	7 (12%)	13 (22%)
25.5.94 to 16.6.94 Just post outbreak	257	53 (21%)	42 (16%)	95 (37%)

The clinical presentation of these cases was typical, in that the majority of clinical cases did not present with the full 'nephritic syndrome' (frank haematuria, reduced urine output, hypertension and oedema) but rather with facial puffiness and microscopic haematuria. The excellent outcome is also in keeping with what is known about the disease[4,7]. In general the prognosis of APSGN is good. In most patients, symptoms and signs will resolve within a week, although persistent urinary abnormalities may be noted for months or years. In a New Zealand study[5], proteinuria was found in 20% of patients two years after APSGN, although haematuria had virtually disappeared by 12 months. Epidemic (outbreak) APSGN has a better outcome than endemic (sporadic) disease, with a mortality of less than 1%[4].

The question of whether APSGN can lead to lasting or progressive renal impairment is unresolved. Most studies have found very little evidence that such progression occurs[4,8-11], although few studies have occurred in populations exposed to persistent GAS infection as is found in Aboriginal communities of Northern Australia. It is possible that the extremely high rates of end-stage renal disease seen in this population may be explained to some extent by recurrent clinical and subclinical episodes of APSGN in early life.

The textbook diagnosis of APSGN requires evidence of nephritis, evidence of current or preceding GAS infection and, in most cases, reduction of serum C3 levels. In Aboriginal communities, GAS infection is endemic and serological evidence of GAS infection (ASOT and anti-DNAase B) is almost invariably present. Moreover, due to reasons unknown, glomerular haematuria and/or proteinuria is also widespread in non-endemic circumstances. In many cases, therefore, the most important laboratory diagnostic criterion is a reduced C3 level.

It is apparent that the health care staff from the community and from RDH were not aware of the need to notify cases of APSGN to the Northern Territory Department of Health and Community Services. Had such notifications occurred at the time it may have been possible to assess this outbreak properly, and to intervene to prevent further cases. This has resulted in two further strategies. First, the Disease Control Centre will be alerting staff at all levels about the diseases which are notifiable, and the importance of notification. Second, a comprehensive protocol is presently being drawn up for investigation and intervention in future outbreaks of APSGN, so that health staff can be assured that notification of this disease will result in practical action to stem the outbreak.

There is the further issue of the reasons for a number of these cases being incompletely worked up and followed up. Investigation of possible cases should include urine microscopy and serum for ASOT, anti-DNAase B, and C3 levels. In questioning the community health staff, it was apparent that the main limitations to their ability to perform these duties were lack of time and staff. This illustrates that even in a community with an on-site DMO the workload is extreme and the provision of facilities may be inadequate. This staffing problem is a long-term one for most remote communities and requires continued emphasis. Service providers in communities need support from researchers, hospital staff and rural health/disease control staff in making their situation a priority.

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