Changing pattern of incidence, aetiology and mortality from acute pancreatitis at Kalafong Hospital, Pretoria, South Africa, 1988-2007: A retrospective evaluation.

I. Chamisa, T. Mokoena, T.E. Luvhengo
Kalafong Hospital, Department of General Surgery, University of Pretoria, Pretoria, South Africa
Correspondence to: **Dr. I. Chamisa**, Email**: charms@doctors.org.uk**

Background: Literature reports from Western countries suggest an increasing incidence of acute pancreatitis (AP) and changing pattern over the past two decades. The aim of this study was to document the incidence, aetiology and mortality from AP over two decades and to examine any emerging trends.
Methods: A retrospective study of all confirmed cases of AP admitted over a 20-year period to the surgical department was performed. Patients' demographics, year of admission, number of attacks, aetiology, management and outcome were entered on a special study proforma.
Results: Altogether 707 attacks of AP (M: F, 5.7: 1) were recorded. The proportion of gallstone AP increased (3.1% to 12.7%) and that of alcohol-related AP decreased (84% to 67.6%). Alcohol was the main aetiological factor for AP. Drugs, hyperlipidaemia, human immunodeficiency virus (HIV) and endoscopic pancreatography-related AP increased in the second decade. The in-hospital mortality rate during the respective periods was 6.5% and 3.1%.
Conclusion: Gallstone AP increased during second decade from more Caucasian admissions and increased gallstones among Blacks. The reduced mortality was attributed to changing trends in the nature and aetiology of AP recorded, heightened awareness of the condition and improved management.

Introduction

The incidence, aetiology, demographics and mortality of AP appears to be changing worldwide[1] but few studies in South Africa [2,3] have examined this. The annual incidence of AP in different Western reports ranges from 5 to 50 per 100 000. [4,5] The differences in consumption and incidence of GD in different parts of the world partly explains this difference. The increased incidence of AP in Western countries [6,7] has been attributed to improved diagnostic tests and imaging procedures, and heightened interest. GD has been reported to be the most common cause of AP in reports from England [8] and Scotland [9] while alcohol has been suggested as an aetiological factor of rising importance. South Africa has undergone significant socio-political changes following the first democratic elections in 1994. Previous racially segregated hospitals are now integrated resulting in a change in the admission patterns and the nature of diseases seen. The aim of this study was to document the incidence, demographics, aetiology and mortality from AP over two decades in a single institution. Patients were classified into decades one (1988-1997) and two (1998-2007) and the results analysed.

Patients and Methods

A retrospective study of all confirmed cases of AP admitted over a 20-year period to the surgery department at Kalafong Hospital was performed. Discharge letters and summaries of all patients with AP were retrieved from a computer database. Patients' demographics, year of admission, number of attacks, aetiology, management and outcome were entered on a special study proforma. The diagnosis of AP was only accepted in those with a consistent clinical history and physical examination, supported by increased serum amylase or lipase levels or from laparotomy, laparoscopy or necropsy findings. Alcohol was considered the aetiology when the patient reported a regular high intake of alcohol or an alcoholic binge directly before the onset of the disease, and no signs of other aetiologies were present.

Ultrasonography for GD disease was routinely performed in most patients. GD was considered the aetiology when there were positive findings of gallstones and no signs of alcohol abuse or other aetiology. The aetiology was considered idiopathic when no aetilogical factor could be found and unspecified if not documented.

Results

Altogether 707 attacks of AP were identified over a 20-year period. Of these 588 (83.2%) were first attacks and 16.8% recurrent. The absolute number of AP admissions between the two decades decreased proportionately to the total number of surgical admissions (34128 and 26723) with a ratio of 1.2% and 1.1 % respectively. Over a period of 20 years, more Caucasian (0.01% to 0.21%) and Asian (0.03% to 0.13%) patients were admitted in the second decade due to the desegregation of health institutions in 1994.

Table 1. Acute Gallstone Pancreatitis by Racial Distribution (Number and %).

Decades	Africans	Asians	Caucasian	Total
First (1988-1997)	**6 (1.4)**	**4 (1.0)**	**3 (0.7)**	**13 (3.1)**
Second (1998-2007)	**14 (4.8)**	**3 (1.0)**	**20 (6.9)**	**37 (12.7)**
Total	**20 (6.2)**	**7 (2.0)**	**23 (7.6)**	**50 (15.8)**

Table 2. Aetiology of Acute Pancreatitis by Decades (Number and %).

Aetiology	First (1988-1997)	Second (1998-2007)
Alcohol	**350 (84.0)**	**196 (67.6)**
Idiopathic	**32 (7.6)**	**22 (7.6)**
Gallstones	**13 (3.1)**	**37 (12.7)**
Unspecified	**20 (4.8)**	**10 (3.4)**
Trauma	**2 (0.5)**	**7 (2.4)**
Post ERCP	**0 (0)**	**6 (2.1)**
Drugs	**0 (0)**	**6 (2.1)**
Hyperlipidaemia	**0 (0)**	**5 (1.7)**
Pancreatic carcinoma	**0 (0)**	**1 (0.3)**
Total	**417 (100)**	**290 (100)**

There was a significant male preponderance (M:F, 5.7:1). The number of females with AP increased by 12.8% between the decades. AP in both decades was most common in 31-40 years age group and the proportion of Black patients with gallstone AP increased by 16% during the study period (Table 1).

In both decades for both genders, alcohol was the main aetiological factor for AP (84.0% and 67.6%) respectively (Table 2). There was an overall increase in gallstone AP between the two decades by 9.7%, which was attributed to changing demographics with admission of Caucasians and absolute increase among the Black population. The incidence of gallstone disease (GD) in general increased by 1.2% (Table 3).The second decade saw a rise in anti-retroviral drugs, endoscopic retrograde cholangiopanctreatography (ERCP) and hyperlipidaemia-related AP. One 73 year old patient had AP related to pancreatic carcinoma. During the study period, the rate of ERCP increased by 23% and six episodes of ERCP-induced AP (2.1%) were identified.

The in-hospital mortality rate during the respective decades was 6.5% and 3.1%. Mortality from alcohol-induced AP decreased by 4.9% but that for gallstone AP decreased less, by 2.3% (Table 3). Two deaths from the first decade were one each blunt trauma-related and 'idiopathic' AP. Three deaths in the second decade were: one 'idiopathic' and two related to HIV and HIV drugs. There was a downward trend in mortality from AP probably attributed to changing pattern in the aetiology of AP and alsoimproved management.

Table 3. Incidence and Demographics of Symptomatic Gallstone Disease in General.

	Decades	
	First (1988-1997)	Second (1998-2007)
Total cases	**240 (0.7%*)**	**500 (1.9%*)**
Average age (Y)	**32.2**	**47.3**
Gender ratio (F:M)	**3.9:1**	**4.3:1**

* Per cent of total surgical admissions.

Table 4. Percentage mortality per decade according to aetiology

	Decades	
Aetiol Aetiology	First (1988-1997)	Second (1998-2007)
Gallstones	**7.7**	**5.4**
Alcohol	**6.9**	**2.0**
Others	**0.6**	**1.5**

Discussion

AP has been known for more than a century, but its epidemiology remains poorly documented especially in African countries. Western studies have indicated an increasing incidence rate during the past two decades.[1,10] Beyers, [11] in a review of surgical diseases at Johannesburg during the 5-year period 1921-1926, stated that no cases of AP were observed in Blacks. Thereafter, in the 3-year period 1981-1983 there were 55 new cases, [12] the increased admission rate occurring some 20 years after Blacks had access to Western-type alcohol. African literature is scanty on this subject and comparing the incidence of AP between different reports is difficult.

There was no significant change in the overall incidence rate of AP during the study period. This finding differs from Western reports, which documented a rise in the incidence of AP during the last 20 years[13] . The change in the admission pattern during the study period could explain the apparent decrease in AP admissions in the second decade when the Black middle class patients gained wider access to private health care after. Although more men than women were admitted throughout the study period, the incidence of AP in women began to increase considerably in the second decade. AP was almost six times more in males than in females, which we linked to the higher consumption of alcohol by this gender group. Differences in lifestyle amongst the young may explain the young age peak incidence.

A significant number of our patients were labelled as 'idiopathic' AP, which serves to highlight the importance of excluding all possible underlying causes to reduce the risk of recurrence attacks. The acquired immunodeficiency syndrome (AIDS) epidemic and the widespread use of HIV drugs saw a rise in HIV and HIV drugs-related AP mostly in the second decade. We partly associated the increase in hyperlipidaemia-related AP and gallstone AP with the change in the admission pattern in the second decade (more Caucasians and Asians admitted in formally Africans-only hospitals). Our rate of ERCP-induced AP (2.1%) was lower than that from a recent review of prospective series which found the mean frequency to be 5.2% after diagnostic and 4.1% after therapeutic ERCP.[14]

In a 12-month audit of patients with a first attack of AP undertaken at Baragwanath Hospital in 1994 [15], alcohol was the predominant aetiological factor in 83.1%, gallstone disease in 7.4% and idiopathic causes in 6.6%. Their overall mortality was 8%. In a study from London (1988-1992), alcohol represented 29% and gallstones 30% of episodes of AP.[16] These Western results contrast sharply with our findings and those from Baragwanath Hospital in which alcohol predominates as the cause of AP although gallstones are on the increase. It is difficult to ascertain whether this trend can be attributed to improvements in patients care or to an increasing incidence of mild attacks. An increase in the diagnostic rate is likely to have resulted in the diagnosis being established in greater numbers of patients with mild disease and thus a fall in the case mortality rate. Previous South African studies have shown that in Soweto, alcohol-related AP is common, [17] and the first attack pursues an aggressive course with significant long-term morbidity. The association between increasing age and death from AP is well-described [18] and was confirmed in this study.

Conclusion

The absolute number of AP admissions between the two decades decreased proportionately to the total number of surgical admissions. Alcohol is the main aetiological factor for AP in our setting, however GD and gallstone AP has increased in the Black population. There has been a steady rise in the incidence of HIV and HIV-drugs related AP over the past two decades. The increase in females has been attributed more to increased alcohol consumption than to GD. There was a general downward trend in mortality from AP but that among the elderly remains high. While improved treatment of AP may have contributed to the falling case fatality rate, another explanation may be the increased diagnosis of mild AP through the wide use of diagnostic pancreatic enzymes elevation, which has lower mortality.

References

1. Appelros S, Borgstrom A. Incidence, aetiology and mortality rate of acute pancreatitis over 10 years in a defined urban population in Sweden. *Br J Surg* 1999; 86: 465-470.
2. D'Egidio A. Acute pancreatitis. Hillbrow Hospital experience. *S Afr J Surg* 1988; 26 (4): 163-165
3. Beiles CB. Acute pancreatitis. Experience at the Johannesburg Hospital. *S Afr J Surg* 1985; 23 (2): 63-66
4. Trapnell JE, Duncan EHL. Patterns of incidence in acute pancreatitis. *BMJ* 1975;ii: 179-183.
5. Thomson HJ. Acute pancreatitis in north and north-east Scotland. *J R Coll Surg Edinb* 1985; 30: 104-111.

6. Wilson C, Imrie CW. Changing patterns of incidence and mortality from acute pancreatitis in Scotland, 1961-1985. *Br J Surg* 1990; 77:731-734.
7. Corfield AP, Cooper MJ, Williamson RC. Acute pancreatitis: a lethal disease of increasing incidence. *Gut* 1985; 26:724-729.
8. Bourke JB. Variation in annual incidence of primary acute pancreatitis in Nottingham, 1969-1974. *Lancet* 1975; ii: 967-969.
9. Imrie CW. Observations on acute pancreatitis. *Br J Surg* 1974; 61:539-544.
10. Mckay CJ, Evans S, Sinclair M et al. High early mortality rate from acute pancreatitis in Scotland, 1984-1995. *Br J Surg* 1999; 86:1302-1305.
11. Beyers CF: Incidence of surgical diseases among the Bantu races of South Africa. *J Med Assoc S Afr* 1927; 1:606-612.
12. Segal I, Leios M, Grieve T. The emergence of chronic calcifying pancreatitis in a developing country; in Gyr K, Singer MV, Sarles H (eds): Pancreatitis: Concepts and Classification. Amsterdam, *Excepta Medica*, 1984, pp417-420.
13. Thomson SR, Hendry WS, McFarlane GA *et al*. Epidemiology and outcome of acute pancreatitis. *Br J Surg* 1987; 74: 398-401.
14. Gottlieb K, Sherman S. ERCP and endoscopic biliary sphincterotomy-induced pancreatitis. *Gastrointest Clin N Am* 1998; 8: 87- 114.
15. MacPhail AP, Simon MO, Torrance JD *et al*: Changing patterns of dietary iron overload in black South Africans. *Am J Clin Nutr* 1979; 32:1272-1278.
16. Mann DV, Hershmann MJ, Hittinger R et al. Multicentre audit of death from acute pancreatitis. *Br J Surg* 1994; 81: 890-893.
17. Segal I. Pancreatitis in Soweto, South Africa: Focus on alcohol-related disease. *Digestion* 1998; 59 (suppl): 25-35.
18. Svensson JO, Norback B, Bokey EL et al. Changing pattern in aetiology of pancreatitis in an urban Swedish area. *Br J Surg* 1979; 66: 159-161.